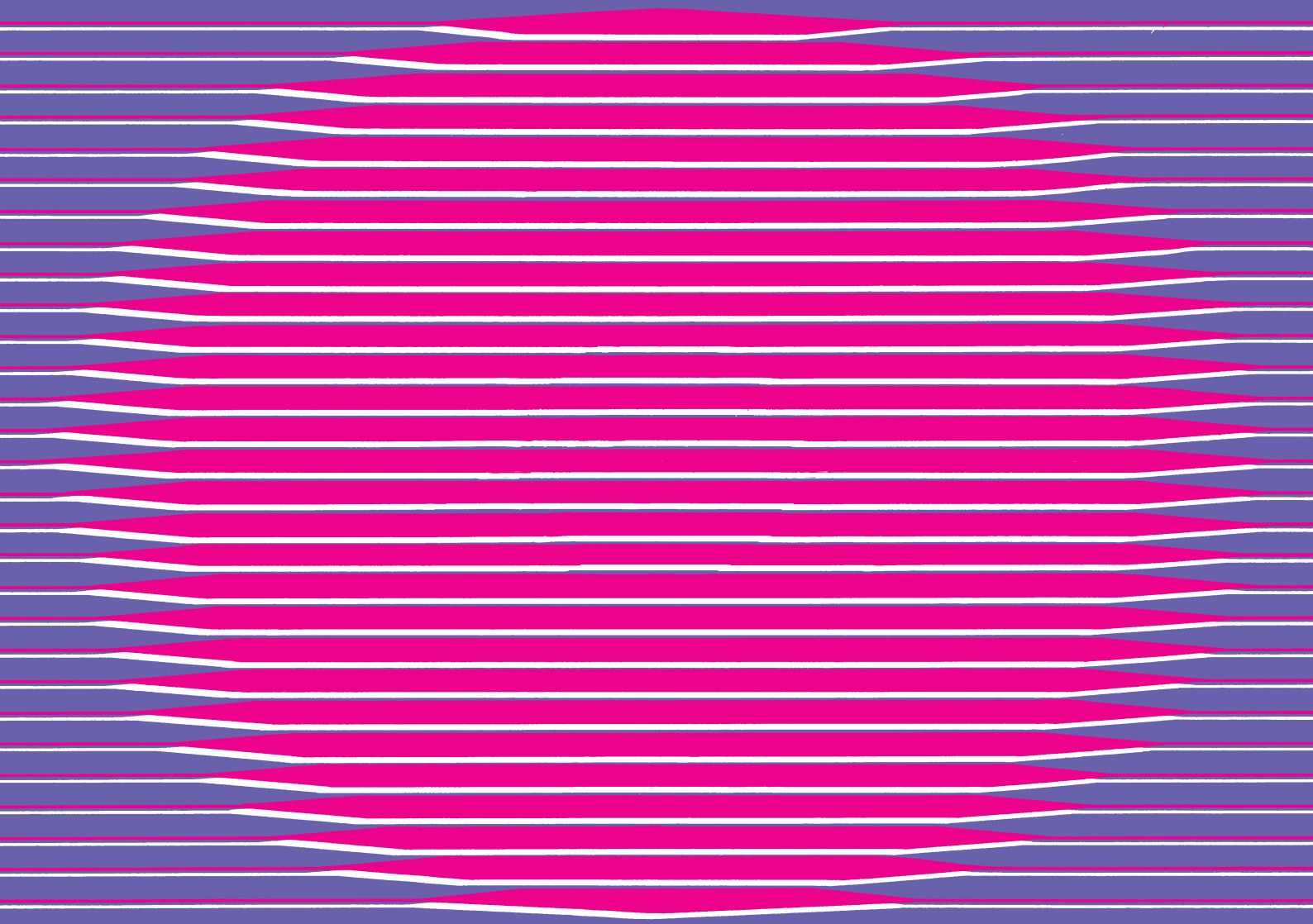
SOURCES, EFFECTS AND RISKS OF IONIZING RADIATION

United Nations Scientific Committee on the Effects of Atomic Radiation
1988 Report to the General Assembly, with annexes



UNITED NATIONS

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1988 Report to the General Assembly, with annexes

UNITED NATIONS
New York, 1988

NOTE

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CONTENTS

Report of the United Nations Scientific Committee on the Effects of Atomic Radiation to the General Assembly

CONTENTS

INTRODUCTION

1. This is the tenth in a series of substantive reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)[a] to the General Assembly[b]. The preparation of this Report and its scientific annexes took place from the thirty-first to the thirty-seventh sessions of the Committee. The material of this report was developed at annual sessions of the Committee, based on working papers prepared by the Secretariat that were modified and amended from one session to the next according to the Committee's requests. During the period of preparation of this Report, which contains seven scientific annexes, another Report containing three scientific annexes was completed at the thirty-fifth session of the Committee. These two reports, referred to as the 1986 and 1988 Reports, constitute the latest comprehensive assessment by the Committee of the sources, effects and risks of ionizing radiation.

2. The following members of the Committee served as Chairmen, Vice-Chairmen and Rapporteurs, respectively, at the following sessions: thirty-first session, Z. Jaworowski (Poland), D. Beninson (Argentina) and T. Kumatori (Japan); thirty-second and thirty-third sessions: D. Beninson (Argentina), T. Kumatori (Japan) and A. Hidayatalla (Sudan); thirty-fourth and thirty-fifth sessions: T. Kumatori (Japan), A. Kaul (Federal Republic of Germany) and A. Hidayatalla (Sudan); thirty-sixth and thirty-seventh sessions: B. Lindell (Sweden), K.H. Lokan (Australia) and J. Maisin (Belgium). The names of those experts who attended the thirty-first to the thirty-seventh sessions of the Committee in an official capacity as representatives or members of national delegations are listed in Appendix I.

3. In approving this Report, and assuming therefore full responsibility for its content, the Committee wishes to acknowledge the help and advice given by a small group of consultants who assisted in the preparation of the text and scientific annexes, upon appointment by the Secretary-General. Their names are given in Appendix II. They were responsible for the preliminary reviews and evaluation of the technical information received by the Committee or available in the open scientific literature, on which rest the final deliberations of the Committee. Additional assistance and financial support for the preparation of some of the scientific annexes were offered to the Committee by various international and national organizations. The Committee would like to express its gratitude to these organizations, which are listed in the relevant annexes.

4. The sessions of the Committee held during the period under review were attended by representatives of the United Nations Environment Programme (UNEP), the World Health Organization (WHO), the Food and Agriculture Organization of the United Nations (FAO), the International Atomic Energy Agency (IAEA), the International Commission on Radiological Protection (ICRP) and the International Commission on Radiation Units and Measurements (ICRU). The Committee wishes to acknowledge their contributions to the discussions.

5. Reports received by the Committee from Member States of the United Nations and members of the specialized agencies and the International Atomic Energy Agency, as well as from these agencies themselves, during the period from 19 April 1986 to 17 June 1988 are listed in Appendix III. Reports received before 19 April 1986 were listed in previous Reports of the Committee to the General Assembly. This information received officially by the Committee was supplemented by, and interpreted with the help of, many other data available in the current scientific literature or, in a few cases, from unpublished communications by individual scientists.

6. In the following Report the Committee summarizes the main conclusions of the specialized studies undertaken, also in the light of previously released substantive documents. The material is presented at the most general level possible, in view of the difficult concepts

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[a] The United Nations Scientific Committee on the Effects of Atomic Radiation was established by the General Assembly at its tenth session in 1955. Its terms of reference are set out in resolution 913 (X). It was originally composed of the following Member States: Argentina, Australia, Belgium, Brazil, Canada, Czechoslovakia, Egypt, France, India, Japan, Mexico, Sweden, Union of Soviet Socialist Republics, United Kingdom of Great Britain and Northern Ireland and United States of America. The membership of the Committee was subsequently enlarged by the General Assembly in its resolution 3154C (XXVIII) to include Germany, Federal Republic of, Indonesia, Peru, Poland and Sudan. By resolution A/RES/41/62B the General Assembly increased the membership of the Committee to a maximum of 21 and invited the People's Republic of China to become a member.

[b] Previous substantive reports of the United Nations Scientific Committee on the Effects of Atomic Radiation to the General Assembly are to be found in Official Records of the General Assembly, Thirteenth Session, Supplement No. 17 (A/3838); ibid., Seventeenth Session, Supplement No. 16 (A/5216); ibid., Nineteenth Session, Supplement No. 14 (A/5814); ibid., Twenty-first Session, Supplement No. 14 (A/6314); ibid., Twenty-fourth Session, Supplement No. 13 (A/7613); ibid., Twenty-seventh Session, Supplement No. 25 (A/8725); ibid., Thirty-second Session, Supplement 40 (A/32/40); ibid., Thirty-seventh Session, Supplement No. 45 (A/37/45); ibid., Forty-first Session, Supplement No. 16 (A/41/16). These documents are referred to as the 1958, 1962, 1964, 1966, 1969, 1972, 1977, 1982 and 1986 Reports, respectively. The 1972 Report with scientific annexes was published as: Ionizing Radiation: Levels and Effects. Volume I: Levels, Volume II: Effects (United Nations Publication, Sales No. E.72.IX.17 and 18). The 1977 Report with scientific annexes was published as: Sources and Effects of Ionizing Radiation (United Nations Publication, Sales No. E.77.IX.1). The 1982 Report with scientific annexes was published as: Ionizing Radiation: Sources and Biological Effects (United Nations Publication, Sales No. E.82.IX.8). The 1986 Report with scientific annexes was published as: Genetic and Somatic Effects of Ionizing Radiation (United Nations Publication, Sales No. E.86.IX.9).

and notation that characterize this field. After a chapter summarizing the developments and trends that have become apparent throughout the years, the highlights and conclusions to be drawn from the most recent studies in the fields of radiation physics and biology are presented. This main text is followed by the supporting scientific annexes, which are written in a format and a language that are essentially aimed at specialists.

7. Following established practice, only the main text of the Report is submitted to the General Assembly, while the full Report, including the scientific annexes, will be issued as a United Nations sales publication[c]. This practice is intended to achieve wider dissemination of the findings for the benefit of the international scientific community. The Committee wishes to draw the attention of the General Assembly to the fact that the main text of the Report is presented separately from its scientific annexes simply for the sake of convenience. It should be understood that the scientific data contained in the annexes are of great importance because they form the basis for the conclusions of the report.

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[c]United Nations Publication, Sales No. E.88.IX.7.

I. HISTORICAL REVIEW

A. GENERAL CONSIDERATIONS

8. Throughout the thirty-three years of its existence, the Committee has assertively attempted to provide the best possible estimates of: (a) doses received by the world's population in the past, and expected to be received in the future, from various natural and man-made sources of radiation, and (b) risks of induction of various types of harm by radiation, both in the short term and the long term, by individuals directly receiving such doses or by their descendants over many generations.

9. With the passing of time and the increase in number and complexity of the Reports issued by the Committee, it is becoming increasingly difficult, even for the specialists, to trace back to earlier publications the development of the main ideas underlying the Committee's assessments and how these assessments have changed with time and as a result of increasing scientific knowledge. It would seem useful, therefore, to make available in compact, summary form the main conclusions reached in the fields mentioned above. This summary is intended to serve a number of purposes. First, it will inform the General Assembly about the Committee's work and its findings. Second, for the Committee's membership which has been changing gradually over the years, it will form a record of how the Committee's thinking has evolved. Lastly, it will be placed at the disposal of the international scientific community, for whom UNSCEAR Reports and scientific annexes have become a basic reference.

10. What follows in this chapter is therefore a summary of the Committee's assessments in the fields of dose estimation (which pertains closely to the subjects of physics) and risk assessment (which involves physical as well as radiobiological and medical considerations). It aims at giving an account of both the general principles underlying the estimates and the conclusions reached, in a language that is as plain as the complexity of the subjects allows but without much of the discussions supporting the choices made at any particular time. For this, as well as for other technical and methodological details, reference is made to the Reports to the General Assembly issued from 1958 to 1986. A complete list of these publications issued by the Committee appears in footnote *b* to paragraph 1 of this Report. Current assessments are examined in more detail in the following chapter II.

B. CONCEPTS, QUANTITIES AND UNITS

11. Radiation is a transport of energy through space. In traversing material, radiated energy is absorbed. In the case of ionizing radiation, which is the type of radiation that concerns the Committee, the absorption process consists in the removal of electrons from the atoms, producing ions. Ionizing radiation may be produced in man-made devices, such as x-ray tubes, or it may come from the disintegration of radioactive nuclides, the phenomenon that is called radioactivity. While nuclides such as these occur naturally, they may also be produced artificially, as in nuclear reactors. The two basic quantities in the assessment of radiation levels and effects are the activity of a radioactive material and the radiation dose. The Committee uses the system of radiation quantities and units adopted in 1980 by the International Commission on Radiation Units and Measurements (ICRU).

1. Activity

12. The *activity* of a radioactive material is the number of nuclear disintegrations per unit time. The unit that the Committee used for this quantity up to and including its 1977 Report was the *curie* (Ci), which is 37 billion ($3.7\ 10^{10}$) disintegrations per second, a number which was originally introduced because it is the approximate activity of 1 gram of radium-226.

13. The present unit of activity has been given the special name *becquerel* (Bq). One becquerel is one disintegration per second.

14. The word radioactivity denotes the phenomenon of radioactive disintegration. It is not a synonym for "activity", nor should it be used to mean "radioactive material".

2. Radiation dose

15. The term radiation dose can mean several things (e.g., absorbed dose, dose equivalent or effective dose equivalent). The *absorbed dose* of radiation is the energy imparted per unit mass of the irradiated material. Up to and including the 1977 Report, the Committee used the *rad* as the unit of absorbed dose (1 rad = 0.01 joule/kg). The present unit of absorbed dose is joule/kg, for which the special name *gray* (Gy) is used. Thus, 1 rad = 0.01 joule/kg = 0.01 Gy.

16. Different types of radiation have different Relative Biological Effectiveness (RBE). The RBE of one type of radiation in relation to a reference type of radiation (usually x or gamma) is the inverse ratio of the absorbed doses of the two radiations needed to cause the same degree of the biological effect for which the RBE is given.

17. When the first UNSCEAR Reports were prepared, the International Commission on Radiological Protec-

tion (ICRP) had recommended certain values of RBE for the purposes of radiation protection. The absorbed doses of various radiations were multiplied by these values to arrive at doses weighted for the purposes of radiation protection (e.g., for comparison with dose limits). The unit of this weighted absorbed dose was called *rem*.

18. The use of the term RBE in two contexts, radiation protection (where it only meant the standard values recommended by ICRP) and in radiobiology (where it meant the most likely value in a given exposure situation for a specified biological effect), caused some problems. ICRP and ICRU therefore decided to establish a new quantity, the *dose equivalent*. This would be the product of the absorbed dose and a so-called quality factor (first denoted QF and later Q), and its unit would be the rem. The quality factor was given by ICRP as a function of the capacity of each radiation to produce ionization, expressed as the linear energy transfer (LET). For practical applications, ICRP suggested that it would suffice to use approximations of average values, i.e., one unique value of QF (Q) for each type of radiation. It suggested values of Q = 1 for x rays, gamma rays and beta particles, Q = 10 for fast neutrons (changed to Q = 20 in 1985), Q = 10 for alpha particles (changed to Q = 20 in 1977), and Q = 20 for heavy particles. The Committee has also used these factors but continued to use Q = 10 for fast neutrons.

19. In the UNSCEAR Reports, when doses are expressed in rem, the ICRP values of "RBE (protection)", QF or Q have been used in most cases, however, when authors express doses in rem, they may have used the primary, LET-related definition of QF (Q).

20. When the Committee began in 1982 to apply the new international unit system and the absorbed dose was given in Gy instead of rad, the new unit for dose equivalent was named the *sievert* (Sv).

21. In addition to absorbed dose and dose equivalent, there is a third quantity that may be meant when an author speaks of radiation dose, namely, the *exposure*. Exposure is the total electrical charge of ions of one sign produced in air by electrons liberated by x or gamma rays per unit mass of irradiated air. Since the exposure is a measure of the ionization that x- or gamma-radiation would produce in air, it is therefore only applicable for those types of radiation. The unit of exposure is *coulomb/kg*, but the old unit, the *roentgen* (R) is still in use. One roentgen is equal to $2.58\ 10^{-4}$ coulomb/kg. The word "exposure" is also used in this Report in its common meaning of being exposed to something, e.g., a radiation source.

22. In this latter meaning, the exposure to radon decay products can be expressed in two different ways: as the amount of inhaled decay products, taking into account their potential to emit radiation energy, or as the product of the time during which the decay products were inhaled and their concentration in the inhaled air. The potential alpha energy of the inhaled decay products may simply be expressed in *joule* (J). The potential alpha energy concentration in air is expressed in J/m^3 or in the older unit, the *working level* (WL), where 1 WL = $2.08\ 10^{-5}$ J/m^3. For radon in equilibrium with its decay product, this corresponds to a concentration of 3700 Bq/m^3. Exposure to the decay products is customarily expressed in terms of the *working level month* (WLM) or, as is now also common, Bq h/m^3.

23. In the 1958 Report of the Committee, the word "dose" was used loosely, and the quantity meant had to be inferred from the units used (roentgen, rad or rem). In the UNSCEAR 1962 Report, doses were sometimes expressed in rad, sometimes in rem. However, in the next five Reports (up to and including the 1977 Report), the approach was more stringent. The absorbed dose was used consistently and the dose equivalent was deliberately avoided. The main reason for this was that one use of the physical and biological information was to provide a basis for estimates of RBE and therefore also to evaluate the appropriateness of the recommended values for Q. To present doses as dose equivalents would have been to beg the issue. Sometimes, however, exposures had to be expressed in roentgen because this was how the original data had been presented.

24. With the UNSCEAR 1982 Report, the practice changed. The Committee had gradually become more concerned with risk estimates and was not satisfied with merely reporting levels of absorbed dose. One reason for this was the growing evidence that radon daughter products caused lung cancer and that these daughter products were present in high concentrations in dwellings. Previously, dose contributions from types of radiation with RBEs other than unity had not been considered important and the presentation of absorbed doses was thought to be sufficient. Now, the situation was different. While it was recognized that the dose equivalent was a quantity designed for radiation protection and that the Q values recommended by ICRP might differ from the true values of RBE, the dose equivalent was still believed to give a better indication of risk than the absorbed dose.

3. Development of dosimetric concepts

25. Paragraphs 25-41 review historical development of other concepts and quantities used by the Committee. When the UNSCEAR 1958 Report was issued, two biological effects were prominent: leukaemia and hereditary harm. For that reason, priority was given to calculating dose in the red bone marrow and gonads. In the case of dose in the gonads, it was obvious that the dose would be relevant to risk assessment only if it were calculated for individuals young enough to expect children. In the case of dose in the bone marrow, the question arose whether the mean dose or the peak dose would be relevant; the ensuing discussion led to the concept of mean marrow dose.

(a) The genetically significant dose

26. It was realized early that for most populations the medical uses of x rays were the main source of

man-made exposure. However, dose distribution within a patient is very uneven, so the dose assessment is not easy. In addition, the age distribution in exposed patient groups differs from that in the general population. To solve these problems, the Committee derived the concept of *genetically significant dose* (GSD), defining it as "the dose which, if received by every member of the population, would be expected to produce the same total genetic injury to the population as do the actual doses received by the various individuals". On the basis of this definition, the Committee developed a formula and an assessment procedure for estimating the genetically significant dose from various types of x-ray examinations. This is described in detail in the 1958, 1962 and 1972 Reports.

(b) The mean marrow dose

27. Assuming that the mean dose in the active (red) bone marrow would be the quantity relevant to assessing the leukaemia risk and using information on the distribution of active marrow in the skeleton, this quantity was assessed for various types of x-ray examinations. While it was recognized that this would not be the relevant quantity if the dose-response relationship was non-linear or showed a dose threshold, it was equally clear that if the relationship was linear and showed no threshold, yet another quantity, the *per caput mean marrow dose* in a population would be of interest, and this quantity was assessed in the UNSCEAR 1958 Report.

(c) The dose commitment

28. Nuclear test explosions in the atmosphere introduced time elements that made this source of radiation different from, for example, medical exposures, in the sense that the period of practice and the period of exposure were different. After each nuclear explosion, some long-lived radionuclides were released that will persist in the biosphere for many years, causing radiation exposures. To have presented the annual doses caused by the tests that had been carried out up to the time the UNSCEAR 1958 Report was drafted would not have given the full picture: namely, it would not have shown that the contamination was expected to last for a long time, thus committing mankind to exposures in future years. The situation was described by diagrams in the UNSCEAR 1958 Report. These diagrams showed the doses to be expected under various assumptions about the period of future testing.

29. In the UNSCEAR 1962 Report, the Committee introduced the concept of *dose commitment*. The dose commitment from one year of practice is the sum of the per caput annual doses inevitably caused by the resulting environmental contamination over future years. It can be shown that the dose commitment from one year of a practice is equal to the highest annual per caput dose in the future, if the practice continues indefinitely at constant rate. This relationship made it possible to assess the future consequences of continuing various practices.

30. In the UNSCEAR 1964 Report, the dose commitment was defined as "the integral over infinite time of the per caput dose rates delivered to the world's population as a result of a specific practice, e.g., a given series of nuclear explosions. The actual exposures may occur over many years after the explosions have taken place and may be received by individuals not yet born at the time of the explosions." This definition was repeated in subsequent Reports and a stricter mathematical presentation was given in 1969 and 1977. It should be mentioned that when the integration of the average dose rates is carried out not to infinity but only to some specified time, one is dealing with truncated dose commitments.

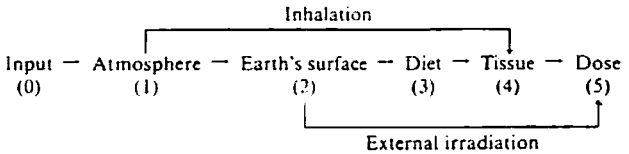
(d) Collective doses and collective dose commitments

31. The use of the dose commitment concept did not carry any implication of assumptions with regard to the dose-response relation at the low doses of radiation that were assessed for the environmental contamination; it was merely a mathematical device for adding inevitable dose contributions.

32. Another concept is the *collective dose*. Assuming a proportionality between dose increments and resulting increments in the risk of harm, the expected number of harmfully affected individuals would be proportional to the collective dose, since the latter is defined as the product of the number of exposed individuals and their average radiation dose. Before 1977, the Committee hesitated to assess collective doses, because doing so would have implied an unproven dose-response relation. In its 1977 Report, however, the Committee assessed collective absorbed doses from various sources and practices. Where a practice was expected to cause exposures over future years, the *collective dose commitment* was assessed. This is simply the total collective dose expected from a given practice over all future time.

(e) Transfer coefficients

33. Dose commitments from practices causing environmental contamination are proportional to the amount of the relevant radionuclides that have been released into the environment. Thus, the assessment involves the study of a chain of events starting from the primary injection of radioactive material into, for example, the atmosphere and ending with the eventual irradiation of body tissues. This chain of events can be represented schematically:



34. Beginning with the UNSCEAR 1969 Report, the Committee has assessed *transfer coefficients*, i.e., the quotients of the time-integrated quantity (e.g., activity concentration) in each step and the corresponding quantity in a previous step. For example, the transfer coefficient P_{34} is the time-integrated activity concentration in a given tissue divided by the time-integrated concentration of the same nuclide in the diet. The product of all transfer coefficients directly relates the amount of radioactive material injected into the

atmosphere to the resulting dose. The mathematical formulation and assessment procedure were described in detail in the UNSCEAR 1969 Report.

(f) Organs of interest

35. As has already been mentioned, in the UNSCEAR 1958 Report the Committee calculated doses for only two organs: the gonads and the active bone marrow. They were the only organs for which some risk estimates had been made at that time. In the UNSCEAR 1969 Report, the Committee added dose assessments for one more tissue, namely the cells lining bone surfaces. Up to 1972, the dose assessments had thus been made for three organs (gonads, active bone marrow and bone surface cells), although the Committee had in fact made risk estimates for other organs, such as the thyroid (1964 and 1972) and breast and lung (1972). One reason for limiting the number of organs was that the dose assessments would become more complicated the more organs the Committee included and comparisons between various sources would become very difficult.

36. Nevertheless, in the UNSCEAR 1977 Report the Committee added still one more organ, the lung, because it had become increasingly evident that the alpha-emitting daughter products of radon in dwellings were biologically significant and that radon escaping from uranium mill tailings was generating very high long-term commitments.

(g) The effective dose equivalent

37. In 1977, ICRP published a revision (ICRP Publication 26) of its general recommendations, in which it suggested that a weighted sum of the radiation dose equivalents in the most radiosensitive organs and tissues should be the basis for radiation protection assessments. This weighted sum was named the *effective dose equivalent*. It was to have the same unit as the dose equivalent, i.e., the sievert. The effective dose equivalent is determined using only the organ weighting factors recommended by ICRP on the basis of risk assessments. Other types of sums of weighted organ doses, with different weighting factors, must not be called effective dose equivalents.

38. The effective dose equivalent was originally intended to reflect the relative organ risks for an average member of a working population. It gave the same weight to a severe hereditary defect in the exposed individual's first two generations of offspring as to the occurrence of a lethal cancer in that individual. It gave zero weight to curable cancer. The concept was appropriate considering the intended use of the quantity. The same quantity has since found widespread use in the assessment of collective doses to members of the public. Here, where its failure to account for the difference between the age distribution of workers and that of the public at large and its non-inclusion of curable cancer and hereditary harm in generations beyond the second are known deficiencies, the use of the effective dose equivalent may be questionable. Various corrections to compensate for these limitations have been suggested, but for the purposes of radiation protection, and considering all other uncertainties, the extensions of the use of the effective dose equivalent have mostly been accepted.

39. In looking for ways of presenting radiation doses from various sources and practices, UNSCEAR faced problems similar to those faced by ICRP. Particularly in the cases of medical exposure and exposure from radon daughter products in the lung, different organs receive quite different doses, and the idea of a weighted whole-body dose was attractive. The Committee is well aware of the fact that the effective dose equivalent has not been designed for its particular purposes, but it has not been able to find an alternative way of expressing radiation exposures by a single number.

40. In the definition of the effective dose equivalent there is an addition of cancer risk and risk of hereditary harm. The risk coefficients for cancer and hereditary harm, as applied to the effective dose equivalent, are clearly identifiable only if all organs receive one and the same dose. In cases where they do not, the effective dose equivalent gives a basis for estimating the total risk but gives no indication of the relative proportions of the cancer risk and the genetic risk (see section II.C).

41. The effective dose equivalent was used in the UNSCEAR 1982 Report, and comparisons were made on the basis of the collective effective dose equivalent commitment. To simplify the presentation of doses and dose comparisons, the Committee has had to resort to more and more complicated terms, and there is, unfortunately, no easy way out of this dilemma.

C. DOSE ASSESSMENTS

1. Natural sources of radiation

42. In preparing its first Report (1958), the Committee concluded that the three main contributors to radiation doses from natural radiation in soft tissues of the human body were cosmic rays, terrestrial gamma-radiation and potassium-40 within the body itself. When the joint dose contribution of these three sources was assessed in the UNSCEAR Reports of 1958-1977, it varied from 93 to 98 per cent of the total absorbed dose from all natural sources, which was estimated to be about 100 mrad per year. The contribution of the three sources were as follows: about 30 mrad from cosmic rays, 30-50 mrad from terrestrial gamma radiation and 20 mrad from potassium-40 in the body.

43. In all UNSCEAR Reports up to and including that of 1972, doses were assessed for three tissues: gonads, osteocytes and active bone marrow. The per caput doses in these tissues were used for dose comparisons in the main text of the Reports. The assessed values varied only a little from one Report to another, with the exception of an overestimate of the dose from the neutron component of cosmic rays in 1962.

44. In the UNSCEAR 1977 Report, the lung dose from radon daughter products inhaled indoors was given in the summary tables, but it did not look so conspicuous since it was presented as an absorbed dose. In 1982, however, the effective dose equivalent was calculated for the first time, and the significance of this contribution became obvious, since it amounted to about one half of the total, as a world-wide average. The assessed value of the annual effective dose equivalent from natural radiation sources was raised accordingly, to about 2 mSv, i.e., to about twice the value implied in previous UNSCEAR Reports, where the lung dose had not been taken into account.

2. Nuclear explosions

45. Most nuclear explosions in the atmosphere occurred before 1963. Their total yields in equivalent amounts of TNT were estimated in the UNSCEAR 1964 Report as follows:

1945-1951	~1 megaton
1952-1954	60 megaton
1955-1956	28 megaton
1957-1958	85 megaton
1959-1960	0 megaton
1961-1962	337 megaton

These numbers have subsequently been somewhat revised in the light of more recent information (see paragraph 143 and Table 5).

46. The atmospheric tests after 1962 were small in comparison with the earlier explosions, and they ceased completely after 1980. The many underground explosions carried out in later years have had few environmental consequences. This temporal picture gives an indication of the environmental situation that prevailed when the Committee prepared its various Reports.

47. Large explosions in the atmosphere carry most of the radioactive material into the stratosphere, where it remains for some time, the mean retention times being estimated from less than a year to about five years, depending on the altitude and latitude. Fallout can therefore occur years after an explosion has injected material into the atmosphere. Smaller explosions carry the radioactive material only into the troposphere, and fallout occurs within days or weeks.

48. When it prepared the UNSCEAR 1958 Report, the Committee did not yet have sufficient information on the global inventory of long-lived radioactive materials to be able to formulate the assessment models used in later Reports. However, the Committee correlated measured fallout rates and deposits with observed radioactive contamination levels in vegetation and food. As explained in section I.B, the quantities that were first assessed were the genetically significant dose and the per caput mean marrow dose, because for these the Committee could make risk estimates.

49. In the first four UNSCEAR Reports (1958-1966), the Committee described in detail the meteorological processes that deplete the stratospheric inventory of radioactive debris. For man, the highest exposure was found to be due to long-lived radioactive material that causes radiation exposures over many years. The dominant radionuclides were strontium-90 (half-life: 28 years), caesium-137 (30 years) and carbon-14 (5,700 years). Some gamma-emitting radionuclides from tropospheric fallout, e.g., zirconium-95 and ruthenium-106, could also contribute significantly through exposure from the ground deposition.

50. Because it was interested in the radiation dose in active bone marrow and in osteocytes, the Committee initially made its most thorough dose calculations for strontium-90. Eventually, however, caesium-137 turned out to cause higher doses because of its double exposure modes: by external gamma-radiation from ground deposition and by internal exposure after intake with food. The exposures from caesium-137 could be verified using direct measurements of the body content, but this was more difficult for strontium-90.

51. With the UNSCEAR 1962 Report, the Committee applied the concept of dose commitment. This made it possible to assess the impact of tests carried out in a particular year or of all the tests up to the time of a Report. In such assessments, however, the contribution from carbon-14 turned out to be high, because of its long half-life. Models for estimating the dose commitment from carbon-14 were developed in the UNSCEAR 1962 and 1964 Reports.

52. In 1964, attention was drawn to the high individual doses caused by enhanced concentrations of caesium-137 in some food chains, in particular the lichen-reindeer chain. This was further discussed in the UNSCEAR 1966 Report, where it was reported that levels of caesium-137 in reindeer meat had in some cases reached 100 nCi/kg (3700 Bq/kg) and in fresh-water fish, 10 nCi/kg.

53. In the UNSCEAR 1969 Report, the mathematical formalism of all calculations was reviewed and the concepts of transfer chains and transfer coefficients were introduced. By the time the UNSCEAR 1972 Report was prepared, the fallout rate had decreased substantially, most of the testing having ceased in 1962. Better estimates could therefore be made of some transfer coefficients, which resulted in somewhat lower dose estimates.

54. In 1977, for the first time, collective dose commitments to most soft tissues of the body from the nuclear test explosions before 1976 were estimated and found to be between 400 and 800 million man rad without the full carbon-14 contribution and about twice as great with the full carbon-14 commitment. For comparison, in the UNSCEAR 1977 Report the annual collective dose to the world population from natural sources of radiation was estimated to be about 300 million man rad.

55. In the UNSCEAR 1982 Report, essentially the same basic information was reviewed. The dose assessment models were then described in a special

Annex, which also listed conversion coefficients, symbols and units. This time the effective dose equivalent was calculated. According to the 1982 assessment, the collective dose contributions from the major radionuclides were as follows:

Radionuclide	*Collective effective dose equivalent commitment (10^6 man Sv)*	
	External	*Internal*
Strontium-90	—	0.5
Zirconium-95	0.6	—
Ruthenium-106	0.2	0.1
Caesium-137	1.5	0.7
Others, except carbon-14	0.2	0.7
Subtotals	2.5	2.0
TOTAL	4.5	

56. One of the main problems in estimating future collective doses is that assumptions have to be made about the size of the population. In deriving estimates in the UNSCEAR 1982 Report, the Committee assumed a world population of 4 10^9 persons when calculating collective doses from radionuclides with half-lives of 10-30 years. The dose commitment from these and from shorter-lived radionuclides was estimated to be about 1 mSv. In calculating the collective dose from carbon-14, the Committee used a world population of 4 10^9 in its 1977 assessment, but a projected population of 10 10^9 in its 1982 assessment. The latter assumption made the estimated collective effective dose equivalent commitment from carbon-14 as high as 26 million man Sv.

3. Nuclear power production

57. In 1970, the world-wide total installed capacity for generating electric energy in nuclear reactors was about 20 GW. Over the next ten years, nuclear electric generation increased by more than 10 GW installed capacity per year, to reach 144 GW in 1981. This rapid introduction of nuclear power on a large scale warranted assessments by the Committee starting with its 1972 Report. Facing a situation similar to that which it had faced with the nuclear explosions, the Committee realized its assessment of future doses would depend on the assumptions it made about the continuation and extension of the practice of nuclear energy generation. It is interesting to note that, at that time, the projections for expansion which the Committee quoted were an order of magnitude higher than turned out to be the case.

58. Thus, in addition to assessing of dose commitments and collective dose commitments per year of practice at the current rate, the Committee therefore also estimated these quantities per unit of electric energy produced, i.e., per MW year. The main contributions to the collective dose commitment were believed to come from global contamination by tritium and krypton-85 released during the reprocessing of spent fuel and from local exposures near the power stations. The total was assessed at about 0.4 man rad/MW year. This value, however, was not used in the summary tables or in the main text of the report. Instead, there was an estimate of the annual per caput dose to the world population if nuclear power production would be maintained at the level expected for year 2000 (an installed capacity of 4,300 GW electric power). This annual dose was estimated to be about 0.2 per cent of the dose from natural sources of radiation.

59. In the UNSCEAR 1977 Report, there was a more systematic approach to assessing the collective dose commitments per unit of electric energy produced for each step of the nuclear fuel cycle (mining, milling, fuel fabrication, reactor operation and fuel reprocessing), including occupational exposures. The estimates made in the UNSCEAR 1977 Report were substantially higher than those made in the UNSCEAR 1972 Report, because more data became available and a fuller treatment was possible. Occupational exposure was estimated to contribute nearly 4 man rad/MW year and exposure of the public between 1.5 and 3.8 man rad/MW year to various tissues. The highest single contribution was again found to come from global distribution due to reprocessing. In the Committee's opinion, these values may be somewhat pessimistic, because the prior experience of reprocessing and research and development, two contributors that were together assessed to cause between 4 and 6 man rad/MW year, may not be able to indicate future experience. The Committee faced a special problem in dealing with the exposures from radon released from uranium mill tailings. This source would cause lung doses that would not be high for any one individual, but the long time period over which radon might emanate from the tailings (determined by the physical half-life of thorium-230) could make the collective dose commitment quite high.

60. The problem posed by radon was recognized more clearly in the UNSCEAR 1982 Report, where the effective dose equivalent was calculated. The various steps in the fuel cycle were together estimated to cause 5.7 man Sv/GW year (0.57 man rem/MW year), excluding global distribution. About 2 man Sv/GW year were estimated to be caused by global distribution from tritium and krypton-85. Occupational exposure was estimated to contribute somewhat less than 30 man Sv/GW year. The total estimate was therefore about 35 man Sv/GW year (3.5 man rem/MW year), somewhat lower than the 1977 estimate.

61. In addition, however, the Committee expected a contribution from the very long-lived radionuclides carbon-14 (half-life 5,700 years) and iodine-129 (1.6 10^7 years); from radon emanation primarily controlled by thorium-230 (8 10^4 years); and from long-lived actinides leaking from high-level waste repositories. With the exception of carbon-14, these nuclides were not expected to cause any significant cumulative collective dose over any 1000-year period (carbon-14, however, would give 10 man Sv/GW year during the first 100 years). But, over 1 million years, assuming a world population of 10^{10} persons, the collective dose from the long-lived radionuclides was estimated at about 3,400 man Sv/GW year:

Radon from mill tailings	2,800
Uranium from mill tailings	460
Carbon-14	110
High-level waste	30
Iodine-129	28

The corresponding doses to any one individual over a lifetime would be negligible, e,g., compared to the doses from natural background radiation, the large numbers being due merely to the long time periods. It is not a scientific question to what extent exposures over such time periods are relevant in decision-making.

62. Using the concept of incomplete (truncated) dose commitment and assuming future annual nuclear energy generation of 10,000 GW years, the Committee finally projected the annual per caput effective dose equivalent to be 25 microsievert i.e., about 1 per cent of the annual dose from natural background radiation.

4. Medical exposures

63. In 1957, when it was preparing the UNSCEAR 1958 Report, the Committee issued an important statement: "It appears most important ... that medical irradiations of any form should be restricted to those which are of value and importance, either in investigation or treatment, so that irradiation of the population may be minimized without any impairment of the efficient medical use of radiation." The statement also solicited further information on medical exposures, which were recognized to constitute a substantial proportion of the total radiation received by mankind.

64. In the UNSCEAR 1958 Report, the Committee gave priority to the assessment of genetically significant dose. It was realized that the highest genetically significant doses were caused by diagnostic x-ray exposures, which, at that time, were frequently carried out with fluoroscopy rather than with radiography. Diagnostic procedures were classified into 23 types, and the exposure data for these were presented for a few countries, permitting comparisons of doses between the various procedures. In addition, crude estimates were made of the per caput mean marrow dose from these procedures. More than 80 per cent of the genetically significant dose was found to be contributed by only six or seven procedures, which together made up only about 10 per cent of all procedures. The data indicated that it might be possible to reduce the doses considerably, simply by careful attention to techniques. The total genetically significant dose from x-ray procedures ranged from 17 to 150 mrem per year in the various national estimates.

65. In the UNSCEAR 1966 Report, the Committee continued its review of the national data that had been submitted. Detailed data were available from 12 countries. The results were similar to those in the UNSCEAR 1958 Report. The values of the genetically significant doses now assessed ranged from 7 to 58 mrem per year. Ways of reducing patient doses were discussed, and the most effective protective measures were listed, such as the use of the smallest possible radiation field and the reduction of fluoroscopy time. This, in effect, was a protection recommendation, released before ICRP had issued any special recommendations on the protection of patients.

66. Medical exposures were next reviewed in the UNSCEAR 1972 Report. The emphasis was still on the genetically significant dose, and the values now assessed ranged from 5 to 75 mrad per year, although the number of x-ray examinations was reported to have increased by between 2 and 6 per cent per year. The Committee felt that, finally, enough information was available from industrialized countries to provide a basis for attempting to eliminate unnecessary exposures. It noted that a large proportion of the world population did not have easy access to modern x-ray facilities and the health benefits they would provide.

67. In the UNSCEAR 1977 Report, the Committee discussed the problems of comparing doses from exposures to sources as diverse as natural radiation, nuclear explosions, nuclear power production and medical exposures. With regard to the latter, the organ doses caused by diagnostic radiology range from a few millirad to a few tens of rad and are usually delivered at high dose rates. The dose distribution is uneven, both within the body and in the population. Moreover, the emphasis that had so far been put on the genetically significant dose might have hidden the possibility of substantial exposures of other organs, so the Committee extended its assessments to include organs other than the gonads and the active bone marrow.

68. In its attempts to find bases for dose comparisons, the Committee looked for, but failed to find, a satisfactory way of combining doses to various organs into some weighted whole-body dose that would be of relevance in cancer risk assessments. As a compromise, in the UNSCEAR 1982 Report, the Committee decided to assess the effective dose equivalent, which, in spite of its shortcomings, best suited its purposes.

69. The 1982 assessment confirmed that medical exposures constitute the largest man-made contribution to radiation doses received by the population and that in some industrialized countries, this contribution approaches the dose received from natural sources. However, the Committee reminded the reader that medical exposures differ from other man-made exposures in that the practice directly benefits those who are exposed. The yearly number of diagnostic x-ray examinations was now found to vary between 300 and 900 examinations per year and per thousand inhabitants in industrialized countries, excluding mass surveys and dental examinations. X-ray examinations contribute the major portion of the collective effective dose equivalent from medical procedures; radiation therapy and nuclear medicine contribute only a minor portion.

70. The Committee expressed disappointment that very little information was available for the two thirds of the world's population that live in countries where radiological examinations are an order of magnitude less frequent than in the more developed countries.

For developed countries, the Committee estimated the annual collective effective dose equivalent from medical procedures at about 1000 man Sv per million of population, i.e., about 50 per cent of the exposure from natural sources.

5. Occupational exposures

71. The Committee discussed occupational exposures in the UNSCEAR 1958, 1972, 1977 and 1982 Reports and pointed out repeatedly that the data that had been submitted were, for a number of reasons, difficult to analyse. The doses reported are those measured by personal dosimeters, and the quantity measured depends on both the type of dosimeter and on its calibration. These recorded doses depend on the location of the dosimeter on the body, and it must be assumed that they approximate a uniform whole-body dose. The number of persons occupationally exposed is not the same as the number of persons monitored, the difference depending on national requirements for radiation monitoring. The objective of most monitoring programmes is not to provide data for purposes such as those of the Committee, but to check that authorized dose limits are not exceeded. So-called investigation levels are usually applied, below which doses are ignored or recorded as zero. Little information is therefore available for the low-dose region.

72. The treatment of the subject in the UNSCEAR 1958 Report was brief. The number of workers in the medical field in countries that had submitted data was estimated to be between 0.2 and 0.7 per thousand of the total population. The treatment of occupational exposures in the UNSCEAR 1962 Report was brief as well. The number of dental workers was found to be about twice the number of medical workers, while the number of persons occupationally exposed in industries or in research was substantially lower. The contribution of occupational exposures to the annual genetically significant dose was estimated at 0.2-0.5 mrem.

73. At the time of the UNSCEAR 1972 Report, there was still very little published data on occupational exposures. The number of workers in the medical field could now be narrowed down to 0.3-0.5 per thousand in the countries for which data were available, and the total number of persons reported as occupationally exposed was 1-2 per thousand of the total population. The mean recorded dose for most workers exposed to radiation was found to be between 0.2 and 0.6 rad per year, but mean doses as high as 2.7 rad were reported from some industrial radiography workers. The annual dose to crews of supersonic aircraft was assessed to be about 1 rem. Occupational exposures in the nuclear power industry were expressed per unit electric energy produced and were calculated to be 2.3 man rad/MW year (1.6 man rad from fuel reprocessing and 0.7 from reactor operation).

74. In the 1977 Report, an Annex was devoted to occupational exposures. For the first time, the Committee systematically reviewed the purposes and methods of assessment. It was found that the distribution of doses within the exposed occupational groups was mostly log-normal, and on this basis a reference dose distribution was defined. To avoid the problems of determining the actual number of workers exposed and therefore, also, average doses, the Committee emphasized collective doses, the values of which would be largely independent of the administrative requirements on the degree of monitoring. The Committee also calculated the fraction of the collective dose accounted for by annual individual doses exceeding 1.5 rad. The submitted data were analysed on this basis. For most occupations, the mean dose was was 0.1-1 rad per year. A detailed mathematical description of the log-normal distribution and of the reference distribution was given. The collective dose from each step of the nuclear fuel cycle was calculated, with the doses from all steps adding up to about 4 man rad/MW year (see section I.C.3). The collective absorbed dose in the lungs of uranium miners was estimated to be 0.1 man rad/MW year, and examples of high radon levels in non-uranium mines were reported.

75. In its 1982 Report, the Committee continued the analysis on the basis of more data. It noted with satisfaction that its 1977 proposal for methods of analysis had been adopted by several organizations and that the arrangement of submitted data had been influenced by the proposal, thus facilitating the analysis. However, the Committee now found that its suggestion of a reference radiation dose distribution had sometimes been misinterpreted, so it limited its presentation to the average dose, the collective dose and the fraction of the collective dose exceeding 15 mSv (corresponding to the previous 1.5 rad).

76. For countries with a high standard of medical care, medical workers were found to receive a collective dose equivalent of about 1 man Sv per million of population. The number of workers in the nuclear industry had increased substantially since 1977. Occupational exposures in each step of the nuclear fuel cycle were assessed more fully, indicating that the total collective effective dose equivalent might be near 30 man Sv/GW year (3 man rem/MW year). However, half of this came from fuel reprocessing and nuclear research, and it was uncertain whether such high contributions should be expected also in the future. In reactor operation, the highest exposures were to maintenance workers and radiation protection staff during special maintenance operations.

6. Miscellaneous exposures

77. In addition to the main radiation sources discussed thus far, a few other sources were identified by the Committee as far back as in the UNSCEAR 1958 Report. Then, as now, they were referred to as miscellaneous sources. Mentioned in the UNSCEAR 1958 Report were watches with radio-luminescent paint, television sets that could produce soft x rays and shoe-fitting equipment that used x-ray fluoroscopy. None of these sources was expected to cause a genetically significant dose exceeding 1 mrem per year, although the shoe-fitting machines could cause high

local doses. The UNSCEAR 1962 Report mentioned enhanced cosmic radiation to passengers in aircraft but considered the dose insignificant. The total genetically significant dose from all miscellaneous sources was not expected to exceed 2 mrem per year, the largest contributor to which was radioactive watches.

78. In the UNSCEAR 1972 Report, a full Annex dealt with the miscellaneous sources. Incidents, transportation accidents and loss of radioactive material were mentioned as additional sources of public exposure. A number of radioactive consumer goods were also described, such as radioluminescent timepieces and other self-luminous devices, ceramic glazes containing uranium, and thoriated electrodes in welding rods. Radioactive substances in patients released from hospitals, pace-makers with nuclear batteries, and demonstration materials in schools were also mentioned. Television sets were again discussed, particularly the colour ones, whose cathode-ray tubes operate on higher voltages. Finally, it was recognized that enhanced levels of natural radiation could cause problems, as, for example, do radioactive building materials. In later Reports this would become an important topic, no longer treated as a miscellaneous source.

79. In the UNSCEAR 1977 Report, the miscellaneous sources were discussed in an Annex dealing with technologically enhanced levels of radiation. One of the many consumer products added to the list was ionization-chamber smoke detectors. However, the discussion centred on enhanced exposures to natural radiation. Enhanced exposures to cosmic rays in aircraft, including supersonic transports, and in spacecraft, were discussed in detail. Another subject was public exposure due to natural radionuclides emitted from coal-fired power plants. A third subject was exposures due to the industrial use of phosphate products containing uranium-238 and radium; in this case, the exposure pathways were via phosphate fertilizers and by the use of waste gypsum as a building material. Normal exposures from radioactive building materials, whether direct (by gamma-radiation) or indirect (by radon daughter products), were dealt with in the discussion on natural sources.

80. In the UNSCEAR 1982 Report, again, miscellaneous sources were considered together with technologically modified exposures to natural radiation. Essentially the same consumer products were discussed as in the previous reports. It was noted that the radium in wrist-watches had now almost entirely been replaced by tritium, thereby eliminating the external exposure and limiting the annual effective dose equivalent to the wearer from leakage tritium to less than 1 microsievert. The average effective dose equivalent to air passengers passing x-ray fluoroscopic scanners was estimated to be much lower still, about 7 nanosievert per scan. Exposures from coal-fired power plants were reassessed and the collective effective dose equivalent commitment was estimated to average 2 man Sv/GW year (this is 50 per cent of the local and regional collective dose from the same energy production in nuclear power stations, see Table 6). The 1977 production of phosphate rock was estimated to have resulted in a collective effective dose equivalent commitment of 300,000 man Sv, predominantly from the use of gypsum in dwellings; the total contribution from other uses was thought to be only 6,000 man Sv.

7. Accidents and incidents

81. The Committee discussed radiation accidents in the UNSCEAR 1962, 1972, 1977 and 1982 Reports. In 1962, it reviewed the eight major accidents known to it at the time; these had caused at least four deaths. Seven of the accidents were criticality accidents (five in the United States, one in the USSR and one in Yugoslavia). The eighth accident involved pulsed x rays from an unshielded electronic tube at a radar station. The course of the accidents and the clinical symptoms of the exposed persons were discussed in some detail.

82. In the 1972 Report, accidents were treated only briefly. The Committee noted that about 100 incidents in connection with the transport of radioactive material had been reported throughout the world from 1954 to 1968. There had been fourteen accidents involving aircraft carrying nuclear weapons or components of nuclear weapons. Two nuclear submarines had disappeared, and a plutonium-238 isotopic generator had burned up in the upper atmosphere. A number of incidents had also been reported wherein radioactive material had been lost or stolen. An analysis of 115 radium incidents occurring from 1966 to 1969 showed that 55 per cent of the incidents were losses. In another study of 299 incidents involving the loss or theft of radium, 66 per cent of the sources were recovered. The same Report also briefly discussed occupational accidents, showing that they had been particularly frequent in x-ray analytical work and in industrial radiography.

83. In the UNSCEAR 1977 Report, the Committee for the first time discussed accidents at nuclear power plants. In its review of the collective dose commitments from the various steps in the nuclear fuel cycle, the Committee approached the difficult problem of dose commitments from accidents that had not yet occurred. Any nuclear power programme is also a commitment to a certain accident probability, so in that sense, the Committee said, there is also an accident dose commitment.

84. In 1982, the Committee observed that there had so far been only two reactor accidents known to have caused measurable irradiation of the public: one at a military plant at Windscale, United Kingdom, in 1957, and one at a nuclear power station at Three Mile Island, Pennsylvania, United States, in 1979. The collective whole-body dose from the latter accident had been estimated between 16 and 35 man Sv within 50 miles, most of it due to xenon-133, and about of equal magnitude outside 50 miles. The collective effective dose equivalent from the Windscale accident had been estimated at about 1,300 man Sv, of which almost half was due to iodine isotopes and thyroid irradiation. The Committee decided that the probabilistic approaches, which predict the risk from reactor programmes by extrapolating into the future, should not be used as a basis for estimating future components of collective dose commitment.

85. In another part of the UNSCEAR 1982 Report, the Committee reviewed information on occupational accidents. It tabulated those accidents on which it had received data or which had been reported in the open literature. The Committee noted that the serious accidents had occurred early in the development of nuclear technology and that not one serious accident had been reported in reactor operation since the mid-1960s. Radiation accidents in other industries had caused one death since 1960; this death occurred in 1975 in an irradiation facility with cobalt-60. As had been noted in the earlier Reports, industrial radiography seemed to have a special potential for accidents. Some severe injuries had occurred when persons picked up lost radiography sources without being aware of the danger.

D. RISK ASSESSMENTS

1. Hereditary harm

86. The methods used so far to quantify genetic risk can be broadly grouped under two headings: the doubling dose (or relative mutation risk) method and the direct (or absolute mutation risk) method. The doubling dose method aims at expressing the risk in relation to the natural prevalence of genetic diseases in the general population; the direct method aims at expressing absolute risk in terms of expected increases in the prevalence of genetic diseases. Owing to the paucity of direct human data on radiation-induced genetic damage leading to disease states, the rates of induction for the pertinent kinds of genetic damage (mutation and chromosomal aberrations) are based on experimental data in animals. These rates are converted, using a number of assumptions and reduction factors, into the expected number of additional cases of genetic disease in man.

87. To apply the doubling dose method, one needs: (a) an estimate of the doubling dose, i.e., the radiation dose that will produce as many mutations as those occurring spontaneously in a given generation; (b) information on the prevalence of naturally occurring genetic diseases in the population and the extent to which these are maintained by mutation; and (c) an estimate of the dose received by the population. Over the years the doubling dose estimates have been based on experimental data obtained in the mouse; the prevalence figures for naturally occurring genetic diseases are those collected in several epidemiological studies. With the doubling dose method, the risk is the product of the prevalence of naturally occurring genetic diseases, the mutation component, the reciprocal of the doubling dose and the dose sustained by the population.

88. Over the past three decades, there have been shifts in emphasis in the use of these methods and there have also been a number of refinements, as extensively discussed in Annex E. The principles that guided UNSCEAR, as well as other scientific bodies, in its early assessments of radiation-induced hereditary risk in the 1950s were those that had emerged from the extensive investigations in Drosophila, the preliminary results in mammals, particularly the mouse, and the sparse human data. Two of these principles were the following: (a) mutations, induced or spontaneous, are generally harmful, and (b) mutations induced by radiation increase linearly with dose without a threshold.

89. In the light of new data from studies on male mice showing that a chronic gamma dose was only about one third as effective as the same dose given at a high dose rate (and even more reduced in female mice), the UNSCEAR 1962 Report suggested that the previously used doubling dose of 30 roentgen would probably be too low by a factor of 3 to 4. With confirmation and extension of these results and other data showing that the interval between irradiation and conception had a dramatic effect on mutation frequency in female mice (all mutations were found in the progeny conceived during the first seven weeks after irradiation), the Committee in 1966 abandoned the doubling dose approach in favour of other methods, two of which will be mentioned here. In one, the estimated rate of induction of dominant visible mutations in mice (range: 10^{-9} to 10^{-7} per locus and rad) was multiplied by the assumed number of loci determining dominant disorders in man (50-500) to obtain the total risk ($5\ 10^{-8}$ to $5\ 10^{-5}$). In the other, the estimated rate of induction of recessive visible mutations in mice (10^{-7} per locus and rad) was multiplied by the estimated total number of gene loci in man (20,000) to obtain an estimate of total risk from the induction of these point mutations ($2\ 10^{-3}$). The risk to first generation offspring was then computed as a fraction (2-5 per cent) of the above figure.

90. In the UNSCEAR 1972 Report the interest of the Committee in the doubling dose method was revived but was given a low profile. The doubling dose was taken to be 100 rad, and the number of extra cases of severe hereditary diseases per million live births and rad of low-LET radiation was estimated to be about 300 for the irradiation of parental males; of these, six to 15 cases occurred in the first generation and the rest occurred in subsequent generations.

91. By 1977 new data on the natural prevalence of genetic and partially genetic diseases had been obtained. Furthermore, data that had been obtained in the mid-1960s on the induction of dominant mutations having their primary effect in the mouse skeleton had been extended in the mid-1970s, demonstrating transmission. By 1982, new data on the induction of another kind of dominant mutation, namely, those that cause cataracts in the eye of the mouse, became available. All these data allowed the Committee to arrive at direct estimates of genetic risks. It is worth noting that from 1977 onwards, both the doubling dose method and the direct method have been used.

92. In 1977, using a doubling dose of 100 rad, the Committee estimated that, if a population is continuously exposed to low-LET radiation at the rate of

1 rad per generation, there will be a total of about 185 cases of Mendelian, chromosomal and other diseases per million live births at equilibrium, of which about one third would appear in the first generation. The first-generation increase was estimated to be about one third of that at equilibrium.

93. These estimates, as well as those arrived at in the 1982 and 1986 Reports, are summarized in Table 1; for convenience, they are expressed on a per Sv basis. It can be seen that (a) for dominantly inherited diseases, the estimates have remained essentially unchanged; (b) the estimates for chromosomal diseases have become lower, this being a consequence of having excluded diseases attributable to numerical anomalies (such as Down's syndrome), for which there is still no good evidence of induction by radiation; and (c) while in 1977 and 1982 the Committee had provided estimates of risk for congenital anomalies and other multifactorial diseases using certain assumptions, in 1986, concerned about persistent uncertainties over the assumptions used, it no longer did so.

94. The risk estimates made using direct methods from 1977 up to 1986, are given in Table 2; they include risks from (a) the induction of genetic changes having dominant effects in the first-generation progeny (i.e., dominant mutations, as well as recessive mutations, deletions and balanced reciprocal translocations with dominant effects) and (b) unbalanced products of balanced reciprocal translocations, which may lead to congenitally malformed children.

95. The first of these estimates (item (a) in the paragraph 94) is based on dominant skeletal and cataract mutations in mice and the second (item (b) in that paragraph) on primate cytogenetic data. The estimates based on experience in mice do not include induced genetic changes so severe as to cause death before they can be detected. It can be seen that the changes in risk estimates from 1977 to 1986 are relatively small. Furthermore, a comparison of these estimates with those arrived at using the doubling dose method (Table 1) for the first generation reveals that they are of the same order of magnitude, in spite of the different assumptions and reduction factors.

Table 1

Estimates of the risk of severe genetic disease per million live births in a population exposed to a genetically significant dose equivalent of 1 Sv per generation of low-dose-rate, low-dose irradiation, according to the doubling dose method
(based on UNSCEAR 1977, 1982 and 1986 Reports)

(The doubling dose equivalent assumed in these calculations is 1 Sv)

Disease classification	Current incidence per million live births	Effect of 1 Sv per generation	
		First generation	Equilibrium
1977			
Autosomal dominant and X-linked	10000	2000	10000
Autosomal recessive	1100	Relatively slight	Very slow increase
Chromosomal (due to numerical and structural anomalies)	4000	3800	4000
Congenital anomalies and other multifactorial diseases	43000 – 47000	450	4500
1982			
Autosomal dominant and X-linked	10000	1500	10000
Autosomal recessive	2500	Relatively slight	Very slow increase
Chromosomal			
Due to structural anomalies	400	240	400
Due to numerical anomalies	3000	Probably very small	
Congenital anomalies and other multifactorial diseases	43000 – 47000	450	4500
1986			
Autosomal dominant and X-linked	10000	1500	10000
Autosomal recessive	2500	5	1500
Chromosomal			
Due to structural anomalies	400	240	400
Due to numerical anomalies	3400	Probably very small	
Congenital anomalies and other multifactorial diseases	60000 – 600000	Not estimated for reasons given in paragraph 186	

Note: The derivation of the above figures is given in Annex E; see also paragraph 93.

Table 2

Estimates of the risk of genetic disease in the first generation (for a genetically significant dose equivalent of 1 Sv) per million live births following low-dose-rate, low-dose exposure of the parental generation according to the direct method
(based on UNSCEAR 1977, 1982, and 1986 Reports)

Risk associated with	Expected frequency of genetically abnormal children in the first generation per million live births after irradiation of	
	Males	Females
1977		
Induced mutations having dominant effects	2000	None given
Unbalanced products of induced chromosomal rearrangements	200-1000	None given
1982		
Induced mutations having dominant effects	1000-2000	0-900
Unbalanced products of induced chromosomal rearrangements	30-1000	0-300
1986		
Induced mutations having dominant effects	1000-2000	0-900
Unbalanced products of induced chromosomal rearrangements	100-1500	0-500

Note: The derivation of the above figures is given in Annex E; see also paragraphs 94-95.

2. Cancer

96. As far back as in the UNSCEAR 1958 Report, the Committee emphasized that any attempt to evaluate the biological effects of radiation sources to which the world population is exposed can produce only tentative estimates, subject to wide margins of uncertainty. Despite these reservations, the Report included assessments of the annual numbers of leukaemia and bone cancer cases that could result from natural radiation and fallout. Data relating the incidence of leukaemia to radiation exposure came mostly from the atomic bomb survivors and patients suffering from ankylosing spondylitis.

97. At that time, the Committee estimated the total probability of leukaemia induction over 15 years to be 12 per million population per rem. It noted, however, that in Hiroshima the probability per unit dose decreased markedly with decreasing dose and that the incidence of leukaemia in that city did not appear to be linearly related to dose. The Committee also made what it called a crude estimate of the leukaemia risk to patients suffering from ankylosing spondylitis who had been treated with x rays. Over 15 years, the risk of induction was estimated to be about 20 per million and rem. Over 35 years, which is the average remaining lifetime of the population and might be the period of risk under conditions of prolonged exposure at lower dose rates, the lifetime risk was assessed to be 52 per million and rem.

98. In discussing the assumed hypothesis of non-threshold linearity between dose and incidence of cancer, the Committee stated in the UNSCEAR 1962 Report that somatic effects were less likely to occur at low dose rates than at the high dose rates employed in many experiments. The only justifications for applying to low doses the relationships observed at higher doses were expediency and the consistency of the assumptions regarding mechanisms in both dose ranges. Nevertheless, the Committee could not say whether, in doing so, it was under- or over-stating the risk. For these reasons, it decided not to estimate absolute risks, but rather to present comparative risk estimates for the gonads (genetic effects), the bone marrow and the cells lining bone surfaces, based on the doses and dose commitments to these tissues from natural radiation sources, medical, occupational and miscellaneous exposure, as well as from nuclear testing.

99. Three basic questions needed to be addressed in the estimation of risk at low dose: the type of effect; the critical tissue for each type; and the function of dose, dose rate and dose distribution to be taken as the relevant parameter for each of the effects. For the somatic effects, the critical tissues were taken to be the active bone marrow and the connective tissue lining endosteal surfaces or trabeculae.

100. Although for genetic effects the experimental data justified an assumption of non-threshold linearity at low doses and dose rates, no such assumption could be made for late somatic effects, because tumour induction at high doses is a very complex function of dose and other exposure factors. Nevertheless, it would be expected that, at low dose levels, the mechanisms by which late effects are produced would

be much simpler and any effects that could arise would result from specific changes induced in individual cells. For certain effects having a non-linear relationship at high dose levels, it was thought probable that the dose-effect curve near the origin would be linear. Thus, protraction of exposure and non-uniformity of dose distribution could be ignored. The Committee also discussed the importance of taking into account the way an effect manifests itself over time.

101. Referring to the problems of obtaining estimates of absolute risk, the Committee noted, in 1964, that it had earlier confined itself to estimating comparative risks except for leukaemia. After having reviewed the available information, the Committee saw no possibility of changing this procedure in the UNSCEAR 1964 Report. It immediately went on to state, however, that data published since 1962 had led it to believe that it would be possible, for a few tissues and mainly in the high-dose range, to make estimates of absolute risk that would be valid for the observed range of doses and the given conditions of irradiation. It was considered unlikely that the risk per unit dose at very low doses would be greater than that at higher doses; in fact, at low doses the risk was likely to be much less.

102. By 1964, tentative dose estimates had become available for some of the survivors from Hiroshima and Nagasaki, and the Committee believed that they were almost certainly not in error by a factor of more than 2 or 3. The new dose estimates made it possible to conclude that the annual incidence of radiation-induced leukaemia was approximately proportional to dose in the range from about 100 to 900 rad, with a proportionality factor between 1 and 2 cases per million and rad. The Committee warned that because the Japanese survivors might have been selected by the lethal effects of the irradiation itself, this estimate of risk could only be applied with caution to the general population. The estimate obtained from the atomic bomb survivors was consistent with that determined from subjects who had been irradiated therapeutically for ankylosing spondylitis, at doses between 300 and 1,500 rad. However, as the latter group was also highly selected, the estimate would apply strictly to spondylitic patients only.

103. New information suggested that for children irradiated in utero, the risk of leukaemia per unit dose could be several times higher than for adults. The doses received had been only a few rad, suggesting that under certain conditions, low doses could induce malignancy. As with the ankylosing spondylitis patients, there was the possibility that the irradiated children might not have been representative of all children.

104. A risk estimate for thyroid cancer was obtained from surveys on the induction of cancer as a result of irradiation of the thyroid region during childhood. In the range 100-300 rad, the Committee estimated the annual risk to be about one per million and rad, over approximately 16 years after irradiation. Once again, the Committee pointed out that the subjects might have been a highly selected group.

105. Irradiation was known to cause other malignancies, including tumours of the bone, liver, skin and lung; however, the information was not considered to be reliable enough for deriving risk estimates. The Committee was not optimistic about being able to obtain such estimates for all, or even many, types of human tissue. Indeed, it concluded that leukaemia might well be the predominant type of malignancy produced, and that the overall risk of all malignancies was unlikely to exceed by any large factor that of leukaemia.

106. In 1972, the Committee decided to review again the subject of radiation carcinogenesis in man. The review pointed out that, in order to assess the extent of radiation effects in man, it was essential to obtain empirical information from epidemiological studies. In evaluating such studies it would be necessary to bear in mind a number of inherent difficulties, such as those having to do with the size of the population studied, the dosimetry, the latent period, the relation to natural incidence of cancer, mortality versus morbidity statistics, the confounding effects of illness and the infrequency of true, uniform whole-body irradiation. The Committee discussed all of these points in detail and also considered the question of absolute and relative risks for the first time. It emphasized that the number of people exposed to substantial doses was so small that the relationship between dose and incidence of malignancies in man could be studied only for the most radiosensitive tissues.

107. Evidence on the induction of leukaemia indicated that its incidence increased with dose in the range 50-500 rad and that above this range the frequency tended to decrease, possibly owing to the cell killing effect of high doses. Radiation-induced leukaemias tended to occur most frequently within a few years of exposure; after 25 years the frequency tended to return to normal, by which time some 15-40 cases per million and rad had been observed.

108. Lung cancers appeared to have been induced at Hiroshima by external gamma exposure at doses of some 30-100 rad. The data indicated a risk coefficient of from 10 per million and rad (at 250 rad) to 40 per million and rad (at 30 rad) during the first 25 years after exposure; this risk estimate was supported to some extent by data from patients treated for ankylosing spondylitis. The Committee noted that an estimate of risk could also be derived from data on uranium miners, but that not much reliance could be placed on such an estimate.

109. The Committee assessed the risk of induction for breast cancer among women exposed in Hiroshima as being between 6 and 20 cases per million and rad during the first 20 years after exposure and over a dose of 60-400 rad. These estimates refer to the 1965 dosimetry. For the induction of thyroid cancers an average risk coefficient was obtained of about 40 per million and rad over a dose of 60-400 rad. For all other malignancies, without clearly identifying their specific types, the Committee tentatively put forward a risk estimate for induction of 40 per million and rad

over the first 25 years after exposure to 250 rad. For a number of reasons, the Committee considered that these risk coefficients were likely to overestimate the risk of environmental exposures, that is, low-dose exposures from both natural and man-made sources.

110. The UNSCEAR 1977 Report also contained a major review of radiation carcinogenesis in man. After dealing extensively with the validity of the data on which risk estimates might be based, the Committee presented its estimates of risk coefficients for leukaemia and tumours in a number of organs. It noted that the risk of a malignancy developing at doses of about 100 rad might vary with the LET of the radiation, sometimes with the age and sex of the subject, and probably with the dose rate and the number of fractions with which the dose is delivered. In that Report the Committee for the first time referred to the induced mortality from leukaemia and other cancers. Previously it had always presented its risk estimates in terms of the incidence of cancer, not in terms of fatality.

111. The thyroid and the breast seemed to have the highest rates of induction, with risk coefficients of around 100 per million and rad. The low mortality rate for radiation-induced thyroid cancers and the moderately low rate for breast cancers were thought to bring the risk of fatality to about one tenth and one half of the incidence values, respectively. Lung cancer also had a high induction rate for males over 35, as judged from the experience of uranium miners. The Committee thought that for lung cancer a mean fatality risk coefficient for all ages of 25-50 per million and rad was probable.

112. The induction of leukaemia, specifically the acute and chronic granulocytic (but not chronic lymphatic) forms, appeared to decrease from about 50 per million and rad at moderately high doses to about 20 per million and rad at lower dose levels. The Committee was rather confident that this estimate would include all the cases likely to appear because, with radiation-induced leukaemia, the average interval between exposure and death appeared to be only about 10 years. With other cancers, which have latent periods of 25 years or greater, it was more difficult to estimate the total number of cases likely to be induced.

113. Risk coefficients were also presented for the stomach, liver and large intestine, brain and salivary glands, all of which had values in the region of 10-15 per million and rad; bone, oesophagus, small intestine, bladder, pancreas, rectum and lymphatic tissue, which had values of 2-5 per million and rad; and skin, for which both the risk of induction and the fatality rate were thought to be low.

114. The Committee also considered the question of estimating the total risk for all fatal malignancies from the observation that this might be four to six times that for leukaemia alone. At doses of a few rad, at which the lower leukaemia risk coefficient of about 20 per million and rad might apply, the total of all fatal induced malignancies, including leukaemia, could be about 100 per million and rad while it was assumed to be about 250 per million and rad at high doses. The risk coefficient for non-fatal malignancies was assumed to be about equal to that for the fatal malignancies. The Committee once again pointed out that the estimate for low doses was derived from mortalities induced at doses greater than 100 rad. The value appropriate to the dose levels involved in occupational exposure, and even more so in environmental exposures, might be substantially less.

115. It was likely that malignancies might be induced by exposure of the foetus in utero at average doses of 0.2-20 rad from diagnostic x rays. The induction rate was difficult to determine with any confidence but was estimated to be around 200 per million and rad.

116. In view of the limited amount of new epidemiological evidence available since the UNSCEAR 1977 Report, and because the dosimetric estimates for the survivors of the atomic bombing of Hiroshima and Nagasaki were in the process of being revised, the Committee decided not to review human carcinogenesis in the UNSCEAR 1982 Report. However, it said that it did not expect that the revisions would change the previous risk estimates by a factor of more than 2. The Committee's risk estimates up to 1977 for cancer are summarized in Table 3 where they are expressed per sievert in order to facilitate comparisons with later estimates.

3. Non-stochastic effects

(a) Irradiation of the adult

117. The Committee considered from time to time the somatic effects of radiation on laboratory animals and human subjects. These effects were first discussed in the UNSCEAR 1958 Report, which attempted to summarize 60 years of knowledge, at a time when information about radiation lesions and their pathogenesis was still rather scanty. Although the Committee had few details on which to base that discussion, the general picture that emerged seemed to be consistent, particularly for the effects induced by high doses. The Committee was aware at that time of the main physical factors affecting the induction of these effects, such as dose, dose rate, fractionation and radiation quality, and it also gave an account of the main biological factors, such as species, age, sex, and partial-body irradiation.

118. The main radiobiological concepts, such as that of cell sensitivity and tissue response, as they manifest themselves in the rate of cell division and differentiation, are to be found in the 1958 Report, although the concept of cell lethality could not be quantified because there were no techniques for single-cell culture. The term recovery was also used in a loose sense, without identifying the many underlying mechanisms. The classification of effects between morphological and functional gave rise to some problems, but the Committee identified, even at that early stage, the difficulties in settling the existence of thresholds, particularly with low doses and late effects.

Table 3

Summary of the Committee's estimates of fatal cancer risk coefficients
(per cent per Sv)

Tissue	1958 Report	1964 Report	1972 Report	1977 Report
Bone marrow	0.2-0.5	0.01-0.02 a/	0.15-0.40	0.20-0.50
Breast	-	-	0.06-0.20	0.50
Lung	-	-	0.10-0.40	0.25-0.50
Thyroid	-	0.16	0.40	0.10
Stomach	-	-		0.10-0.15
Liver	-	-		0.10-0.15
Brain	-	-		0.10-0.15
Salivary glands	-	-		(0.10-0.15)b/
Large intestine	-	-		0.10-0.15
Small intestine	-	-		(0.02-0.05)
Bone	-	-	0.40	(0.02-0.05)
Oesophagus	-	-		(0.02-0.05)
Bladder	-	-		(0.02-0.05)
Pancreas	-	-		(0.02-0.05)
Rectum	-	-		(0.02-0.05)
Mucosa of cranial sinuses	-	-		(0.02-0.05)
Lymphatic tissue	-	-		(0.02-0.05)
Skin	-	-		Low
Estimated total	-	-	-	1.0-2.5

a/ Per year.
b/ Numbers within parentheses refer to total incidence, the fatality risk not having been estimated.

119. Many of the same criteria were used in 1962 in classifying the somatic effects into early and late effects, with the result that effects very different in nature from tumours and leukaemia, such as lens opacification, induction of sterility or non-specific life shortening, ended up being classified together with them just because they also appeared late. The UNSCEAR 1962 Report contained no important departures from the generalizations described above, particularly with respect to the form of the dose-effect relationships, the uncertainties as to the precise form of these relationships at doses below those tested directly, and the pronounced dependence of the effects on the irradiation dose rate.

120. Twenty years elapsed between that Report and the next one, released in 1982, when an extensive Annex discussed the non-stochastic effects of radiation on normal tissues. The new treatment reflected the impressive advances in the understanding of somatic effects that had taken place during the interim. The very title of the Annex implied that there had been a re-classification of the effects into the stochastic and the non-stochastic. To the first class belong those effects for which only the probability of induction is a (linear) function of dose; to the second belong those effects for which severity (as well as probability, for a given severity) is a (sigmoid) function of dose. The Report discussed mainly the effects of irradiation of single tissues and organs; it reviewed a large body of human data interpreted in the light of experience gained in experimental animals.

121. The Committee considered the nature of these effects, their pathogenesis as it results from the interplay of cell killing and tissue kinetics, and the quantitative relationships between them and the time of appearance and degree of the non-stochastic clinical damage. The most general conclusions drawn by the Committee pertained to the existence of a dose threshold for the induction of these effects and the variability of this threshold according to the type of effect. The Annex also contained a detailed analysis of how the dose threshold for each specific type of effect would be expected to vary as a function of the important radiobiological variables such as radiation quality, dose, dose rate, dose fractionation and protraction.

(b) Pre-natal irradiation

122. The earliest mention that the tissues of the embryo and foetus could be particularly sensitive to the action of radiation and that the exposure of pregnant mothers might cause teratological effects to be induced in the product of conception dates from the first UNSCEAR Report (1958). Also, the fact that there are critical periods in development, during which some structures may be particularly vulnerable to the specific action of internal or external irradiation, was already recognized at that time. Finally, it also discussed the shape of dose-effect relationships for effects in utero, without specifying the nature of the effects or their induction mechanisms, although implying that the relationships would be of the threshold type.

123. The UNSCEAR 1962 Report reiterated the notion of the special sensitivity of embryonic and foetal structures, pointing out that minor injuries during development could be amplified by the growth

of the relevant structures to produce major anomalies. From data on the pre-implanted mouse it was inferred that doses of 0.25 Gy to the embryo could be lethal to 40 per cent of the animals. The Committee also concluded, on the basis of the fairly large set of experimental results then available, that irradiation during major organogenesis would cause developmental malformations and that there was a good correspondence between the malformed structures of animals and man for corresponding stages in development. In man, malformations were found more frequently in the central nervous system, the eye and the skeleton.

124. In the context of a special discussion of the effects of radiation on the nervous system, contained in the UNSCEAR 1969 Report, the Committee paid special attention to the damage caused in the brain structures of the developing mammal. It confirmed that pre-natal irradiation during the time when the relevant structures are undergoing differentiation could produce severe developmental anomalies. Depending on the time of the irradiation, specific anomalies (microcephaly, encephalocele, hydrocephalus) could be produced in man, probably following threshold-type kinetics as a function of dose. Disorganization of the cortical architecture was described in animals, accompanied by functional impairment in the form of loss of visual, olfactory and distance discrimination. Other learning processes were impaired in animals after doses of 1 Gy or more had been administered during the second or third week of pregnancy in rats; effects of doses below 0.5 Gy were regarded as uncertain. Although changes in conditioned reflexes had been described in animals irradiated near-term with doses as low as 0.01 Gy, the relevance of these effects to risk estimation in man was also doubtful. In man, the Committee recognized small head size and the induction of mental retardation as true effects, but it could not detect any correlation between such morphological and functional abnormalities and structural changes in the central nervous system. The Committee even ventured to derive a risk coefficient for mental retardation in the survivors of Hiroshima and Nagasaki: 1 per thousand and rad for doses over 50 rad delivered at high dose rates.

125. Recognizing the importance of keeping the effects of radiation on growth and development under observation because of their relevance to the general population and to female workers, the Committee undertook another review of this subject in Annex J of the UNSCEAR 1977 Report. This review centred on experimental animal data, which was the only information available, and on the mechanisms whereby effects are induced in utero; it also described dose-time relationships obtained from the more quantitative data.

126. The Annex J of the UNSCEAR 1977 Report generalized the so-called "periods of maximum sensitivity" of the various anatomical structures, to coincide with the growth spurt; it also generalized across species the notion that lethal effects were typical for the pre-implantation period, teratogenic effects for the major organogenesis period and growth disturbances for the foetal period. An analysis of the dose-effect relationships showed that these were mostly curvilinear. The Committee confirmed its previous risk assessment for mental retardation and suggested, on the basis of mouse data, that the risk coefficient for the increment of embryonic killing soon after fertilization could be taken at 1 per cent per roentgen.

127. From this review the Committee concluded that although data in man on the induction of malformations by radiation were very scarce, the data on other animal species were so unanimous and uniform in indicating a pronounced sensitivity to such effects that the human species could not be regarded as an exception. While the Committee found it impossible, given the paucity of human data, to derive reliable, quantitative estimates of risk from pre-natal human irradiation at comparable developmental stages, particularly at low doses and dose rates it could on the basis of experimental animal data exclude that the sensitivity of the human species might be a factor of 10 higher than expected.

4. Other types of harm

128. At various times and in different Reports, the Committee gave special attention to types of harm not easily classifiable into one of those treated above. One such harm is the shortening of life-span, which was said in the UNSCEAR 1958 Report to result from a number of acute or late radiation-induced changes, both specific, such as leukaemia in radiologists, or pathologically diffused in all organs or tissues. These latter conditions were thought to accelerate the normal aging processes and so were termed non-specific, life shortening.

129. The Committee carried out a special study of the so-called aging effects of radiation and presented the results in the UNSCEAR 1982 Report. There seemed to be insufficient grounds to define aging in precise, biological terms, which would allow postulating non-specific effects of radiation at low doses and dose rates that might cause an animal to prematurely age. The Committee therefore focused on the life-shortening action of radiation, an effect that can be more objectively defined. At the doses of greatest interest for practical purposes, that is, those well below the LD_{50} range and down to the smallest doses and dose rates, evidence showed overwhelmingly that irradiated animals live, on the average, fewer years than non-irradiated controls.

130. This life-shortening effect has precise relationships with dose and time. A very large body of evidence in experimental animals allowed the Report to conclude that at low to intermediate doses and dose rates, life shortening is essentially due to the induction of malignancies at a rate above the natural rate characteristic of the species investigated. This conclusion applies to experimental animals and, as far as could be judged from limited human experience, also to man.

131. In the UNSCEAR 1969 Report, the Committee presented a special study of the effects of radiation on

the nervous system. That review also covered aspects of morphological and functional disturbances produced by irradiation during the pre-natal stages. Irradiation of the nervous system can cause effects in adults only at high doses, in which case there are profound structural and functional alterations. It was recognized, however, that for doses as low as 0.1 Gy or less, reactions of a "physiological nature" could be induced. The most remarkable finding remained the striking difference in sensitivity between the pre- and post-natal stages, the former being much more vulnerable than the latter.

132. The same Report contained a separate Annex on the induction of chromosomal aberrations in human germinal and somatic cell lines. The induction of chromosomal aberrations in somatic cells is an interesting effect by virtue of its potential use as an in vivo dosimeter and its biological significance with respect to the causation of (or correlations with) induction of malignancies. The Annex covered in depth the dose-time relationships for the induction of chromosomal damage and the variability of aberrations as a function of other physical and biological agents. It concluded that, aside from its practical applications in biological dosimetry, chromosomal analysis could be of little use in assessing the risk of neoplastic, immunological or life-shortening effects of radiation. Risk estimates would continue to be based on the observed incidences of the specific clinical conditions as a function of dose, a conclusion that remains true to this day.

133. The UNSCEAR 1972 Report contained a special study on the effects of radiation on the immune response wherein the Committee, mostly on the basis of experimental data, tried to discuss the role the immune system plays in the development of early and late radiation effects, essentially those of the non-stochastic type. The study concluded that the immune system has large, built-in safety factors that allow it to withstand and recover from substantial injury by radiation. The Committee reported that at whole-body doses around 0.1 Gy, damage to the immune system could be observed but that such damage did not cause great concern. Whole-body doses higher by an order of magnitude could increase susceptibility to infection, while doses of 2 or more Gy could significantly increase the risk of mortality from infection. For non-stochastic effects, these conclusions still appear to be valid.

134. Another special study was carried out of the possible interaction between radiation and other agents that are widely distributed in the environment. This study too, was contained in the UNSCEAR 1982 Report. In it, the Committee paid particular attention to exposure conditions that affect large numbers of people, thereby substantially changing average risk coefficients.

135. The Committee found that for effects of wide practical significance (induction of cancer, genetic effects or developmental abnormalities), there was little systematic information to substantiate claims of non-additive interactions between radiation and other agents. The theoretical analyses, which were accompanied by illustrative examples from experimental or epidemiological work, treated this matter in all its complexity: The different natures of the interacting agents, their different mechanisms of action, the different dose levels and the different ways of administering the doses could all give rise to a variety of possible interactions, in the additive, inhibiting or synergistic sense, but only one case of synergism appeared to be well documented, that between tobacco smoke and radon decay products in uranium miners. This synergism prevents the direct extrapolation of findings in the miners to the general population.

II. THE PRESENT SITUATION

136. This chapter describes the Committee's findings and conclusions in its most recent Reports. For the most subjects the latest account is the one contained in the present (1988) Report, but for some subjects that are not reported here, e.g., exposures from nuclear explosions, the latest account is contained in the UNSCEAR 1982 Report.

A. RADIATION LEVELS AND DOSES

1. Natural sources of radiation[d]

137. The assessment of the radiation doses in humans from natural sources is of special importance because natural radiation is by far the largest contributor to the collective dose received by the world population. The natural radiation sources are classified into: (a) external sources of extraterrestrial origin (that is, cosmic radiation) and radiation of terrestrial origin (that is, the radioactive nuclides present in the crust of the earth, in building materials and in air) and (b) internal sources, comprising the naturally occurring radionuclides that are taken into the human body.

138. Some of the contributions to the total exposure from the natural radiation background are quite constant in space and time and practically independent of human practices and activities. This is true, for example, of the doses received from the ingestion of potassium-40, an element that is homeostatically controlled and also of doses from the inhalation and ingestion of cosmogenic radionuclides, which are relatively homogeneously distributed over the surface of the globe. Other contributions depend strongly on human activities and practices and are therefore widely variable. The doses from indoor inhalation of radon and thoron decay products are examples: building design, as well as the choice of building materials and of ventilation systems, influences the indoor levels, so that as techniques and practices evolve, the doses received from radon will also change. Between those extreme types of exposure, there are some intermediate types: external doses from cosmic rays, which are affected by human practices and are quite predictable but uncontrollable (except by moving to an area where the dose is lower); doses from the inhalation and ingestion of long-lived nuclides of the uranium-238 and thorium-232 decay series, which make a small contribution to the total dose from natural sources and are relatively constant in space; and doses from external irradiation by terrestrial sources, which are also significantly altered by human activities and practices, especially through indoor exposure.

139. The Committee has re-assessed the doses received globally from natural radiation sources (Table 4). The

[d]This subject is reviewed extensively in Annex A, "Exposures from natural sources of radiation".

Table 4

Annual effective dose equivalent from natural sources

Source of irradiation	Annual effective dose equivalent (mSv)		
	External	Internal	Total
Cosmic rays			
Directly ionizing component	0.30	-	0.30
Neutron component	0.055	-	0.055
Cosmogenic radionuclides	-	0.015	0.015
Primordial radionuclides:			
Potassium-40	0.15	0.18	0.33
Rubidium-87	-	0.006	0.006
Uranium-238 series:	0.1		1.34
Uranium-238 to uranium-234		0.005	
Thorium-230		0.007	
Radium-226		0.007	1.24
Radon-222 to polonium-214		1.1	
Lead-210 to polonium-210		0.12	
Thorium-232 series:	0.16		0.34
Thorium-232		0.003	
Radium-228 to radium-224		0.013	0.18
Radon-220 to thallium-208		0.16	
Total	0.8	1.6	2.4

mean annual effective dose equivalent is estimated to be 2.4 mSv; it refers to the adult part of the population. Variation around this mean is due mainly to variations in the external exposure to terrestrial sources and in the internal exposure (inhalation) to short-lived decay products of radon isotopes. The external exposures typically vary around the mean by a factor of 1.5 and the internal ones by a factor of 2.5. For both types of exposure, the extreme values vary around the mean by a factor of 100.

140. There are several changes from the estimates given in the UNSCEAR 1982 Report: (a) for external exposure to cosmic radiation, the new estimate of the annual effective dose equivalent is higher by 50 microsievert, from taking into account the geographical distribution of the world population as a function of altitude as well as the shielding effect of the building materials; (b) for external exposure to terrestrial sources of radiation, the estimate of the annual effective dose equivalent has been raised by 60 microsievert as a result of a better knowledge of the indoor gamma absorbed doses in air; (c) the estimates of the annual effective dose equivalents from internal exposure to primordial radionuclides have been slightly decreased for the uranium-238 and lead-210 series as well as for the decay products of radon-220, whereas those for the short-lived decay products of radon-222 have been increased by about 300 microsievert on the basis of the results of nation-wide indoor surveys. The net effect of these corrections is a 20 per cent increase in the estimate of the annual effective dose equivalent from all natural sources of radiation.

141. Table 4 shows the paramount importance of doses from the inhalation of radon-222 and its short-lived decay products. Industrial activities that release materials with enhanced concentrations of naturally occurring radionuclides do not significantly alter the overall exposure estimates.

2. Nuclear explosions

142. In the UNSCEAR 1982 Report, the Committee assessed the exposures to the world's population from the release to the environment of radioactive materials produced in nuclear explosions carried out in the atmosphere since 1945. Since no atmospheric nuclear tests have taken place since 1980, the assessment remains complete and valid.

143. The number and yield of atmospheric nuclear explosions are summarized in Table 5, which shows that the most test programmes took place during 1957-1958 and 1961-1962. Large-yield explosions carry radioactive debris into the stratosphere, from where it is dispersed and deposited around the world (this is known as stratospheric radioactive fallout). Exposures to populations are highest in the temperate regions and in the northern hemisphere, where most of the testing occurred. The dose commitment for the southern temperate zone is about 70 per cent of that for the northern temperate zone. The radiation doses are due mostly to the ingestion of radionuclides that have become incorporated in foods and to external irradiation from ground deposition.

Table 5

Number and yield of atmospheric nuclear explosions

Year	Number	Estimated yield (Mt)	
		Fission	Total
1945-1951	26	0.8	0.8
1952-1954	31	37	60
1955-1956	44	14	31
1957-1958	128	40	81
1959-1960	3	0.1	0.1
1961-1962	128	102	340
1963	0	0.0	0.0
1964-1969	22	10.6	15.5
1970-1974	34	10.0	12.2
1975	0	0.0	0.0
1976-1980	7	2.9	4.8
1981-1987	0	No further tests	

144. The most significant radionuclides contributing to the assessed dose commitments for various parts of the world from all atmospheric tests carried out so far are, in decreasing order of importance: carbon-14, caesium-137, zirconium-95, strontium-90, rubidium-106, cerium-144 and tritium. Residual irradiation from only four of these, carbon-14, caesium-137, strontium-90 and tritium, remains to be received by the present and future world population. An additional contribution of about 0.1 per cent of the total effective dose equivalent commitment will be received from plutonium-239, plutonium-240, and americium-241 at very low dose rates over thousands of years.

145. The collective effective dose equivalent commitment due to all atmospheric nuclear explosions was estimated in the UNSCEAR 1982 Report to be $3\ 10^7$ man Sv, an estimate that is still valid. This value, which takes into account projected future growth of the population of the world, was found to be equivalent to about four years of exposure to natural sources for the population of the late 1970s, on the basis of an annual per caput exposure to natural sources of 2 mSv and a world population of $4\ 10^9$. Owing to the increase in the world population to about $5\ 10^9$ at the present time and to the revised estimate, 2.4 mSv, for the annual per caput exposure to natural sources, the collective effective dose equivalent commitment due to all atmospheric nuclear explosions is now assessed to be equivalent to about three years of exposure to natural sources for the present population.

3. Nuclear power production[e]

146. The number of nuclear reactors being operated to generate electricity has increased since the UNSCEAR 1982 Report. At the end of 1987, the 417 reactors operating in 26 countries had an installed capacity of 298 GW. This represents a 100 per cent increase in capacity since the Committee last reported in 1982, when installed capacity was 144 GW. Projections to

[e]This subject is reviewed extensively in Annex B, "Exposures from nuclear power production".

the year 2000, although still somewhat speculative, amount to around 500 GW, a further growth of 80 per cent from present capacity.

147. The nuclear fuel cycle includes several steps: mining and milling of uranium ores; enrichment of the isotopic content of uranium-235 for some types of reactors; fabrication of fuel elements; production of energy in the reactors; reprocessing (although this is not always undertaken) of irradiated fuel and recycling of the fissile and fertile nuclides recovered; transportation of nuclear materials between fuel cycle installations; and, finally, the disposal of radioactive wastes. Although most of the radioactive materials associated with nuclear power production are present in the irradiated fuel, small amounts are released to the environment in effluents at each of the steps in the cycle. Most of these releases are only of local and regional concern, because the radionuclides have short half-lives and are limited in their environmental mobility. However, some nuclides, because of their long half-lives or rapid transfer through the environment, may contribute to the irradiation of man on a global scale.

148. For each step in the fuel cycle and its associated release of radioactive materials, the Committee has evaluated the doses to workers within nuclear installations and to members of the public. In its evaluations, four population groups have been considered: those exposed in normal conditions because of their work within the fuel cycle; the population living within about 100 km of the plant; the population within a few thousand kilometres; and, finally, the world population.

149. The concentrations of radionuclides in effluents are generally low, and it is hardly feasible and not practicable to monitor members of the population for uptake of radionuclides. Instead, environmental modelling has been used by the Committee to estimate doses at long distances from the plant. The transfer of radionuclides through environmental media can be predicted from measured values obtained by monitoring foodstuffs and water and from experimental studies.

150. The starting point for environmental modelling at long distances is data on the quantities and composition of radioactive materials emanating from various nuclear installations. This information is usually available to the Committee from those countries having nuclear power programmes and has been collected for the six-year period 1980-1985. Since the size of a particular stage in the nuclear fuel cycle is proportional to the nuclear generating capacity served by the stage, the releases have been normalized per gigawatt year of generated electric energy, enabling comparisons to be made and to facilitate the use of averages over all plants of a similar conceptual design; the results are not representative of a specific site, but they do give an idea of the impact of each type of facility. Averaging over all energy production and for all plants of a particular type accounts also for releases that may arise during maintenance shutdowns, when little or no electricity is generated.

151. To assess the collective doses corresponding to the normalized releases, the Committee had previously specified hypothetical sites with broadly representative characteristics for each stage of the fuel cycle: mining and milling, enrichment and fabrication, reactor operation and reprocessing. The Committee also assumed that the environment receiving the releases from each model facility was a hypothetical environment containing the main features of existing sites, so that the most common pathways to man are included. The Committee has used the same models again because it believes they are still adequate for the purpose and because doing so allows the current impact to be compared with the previously assessed impact of 1974-1979.

152. Uranium mines give rise to effluents, which when operating consist mainly of ventilation air in the case of underground mines and of releases into the pit in the case of surface mines. Further effluents are produced during milling operations to extract the uranium. The stockpiles of ore and other extracted materials are the source of airborne emissions when the mine is operating, and this source persists even after the mine has been closed. The tailings that are discharged from the mills also become long-term sources of airborne emissions. The most important radionuclide in all these airborne releases is radon-222. Using the same general models as in the UNSCEAR 1982 Report doses have been assessed both for the operational period and for the long term (10^4 years). Doses from fuel fabrication and transport have also been assessed, but since these are so much smaller than the doses from other components of the nuclear fuel cycle, they are not considered separately.

153. During operation of nuclear power stations and reprocessing plants, solid wastes are produced and have to be disposed of. For purposes of analysis, these wastes have been characterized in terms of volumes and activity concentrations of important radionuclides per unit energy generated. Two typical disposal facilities of the shallow land burial type were specified and terrestrial dispersion models used to calculate the release rates of radionuclides and the resulting effective dose equivalents.

154. The only operating commercial fuel reprocessing plants are at Sellafield in the United Kingdom and at Cap de la Hague and Marcoule in France. The Committee assessed in the UNSCEAR 1982 Report the impact of reprocessing using a notional plant representative of plants that would be reprocessing oxide fuel in the future. At present the throughput of fuel at the three reprocessing plants represents an energy output equivalent to about 5 per cent of that generated by nuclear power. The Committee has therefore decided to assess the impact of the actual reported discharges from these commercial reprocessing plants and weight the resulting collective doses by the fraction of fuel reprocessed to obtain values of exposure per GW year generated.

155. Calculations of collective dose to the world's population and various subgroups require assumptions to be made about the size of these populations, their

dietary and other habits, and agricultural and fishing practices. The broadly representative values of these parameters previously used by the Committee have been retained to evaluate the radiological impact of each stage of the fuel cycle.

156. The estimates of collective effective dose equivalent to local and regional populations and to the global population from widely dispersed radionuclides are given in Table 6. Occupational exposures per GW year are approximately three times those received by the local and regional population.

157. Estimates of dose to the public have been reduced, partly because discharges to the environment from reactors have generally decreased and also the estimate for carbon-14, which accounts for half the public exposure from routine reactor releases, is much lower than the estimate in the UNSCEAR 1982 Report due to new, lower measured values of carbon-14 releases from heavy-water reactors.

158. The annual exposure received by the world's population from the release of radionuclides that become globally dispersed is currently much less than that received by local and regional populations. Only if the current levels of discharge of these radionuclides continued and all fuel from all reactors were reprocessed could the global component of the annual collective effective dose equivalent eventually equal the local and regional components.

159. The collective and per caput doses from nuclear power production may be compared to the doses to the world population from natural sources of radiation. The more immediately delivered component of the normalized collective effective dose equivalent commitment has been estimated to be 4 man Sv per GW a from radionuclides in the effluents of nuclear fuel cycle installations. For the present annual nuclear power production of about 190 GW year, the annual collective dose is assessed to be 760 man Sv. Dividing by the world population of $5\ 10^9$ gives an annual per caput dose estimate of 0.15 microsievert. The doses are around 0.01 per cent of the collective and per caput doses from natural background sources.

4. Medical exposures

160. Good data on the frequency of examinations and absorbed doses from medical examinations come mainly from the well-developed countries, which comprise less than 25 per cent of the world's population. There are fragmentary data on examination rates or number of diagnostic units and little or no data on absorbed doses for approximately another 25 per cent of the population. For 50 per cent of the world's population there are no data at all. For this reason, the Committee has developed a modelling approach based upon the good correlation that exists in most countries between population per physician (about which there is more information) and the medical uses of radiation.

161. Access of populations in the world to radiodiagnosis is very uneven: one x-ray machine is shared by fewer than 2,000 people in some countries and by 100,000-600,000 people in other countries. The frequency of procedures is also very uneven: 15-20 procedures per year are carried out per 1,000 population in some countries and 1,000-2,000 procedures per year in others. At the present time, there are about $5\ 10^9$ people in the world, and some estimates are that more than three quarters of the world's population have no chance of receiving any radiological examination, regardless of what disease they have.

162. While absorbed dose data exist for many standard radiographic and nuclear medicine procedures, information now available suggests that the previous absorbed dose estimates for the world population may be somewhat low. An important reason for this is the widespread use of fluoroscopy in developing countries. There are also large numbers of malfunctioning machines, which produce high doses. Neither of these factors was widely appreciated in the past.

163. The collective effective dose equivalent from diagnostic x-ray procedures is far greater than that from dental or diagnostic nuclear medicine examina-

[f] This subject is reviewed extensively in Annex C, "Exposures from medical uses of radiation".

Table 6

Collective dose per unit practice of nuclear power generation
(man Sv per GW a)

	Over next 100 years	Over all time
Mill tailings (radon), long term	1.5	150 a/
Globally dispersed nuclides and waste	6	60
Local and regional exposures	4	4
Occupational exposures	12	12
Total	24	230

a/ Over 10,000 years.

tions. The per caput annual effective dose equivalent is likely to be no lower than 0.4 mSv (the Committee's previous estimate) and may be as high as 1.0 mSv. Similarly, the annual genetically significant dose may range from 0.1 to 0.3 mSv. However, considering the age structure of the population, the effective dose equivalent may overestimate the detriment. This would be particularly true in countries where the older portion of the population receives most of the medical irradiation.

164. The world-wide collective effective dose equivalent is estimated to be between 2 and 5 10^6 man Sv. Of this, 90-95 per cent is attributable to diagnostic x-ray procedures. Dental radiography, nuclear medicine and radiation therapy (ignoring target doses) together contribute only 5-10 per cent of the collective dose. In developed countries, the contribution to the collective effective dose equivalent is about 0.001 man Sv per examination.

165. There are many possibilities for reducing dose without jeopardizing the benefits of the radiological practices. In the developed countries, it may be possible to reduce the per caput effective dose equivalent by half. In the less-developed countries, the use of radiography rather than fluoroscopy, appropriate collimation, proper film developing, as well as the calibration and maintenance of equipment, would reduce the dose per examination; however, the feasibility and costs of these measures are not known. The genetically significant dose can be significantly reduced through the use of gonadal shielding, a practical, low cost method. Still, the collective effective dose equivalent may increase as x-ray examinations become more widely available in a number of countries, and such an increase may in fact be appropriate.

166. The frequency and total use of medical irradiation is expected to increase over the next several decades because of the aging of the world's population, the growth of this population, and urbanization in the developing countries. By the year 2000, the collective dose will probably have increased by 50 per cent, and by 2025 it may have more than doubled.

5. Occupational exposures[g]

167. Two categories of workers are exposed to radiation: workers in the nuclear industry and in the medical field, where radiation sources are managed, and workers in occupations where higher background radiation levels are encountered (air crews and non-uranium miners are examples). The Committee gave a full assessment of occupational exposures in the UNSCEAR 1982 Report. Updated estimates of exposures to workers in nuclear fuel cycle activities (average annual doses in the range of 3 to 8 mSv for reactor operation, and a collective dose of 12 man Sv for each GW year of electric energy generated, in total for all work in the whole nuclear fuel cycle, cf. Table 6) and to medical personnel (average annual doses in the range of 0.3 to 3 mSv, and a collective dose of about 1 man Sv per million of population, cf. also paragraph 166; in developed countries an average occupational dose of about 1 microsievert per examination) are included along with exposures of the general public in the respective Annexes dealing with these subjects.

168. Exposures of radiation workers are subject to detailed regulatory control in all countries and in the majority of cases the doses are but a small fraction of established limits, partly as a result of the current emphasis on optimizing radiation protection. The collective effective dose equivalent commitment per unit of electricity generated to workers in all nuclear fuel cycle installations is estimated to have changed little from the commitment previously estimated by the Committee, but such stability is only to be expected if reductions in exposures are balanced by the greater numbers of workers employed in the expanding industry.

169. Occupational exposure from medical practices includes the contributions from diagnostic x-ray procedures, dental radiography, nuclear medicine and radiation therapy. The average annual collective effective dose equivalent from occupational exposures in these practices is about 1 man Sv per 10^6 population. In spite of the increase in the medical uses of radiation in most countries, the limited trend data indicate that both individual and collective annual occupational doses are decreasing by 10-20 per cent every decade. For developed countries, the average occupational exposure is about 1 microsievert per examination.

6. Miscellaneous exposures

170. Exposures from miscellaneous sources of radiation are evaluated by the Committee whenever warranted by new information or new developments. The latest assessment, in the UNSCEAR 1982 Report, dealt with various consumer devices that contain radioactive materials and with electronic and electrical equipment that emit x rays. Individual exposures to these various sources were generally very small. The Committee believes that assessment to be still valid and feels that no new evaluation is required.

7. Accidents

171. With the large size of the nuclear industry in some countries and the large number of radiation sources used for industrial and medical purposes, accidents are bound to happen. The accidents that have occurred have generally been criticality and other industrial accidents that exposed one or a few workers; transport accidents, including also accidents involving satellites, aircraft and submarines; losses or thefts of radiation sources; and reactor accidents.

172. Three reactor accidents have caused measurable exposures of the public: Windscale in 1957, Three Mile Island in 1979, and Chernobyl in 1986. The

[g] This subject is reviewed in Annex B, "Exposures from nuclear power production" and in Annex C, "Exposures from medical uses of radiation".

Chernobyl nuclear reactor accident was a significant event and is discussed in detail in two Annexes (Annex D, "Exposures from the Chernobyl accident" and Annex G, "Early effects in man of high doses of radiation").

173. In all, six notable accidents have occurred since 1982, when the Committee last dealt with this subject:

1983: Constituyentes, Argentina. An accidental prompt critical excursion occurred during a configuration change in a critical assembly, resulting in the death of an operator, who was only 3-4 metres away. The dose to the victim was estimated to be 5-20 Gy from gamma rays and 14-17 Gy from neutrons.

1983: Ciudad Juarez, Mexico. An improperly disposed of cobalt-60 source found its way into a scrap metal shipment, contaminating the delivery truck, the roadsides and the processed steel into which the scrap was incorporated. Some 300-500 individuals were exposed, ten to doses of 1-3 Gy. There were no deaths.

1984: Mohammedia, Morocco. A source of iridium-192 used to make radiographs of welds at a construction site became detached from the take-up line to its shielded container. The source dropped to the ground and was noticed by a passer-by, who took it home. Eight persons, an entire family, died from the radiation over-exposure with doses of 8-25 Gy.

1986: Texas, United States. An accident at a linear accelerator caused two deaths from over-exposure.

1986: Chernobyl, USSR. The accident at the nuclear power station resulted in two immediate deaths of reactor operating personnel from the explosion. About 145 firemen and emergency workers suffered acute radiation sickness; 28 of them died during the three months following the accident. There were 30 deaths in all; one worker died from mechanical injury and one from burns. Local residents, none of whom received high exposures, were evacuated. The widespread dispersion of the released materials caused low exposures, primarily to populations of the western part of the USSR and other European countries.

1987: Goiania, Brazil. A caesium-137 source was dismantled in a residential area causing some 240 people to become contaminated. Fifty-four of them were hospitalized and four died.

8. The Chernobyl accident[h]

174. The accident at the Chernobyl nuclear reactor in the USSR, which occurred on 26 April 1986, caused extensive contamination in the local area and resulted in radioactive material becoming widely dispersed and deposited in European countries and throughout the northern hemisphere. The extent to which such a wide region could be affected by an event of this type was unanticipated. Intensive monitoring was undertaken to evaluate the radiation levels.

175. It was apparent soon after the arrest of releases from the reactor that the radiological impact of the accident, from the point of view of individual risk, would be insignificant outside a limited region within the USSR, either because contamination levels were generally low or because remedial actions to ban the consumption of particularly contaminated foodstuffs prevented high exposures.

176. The accident at the Chernobyl reactor occurred in the course of a low-power engineering test, during which safety systems had been switched off. The uncontrollable instabilities that developed caused explosions and fire, which damaged the reactor and allowed radioactive gases and particles to be released into the environment. The fire was extinguished and the reactor core sealed off by the tenth day after the accident.

177. The death toll within three months from the accident was 30 members of the reactor's operating staff and the fire-fighting crew. Two died immediately, 28 died from radiation injury. Radiation doses to the local population were well below the doses that could cause immediate effects. Local residents were evacuated from a 30 km exclusion zone surrounding the reactor. Agricultural activities were halted and a large-scale decontamination effort has been undertaken.

178. The initial release of radioactive materials from the accident spread with winds, in a northerly direction. Subsequent releases dispersed towards the west and south-west and in other directions as well. Deposition on to the ground was governed primarily by rainfall, which occurred sporadically at the time in Europe. The deposition pattern and the associated transfer of radionuclides to foods and irradiation of individuals was very inhomogeneous, necessitating a regional approach for dose calculations.

179. Measurements since the accident have shown that the radionuclides contributing most significantly to doses are iodine-131, caesium-134 and caesium-137 mainly by external irradiation from deposited material and by ingestion of contaminated foods. The Committee's dose assessment takes most account for these important radionuclides and pathways.

180. Detailed information was available to the Committee to calculate first-year radiation doses in the USSR and all European countries. To extend these results and to estimate the projected doses from deposited materials, wider regions were evaluated. Since there is insignificant interhemispheric mixing of material released into the troposphere, southern hemisphere countries could only have been affected through imported food; this possibility is accounted for in the assessment by considering total food production as well as local consumption in northern hemisphere countries.

[h] This subject is reviewed extensively in Annex D, "Exposures from the Chernobyl accident".

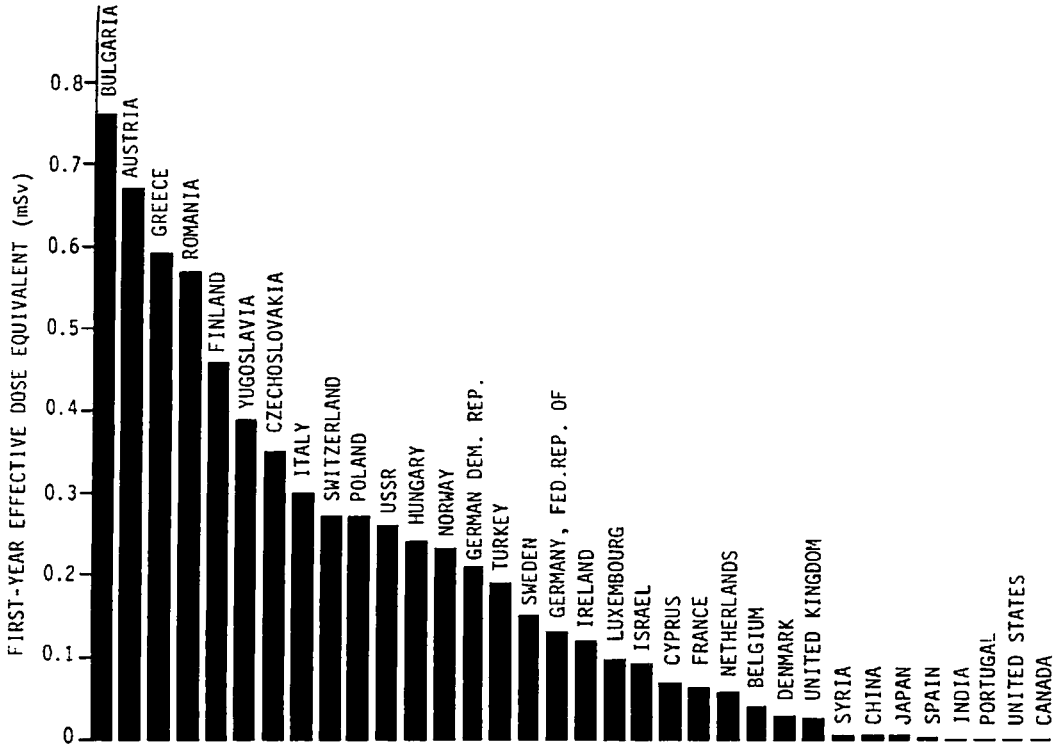


Figure I. Country average first-year committed effective dose equivalent from the Chernobyl accident.

181. The input values for the calculation made full use of measurements during the first year following the accident. Thereafter, projections are required to estimate the further contributions to dose, primarily from ^{137}Cs. The projections are based on experience acquired from past studies of radioactive fallout from the atmospheric testing of nuclear weapons.

182. The results of calculations of the first-year committed effective dose equivalents in 34 countries are illustrated in Figure I. The highest values are for Bulgaria, Austria, Greece and Romania, followed by other countries of northern, eastern and southeastern Europe. Countries further to the west in Europe and also countries of Asia, North Africa, North and Central America were less affected, which is in accord with the deposition pattern.

183. The dose commitments from the accident are delivered over several years, mostly due to continuing exposures from caesium-137. On average, some 30 per cent of the effective dose equivalent commitments were delivered in the first year following the accident. The dose commitments over all time in wider regions of the world are illustrated in Figure II.

184. The main outcome of the dose assessment is the collective effective dose equivalent commitment. This is estimated to be approximately 600,000 man Sv. Of this amount, 40 per cent will be received in the USSR

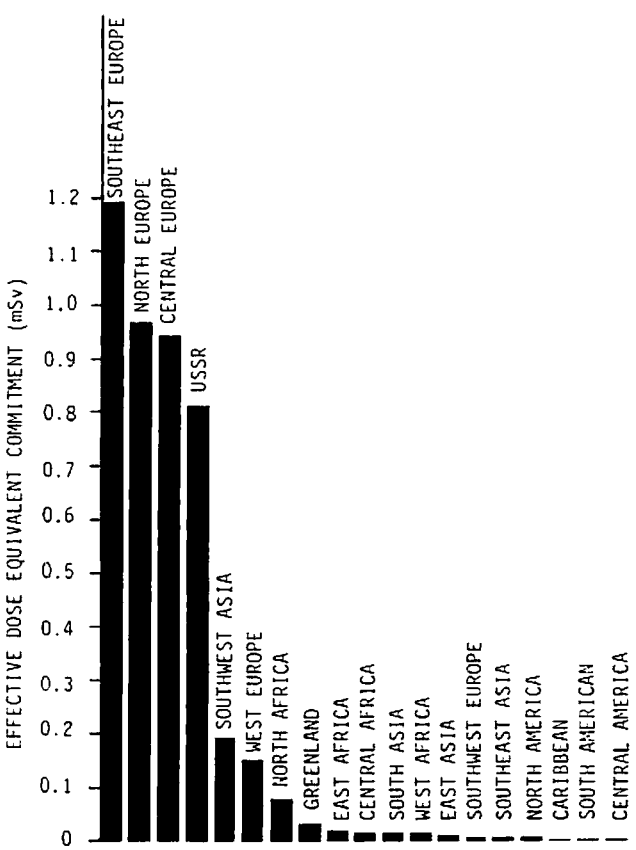


Figure II. Regional average effective dose equivalent commitment from the Chernobyl accident.

and 57 per cent in the rest of Europe. The remaining 3 per cent will be received by other countries of the northern hemisphere.

185. For comparison with Figure I, the one year effective dose equivalent from natural sources is 2.4 mSv. For comparison with Figure II, it should be noted that most of the dose commitment will be received within 30 years of the accident. The 30-year effective dose equivalent from natural sources is about 70 mSv. In using these comparisons, it should be remembered that the doses are averages over large geographical areas within which there will be local variations, in the doses from Chernobyl and those from natural sources.

B. RADIATION EFFECTS

1. Hereditary harm[i]

186. In spite of the considerable progress made during the past few years in understanding the mutation process, there have been no major conceptual changes in the formulation of risk estimates between the UNSCEAR 1986 Report and the present one that would warrant revising the estimates of natural or radiation-induced Mendelian and chromosomal disorders using the doubling dose method. However, an attempt has been made to quantify risks of induction of recessive diseases by this method. New data on the prevalence of congenital anomalies and other disorders of complex aetiology (discussed in 1986) raise a number of questions: Can the doubling dose of 1 Gy be confidently applied to disorders of complex aetiology? What is the magnitude of the mutational component of these disorders? Is it meaningful to provide estimates for these disorders in the continuing absence of experimental or human data bearing on the mechanisms of their maintenance in a population and on their possible response to radiation? Until new data become available, the Committee concluded that it was unable to provide meaningful risk estimates for these disorders. However, even with extreme assumptions (e.g., a 100 per cent mutational component) the risk of severe hereditary harm in the first generation of offspring to the exposed individual does not appear to be higher than the present estimate of the cancer risk. Since this situation remains true in 1988, the risk estimates for hereditary effects that the Committee offers at the present time are those shown in Table 7.

187. Using direct methods, the Committee estimated 10-20 per 10^{-2} Gy per million live born as having genetic diseases caused by induced dominant mutations. The Committee also estimated about 10 extra cases of genetically abnormal children would be expected in the first 10 generations per million live births per 10^{-2} Gy due to recessive mutations. Finally, as to balanced chromosomal rearrangements, the Committee assessed the risk to be between 1 and 15 cases of congenitally malformed children per million live births per 10^{-2} Gy of paternal irradiation (0-5 cases for maternal irradiation). These figures (see Table 2) are also thought to remain valid.

188. Although it did not explicitly say so until 1982, the Committee has always realized that simply presenting the number of serious genetic diseases is to

[i] This subject is reviewed extensively in Annex E, "Genetic hazards".

Table 7

Estimates of risk of severe genetic disease per million live births in a population exposed to a genetically significant dose equivalent of 1 Sv per generation of low-dose-rate, low-dose irradiation, according to the doubling dose method
(based on the UNSCEAR 1986 Report and subsequent work)

(The doubling dose equivalent assumed in these calculations is 1 Sv)

Disease classification	Current incidence per million live births	Effect of 1 Sv per generation		
		First generation	Second generation	Equilibrium
Autosomal dominant and X-linked	10000	1500	1300	10000
Autosomal recessive	2500	5	5	1500
Chromosomal				
Due to structural anomalies	400	240	96	400
Due to numerical anomalies	3400	Probably very small		
Congenital anomalies	60000	Not estimated a/		
Other multifactorial diseases	600000			
Early acting dominants	Unknown	Not estimated a/		
Heritable tumours				
Totals of estimated risk		1700	1400	12000

a/ See paragraph 186.

ignore the full measure of the harm. In the absence of objective and quantifiable indicators of severity, it is hard to assess the full impact of radiation risks in terms of the individual, familial and social burdens imposed by these diseases. Therefore, starting with the UNSCEAR 1982 Report, the Committee began systematically to review data bearing on these problems, to gain a better idea of the true detriment associated with hereditary diseases. Although it is confident that an enquiry of this nature will provide a more refined way of assessing the impact of radiation-induced disorders, the Committee feels that its methodology is not yet ready for use.

189. The Committee wishes to stress that there are still no direct data in man on the induction by radiation of hereditary diseases. Until such data become available there is no alternative but to continue to use data obtained in other mammalian species, suitably corrected to accord with what is known of human genetics, to estimate the risk of hereditary diseases in man.

190. All the numerical estimates of genetic risks discussed thus far have been obtained on the basis of genetically significant doses, i.e., on the assumption that the doses are received by individuals before or during the reproductive period. It is obvious that in the exposure of an entire population, the genetically significant doses are markedly less than the total doses received over a lifetime: damage sustained by the germ cells of individuals who are beyond the reproductive period or who are not procreating for any other reason poses no genetic risks. If is assumed that the mean age at reproduction is 30 years and that the average life expectancy at birth is 75 years, the dose received by age 30 is 40 per cent of the total dose.

191. To derive risk coefficients for genetic diseases in a population, one needs, accordingly, to multiply the genetic risk estimates discussed earlier by 0.40. The calculations shown below make use of the most recent risk estimates presented in Table 7 of Annex E "Genetic hazards", and give the risk coefficients per sievert:

(a) Risk coefficient on the basis of gonadal dose in the reproductive segment of the population (from Annex E, Table 7); for quantifiable damage only, over all generations $12{,}000/10^6$ or 1.2%

(b) Risk coefficient for the whole population, not only the reproductive segment, all generations ($0.4 \times 1.2\%$) 0.5%

(c) Risk coefficient for the first two generations, but otherwise as in (a) above $3{,}100/10^6$ or 0.3%

(d) Risk coefficient for the whole population, for the first two generations ($0.4 \times 0.3\%$) 0.1%

2. Radiation carcinogenesis in man[/]

192. The most recent data in the field of radiation-induced cancer in man have been examined with the following in mind: (a) impressive advances in understanding the molecular mechanisms of cancer induction; (b) the analysis made in Annex B of the UNSCEAR 1986 Report, "Dose-response relationships for radiation-induced cancer"; (c) extensive additional follow-up data on major epidemiological studies such as those of the survivors of Hiroshima and Nagasaki; and (d) a revised dosimetric system for the survivors of Hiroshima and Nagasaki that allows a better analysis of this important epidemiological series.

193. Several factors influence the probability that an individual exposed to radiation will develop cancer. Some of these, the host factors, pertain to the individual, such as his genetic background, age, sex and state of health; others pertain to the conditions of irradiation, such as the dose delivered, the time period over which the dose was received and the quality of the radiation; still others are factors that may interact with radiation to affect the susceptibility of the host, such as his living habits or his exposure to other toxic agents. Thus, there is no single, simple way to assess the effects, so several approaches have been taken.

194. One approach is to study the effects of different exposure or host conditions on biological models of carcinogenesis. This approach allows analysing one or another aspect of the risk, e.g., its variation with time or with the age of the exposed individuals. Another approach aims at analysing dose-response and risk-projection relationships. A third approach is the direct regression study of epidemiological data, especially through modern multiple regression techniques, which are particularly suited to the complexity of these phenomena.

195. The most informative epidemiological series are those which were carried out in the following groups: (a) people who were chronically exposed to high or intermediate doses of radiation when the dangers of such exposures were as yet unknown; (b) people who were chronically exposed to low doses for occupational, medical or environmental reasons; (c) people who received high doses to some parts of the body over short periods for therapeutic purposes; (d) people who were, and are, exposed to low doses of radiation for medical diagnostic purposes; (e) special cohorts who were irradiated externally as a consequence of the atomic bombings at Hiroshima and Nagasaki or internally as a consequence of fallout from the testing of nuclear weapons; and finally, (f) isolated individuals who received fairly high doses in accidents of various sorts.

196. Two methods have been employed in the epidemiological investigation of the groups listed above: (a) cohort studies, in which exposed individuals are analysed usually prospectively for their cancer experience compared with a suitably chosen non-exposed control group and (b) case control studies in which individuals with cancer are matched with normal individuals of a control population and exposures are determined retrospectively. The first method has distinct advantages but of course can be employed only in special circumstances.

[/] This subject is reviewed extensively in Annex F, "Radiation carcinogenesis in man".

197. Most of the retrospective studies discussed in the UNSCEAR 1977 Report have continued up to the present time, and new results have been reported. In several series, such as that on radiation-induced breast cancer, earlier findings were improved and dose-response patterns were made more precise by combining data from several investigations. In other series, such as that on pelvic irradiation for tumours of the uterine cervix, earlier findings were at least partially called into question. In yet other series, such as those on occupationally exposed groups, the earlier findings have, on closer examination and re-interpretation, been criticized for different types of investigating and reporting bias. Uncertainties in the dosimetry, the unsuitability of control groups and potential or actual difficulties in the ascertainment of tumours were some of the problems encountered.

198. All of the most important prospective studies that were in progress in 1977 are still in progress. Three more sets of mortality data, as well as additional incidence data, are now available from the survivors of Hiroshima and Nagasaki, and these have improved the dose-response estimates for some tumour types and have added other malignancies (colon, ovary, multiple myeloma) to the list of those already known to be radiation-induced. Some information has also been added to the studies of people exposed at the Hanford nuclear facility and to fallout in the Marshall Islands and of patients exposed for medical conditions such as ankylosing spondylitis, mastitis, pneumothorax or thymus-related irradiations. The absolute risks in these cohorts of people continue to increase (save, possibly, in the patients with ankylosing spondylitis and in those who were youngest at the time of the bombings in Hiroshima and Nagasaki). All these studies must obviously continue throughout the lifetimes of the exposed individuals in order to complete the data on dose- and time-response relationships for cancer induction. Moreover, for the relevant information to be generalized, it is also vital to know to what degree these cohorts are similar to other populations; how, and with what consequences, exposure to non-radiation risks may have changed; and how, for a general population, the risk of a given dose of radiation relates to the background cancer risk. One of the central problems in risk estimation continues to be the shape of the dose-response relationship, an issue extensively treated in the UNSCEAR 1986 Report. Although a number of models may be used to analyse the risk, each of them represents no more than an approximation to the true dose-response relation and has potential limitations or pitfalls.

199. The mortality experience of Hiroshima and Nagasaki survivors has been the single most important source of information on the radiation-related risk of cancer induction. A recent re-evaluation of tissue-absorbed doses in these survivors has made clear that their exposure to neutrons was substantially less than had been thought, and the relevant data, particularly those from Hiroshima, are now believed to be much less informative about the effects of neutrons than had once been presumed. The large body of experimental data and the very limited amount of epidemiological evidence on the relative biological effectiveness (RBE) of neutrons must therefore be carefully re-examined, with a view to arriving at some estimate of risk for this type of radiation.

200. A new international study of patients surviving treatment for carcinoma of the cervix has provided additional data on second cancers at selected sites.

201. Lifetime cancer experience is not yet available for any of the large epidemiological studies. Therefore, to project the overall cancer risk for an exposed population, it is necessary to use models that extrapolate over time data based on only a limited period of the lives of the individuals. Two such projection models have received particular attention: (a) the additive model which postulates that the annual excess risk arises after a period of latency and then remains constant and (b) the mulitiplicative model in which the time distribution of the excess risk follows the same pattern as the time distribution of natural cancers, i.e., the excess (after latency) is given by a constant factor applied to the age dependent incidence of natural cancers in the population. Data are now available that may provide a deeper insight into the applicability of the two models, and recent findings in Japan suggest that the relative risk projection model is the more likely, at least for some of the most common cancer types. Firmer conclusions should be possible soon.

202. Cancer is generally understood to develop in a number of stages. That is, for malignancies to be expressed a series of events must occur and the rate at which they occur is thought to be reflected in the way cancers appear in the population over the course of time. Analysis of the various epidemiological series in the light of this notion reveals a number of inconsistencies, so that it is not yet feasible to say which stages in carcinogenesis are affected by radiation or whether more than one stage is affected or whether the multistage model is able to explain the actual process. All of these possibilities may apply to some extent. It may even be that events postulated at the cellular or subcellular level cannot be easily related to the clinical data on radiation carcinogenesis.

203. A limited number of genes, known as oncogenes, have been implicated in the malignant transformation of normal cells. The precise ways in which these oncogenes can be activated by radiation are not known, but so far data have not revealed any modifications that would suggest radiation plays a special role in inducing cancer or that would help to differentiate, at the genetic level, radiation-induced tumours from tumours induced by other carcinogens.

204. The Committee has carried out a detailed review of the information available on time-specific susceptibility to radiation-induced cancer and has considered separately the evidence pertaining to the exposure of children and adult subjects. Data on children show that the thyroid, the bone, the bone marrow and the breast are definitely responsive to the carcinogenic action of radiation. The bulk of the children successfully treated by radiation for cancer (i.e., those carrying localized primary tumours) who

have developed secondary tumours are those whose primary tumour had a large heritable component of cause. These children are obviously more prone to develop cancer than a normal child. In general, certain sites are susceptible, and the genetic evidence shows that this has to do with gene regions expressed in both the tissue involved in the original primary tumour (e.g., retinoblastoma) and in the tissue of the second tumour (e.g., bone sarcoma). Individuals with the hereditary form of retinoblastoma are also known to develop osteosarcomas away from the irradiated field or in the absence of irradiation. The spontaneous risk of second tumours in retinoblastoma patients is due to the somatic development of homozygosity in those children who inherit a single copy of the relevant mutation, but it is not yet known whether this is also the mechanism by which radiation induces second tumours. There are indications in the case of second tumours following retinoblastoma that a multiplicative projection model may apply, as it does to most adult tumours.

205. A number of general principles concerning the induction of tumours by radiation can be derived. Radiation is detectably carcinogenic if the dose is high enough, but no cancers unique to radiation are induced. Leukaemia (except chronic lymphatic leukaemia) is the most prominently induced cancer but tumours of the breast, thyroid, lung and bone marrow and at a number of other sites are also induced. The frequency of induction per Gy varies with the site. Some tumours such as chronic lymphatic leukaemia, squamous cell carcinoma of the cervix and Hodgkin's disease are not induced by radiation. Induced tumours are expressed some time after exposure, the latency being at least 2-5 years for leukaemia and about 10 years or more for other tumours. Age is the most significant host factor but other factors such as genetics play a role. These features are explained further in Annex F.

206. In general, the results from cancer patients are similar to those from other exposed groups in regard to the post-irradiation pattern of risk. However, in some instances, the risk in cancer patients appears to be different from that in the general population. This could be due to differences in susceptibility to cancer, but it could also be due to differences in exposure to environmental risk factors, e.g., smoking. Excess cancers occur in both irradiated and non-irradiated patients, making the estimation of radiogenic risks problematic and suggesting that inferred results may not be generally applicable.

207. The dose-response relationships for various forms of malignancy were discussed extensively in Annex B of the UNSCEAR 1986 Report. The conclusion reached there was that each type of tumour may have a characteristic dose-response pattern and that it is still difficult to assess satisfactorily the pattern for the majority of the tumours. However, a general conclusion could be drawn that for low-LET radiations most dose-response relationships were upward concave reaching a maximum that would be followed by decline of the response with further increasing of the dose. This decreasing slope and decline of the curve at high doses seems due to killing of the radiation-initiated cells from which tumours eventually arise.

208. The Committee concluded in 1986 that for some tumours, i.e., carcinomas of the female breast and perhaps of the thyroid a linear relationship at low and intermediate doses of low-LET radiations gave a good fit; for others a linear fit could not be rejected statistically but other models, e.g., linear quadratic and quadratic approximated the data equally well. These observations are still assumed to be basically correct, however, evidence presented recently to the Committee suggests that fractionated doses at very low doses per fraction may be less effective in inducing breast cancer than deduced previously from the linear relationship and apparent lack of dose-fractionation effects. Recent epidemiological studies on patients administered 131-iodine-iodides for diagnostic purposes suggest that low-LET radiation at low dose rates is also significantly less effective than intermediate and high doses delivered at high dose rates. This means probably that the dose-response relationship for induction of cancer of the thyroid gland is also non-linear (upward concave) as was suspected in the UNSCEAR 1986 Report.

209. Many biological differences among human beings are known to modify their susceptibility to radiation-induced cancer, and the Committee examined these differences, known as host factors. Current information generally suggests sex has little or no effect on radiation carcinogenesis, in the sense that the sex ratio for individuals with radiation-induced malignancies (thyroid, breast, lung, leukaemia) is similar to that for non-irradiated individuals with the same malignancies. Data show further that susceptibility to radiogenic tumours decreases with increasing age, the latency periods being related not so much to age at exposure as to the tissue involved. The mean age and the age distribution of cases in adults exposed to single doses are in general similar to those in the population at large. Data on the effect of genetic constitution suggest that there may be a small, but not trivial, fraction of the population which is prone to cancer development and could thus be more susceptible to radiation or other carcinogenic agents. To improve the risk estimates, better means of identifying susceptible individuals should be developed.

210. The concluding section of the Committee's study contains an overall analytical summary of radiogenic cancer effects drawn from the most comprehensive sources available. From only a few epidemiological studies, primarily the survivors of the atomic bombings and patients exposed during treatment of ankylosing spondylitis or cervical cancer (leukaemia only), the carcinogenic risk of radiation can be estimated for many different sites. All three studies comprise large numbers of people exposed to x- or gamma-radiation for short times and followed for long times; however, each set of data has unique characteristics. The Committee considered the results on tissue-specific tumours from these series and compared them with risk estimates produced by various other studies. The Committee's evaluation of risk estimates is discussed in section II.C.2.

3. Early effects in man of high doses of radiation[k]

211. The Committee has reviewed what is known about the effects that occur in man within two to three months from receiving uniformly distributed whole-body doses above approximately 1 Gy of x- or gamma-radiation. The data were collated from three main sources: accidents; the atomic bombings; radiotherapy treatments. Important information on this subject has recently become available as a consequence of the nuclear accident at the Chernobyl power plant, in the course of which about 100 people were exposed to external and internal irradiation amounting to 1 Gy or more. The USSR delegation has prepared especially for UNSCEAR a detailed report entitled "Acute radiation effects in victims of the Chernobyl nuclear power plant accident", which is presented as an Appendix to Annex G.

212. Early prodromal responses during the first 48 hours after irradiation are mediated through the autonomic nervous system and appear as gastro-intestinal and neuromuscular signs. The incidence and latency periods for these effects are dose-dependent. For instance, the dose that induces vomiting in 50 per cent of individuals is approximately 2 Gy, and the mean latency period after this dose is about 3 hours.

213. Doses higher than 50 Gy generally lead to death within two days from cerebrovascular and neurological injuries (the so-called neurological syndrome). Uniform, whole-body doses between 10 and 50 Gy cause the gastrointestinal syndrome, which is generally fatal, with most deaths occurring during the second week after irradiation. In spite of the experience of those who died after the atomic bombings, there is insufficient information to estimate precisely the relationship between the dose and the probability of death due to this syndrome. The time to death of the gastrointestinal syndrome depends on the renewal time of the intestinal lining and is influenced by secondary factors such as infection, haemorrhage, loss of fluid, protein and electrolytes.

214. Uniform, whole-body doses of less than 10 Gy but greater than 1 Gy cause the bone-marrow syndrome, the incidence and severity of which depend on dose. The initial marrow damage after low doses reduces the number of white cells in the blood, the lymphocytes being the most sensitive indicators of injury. Doses of 1-2 Gy reduce the concentration of blood lymphocytes to about 50 per cent of normal within 48 hours of irradiation. Neutrophils show an initial increase over the first few days, then a dose-related fall. Ten days after 2-5 Gy, there is the beginning of a second abortive rise; however, if the marrow does not recover, a final decline is observed. The loss of neutrophils is associated with the onset of fever and is predictive of survival. The time course of platelet loss is broadly similar to that for granulocytes. Platelet levels in the blood below 30,000-50,000 per microlitre are associated with bleeding. People with the bone-marrow syndrome show an increased susceptibility to infection due to injury to the haematopoietic and the immune system.

215. In addition to the systemic effects described, irradiation may also cause damage to many other tissues and organs exposed separately. The resulting clinical symptoms vary as to time for appearance and severity. They may or may not be part of the syndromes described, depending upon the tissues irradiated, the dose level, the modalities of irradiation, and other physical and biological factors.

216. Irradiation of the skin causes lesions that are well known and very dependent on the dose and the area irradiated, in the sense that smaller doses have to take place over larger areas to elicit the same level of damage. Skin lesions include erythema, abnormal hair growth, epilation, desquamation and vascular and dermal injury. The dose in the basal layer of the epidermis determines the amount of cell killing and hence the degree of desquamation.

217. Injury to the mucous membranes in the mouth and throat evokes inflammation and swelling, with ulceration and necrosis after high doses. Mucosal injury is greatest in the cheeks, soft palate, and hypoglossal region. Acute effects on the eye are also well described and very dependent on the structures irradiated and the doses received.

218. When the thorax is irradiated, pneumonitis is the earliest sign of radiation injury in the lung. It appears at 1-3 months for doses greater than 8 Gy. The time of onset of pneumonitis is not significantly dose-dependent between 6 and 12 Gy. At Chernobyl there were some patients with early lung reactions. These changes were probably multifactorial in origin.

219. High acute doses of up to 4 Gy induce temporary sterility in some male individuals, but the dose inducing prolonged sterility in all males is at least 6 Gy. Although some of the differentiating forms of spermatogonia respond early and are very radiosensitive, the sperm count begins to decrease only after 6 weeks. In women, temporary sterility is induced by high doses up to 4 Gy and prolonged sterility by 4-10 Gy. Older women are more susceptible, probably because the number of ovarian follicles decreases with age.

220. It is of interest to know the dose of radiation that causes, on average, 50 per cent of individuals to die within 60 days ($LD_{50/60}$). The LD_{50} is a concept widely used in experimental work but there is doubt as to its applicability in human radiation biology, except for statistical purposes. The epidemiological series available for estimating this dose in man comprise radiotherapy patients, accident cases, and the Japanese exposed to atomic bombings in the Second World War. The $LD_{50/60}$ reflects marrow failure. The most recent studies of the $LD_{50/60}$ from experience in Japan (after revision of the doses) yield values of around 3 Gy. The figure is thought to apply to the very special conditions prevailing after the bombing for irradiated human beings who have no access, or only minimal access, to medical treatment.

[k] This subject is reviewed extensively in Annex G, "Early effects in man of high doses of radiation".

221. Some groups of radiotherapy patients have been useful for assessment of the $LD_{50/60}$. None of 20 children and adolescents given 3 Gy to the whole body to treat Ewing's sarcoma died of marrow failure. The $LD_{50/60}$ for groups of adults irradiated for disseminated cancers was 2.9 Gy in one series and 3.4 Gy in another. All these data indicate that for cancer patients, although they receive supportive treatment, the $LD_{50/60}$ is probably about 3 Gy, while for healthy individuals receiving conventional supportive treatment after irradiation, it may be 4-5 Gy.

222. In the accident at Chernobyl, 43 individuals received doses estimated to have been between 2 and 4 Gy, and one of them died. Of 21 people receiving doses between 4.2 Gy and 6.3 Gy, seven died. Of 20 patients receiving doses between 6 and 16 Gy, 19 died. Because of the complications suffered by many of the patients during the accident, such as thermal and skin injury, it is difficult to derive a value for $LD_{50/60}$ from these data.

223. From its review and discussion of the above data, the Committee concludes that it is impossible to assign a unique value to the LD_{50} in man; it may change substantially depending on age, the state of health of the individuals irradiated and on the prophylactic or therapeutic measures adopted before and after irradiation. For the planning of emergency responses, it is important to know which values of the LD_{50} would apply in which situation. The Committee underlines, however, the purely statistical nature of the LD_{50} and warns that using it to predict the chance of survival of a single individual would be totally unwarranted.

224. Neutrons are more efficient in causing acute injury than x- or gamma-radiation, by a factor of 2-3, using single doses. There is little experience in man of the lethal effects of neutrons, except in a few isolated accidents. The neutron component of the doses to the survivors of the atomic bombings is now considered to be much smaller than had previously been estimated so the data collected from this group of people are therefore of little use in assessing the effects of neutrons.

225. As is well known in the field of radiobiology, dose protraction and fractionation cause less effect than the same total dose given singly. The early effects of high doses in man are no exception to this general rule. Thus, prodromal responses are somewhat alleviated by dose protraction or fractionation. Similarly, low-dose-rate or multi-fractionated irradiation markedly reduces injury to the intestine and the bone marrow in all species including man. Various quantitative formulae have been proposed to estimate the changes in dose or effect brought about by protracted irradiation; however, because the data base for many tissues is sparse, these formulae are only very rough guidelines for prediction. There is, moreover, one exception—the testis—to the general rule on protraction and fractionation; the progression of cells into sensitive phases makes this organ more sensitive to fractionated doses than to single doses.

226. In general, large amounts of internal emitters are required to produce early effects in man. Bone marrow depression is observed after single large intakes of iodine-131 and caesium-137. Gold radiocolloids have produced mild radiation sickness and haemotological complications, as have phosphorus-32 and sulphur-35. Severe acute intestinal injury in man from internal emitters has not been reported, and lung injury has been rare. Treatments for internal contamination with radionuclides are based on local removal, reduced retention, enhanced excretion and diminished translocation.

227. A small fraction of the population may be particularly sensitive to early radiation injury by virtue of inherited genetic disorders, such as ataxia telangiectasia. Persons with this disease are more radiosensitive than normal. Many other genetic disorders predispose to increased chromosomal or cellular injury, but quantitative estimates of this increase are not available.

228. It is difficult to form a prognosis in irradiated patients solely from an estimate of the dose. There are many confounding factors, including intercurrent disease, dose protraction and radiation quality. The type and duration of prodromal symptoms, including erythema, may assist in the prognosis. Haematological signs, particularly the lymphocyte count, are good prognostic indicators. The lowest blood counts and their time of occurrence for the various blood cell types are also important, as is the duration of marrow aplasia after high doses. The appearance and persistence of immature cells in the blood is usually a favourable sign of marrow recovery. A valid prognosis must be founded on a wide range of different types of data and constantly updated.

229. The information provided by the USSR and contained in the Appendix to Annex G on the victims of the Chernobyl accident is exhaustive and valuable. While the nature of the lesions observed is not unexpected, the degree of precision achieved in the analysis of their time of onset and their magnitude and duration adds considerably to our understanding of the biological effects of high doses of radiation in man. Further analysis of these findings is definitely warranted, particularly in respect to the following points: the precise assessments of the doses received by the victims; the correlation of the various symptoms and signs with the causal agents (the pattern of exposure was complex and involved internal and external irradiation and additional thermal exposure in a few cases). These new studies will substantially enhance the present knowledge and will eventually allow the data collected at Chernobyl to be consolidated with other findings discussed in Annex G. The Committee is indebted to all those who contributed to the Appendix for their willingness to share this experience and wishes to commend them for the professional skill and the human compassion shown on such a tragic occasion.

4. Effects of pre-natal irradiation

230. In its latest study of the biological effects of pre-natal irradiation contained in the UNSCEAR 1986 Report, the Committee reviewed the most recent information on developmental events, particularly in the brain of mammalian embryos and foetuses; the irradiation of experimental animals before birth; and children exposed to radiation pre-natally by the atomic bombings at Hiroshima and Nagasaki. Its review centred as much as possible on human experience and included effects that had not previously been considered before in this light, such as the carcinogenic effects of irradiation *in utero*.

231. The 1986 data showed that mental retardation is the most likely type of developmental abnormality to appear in the human species. In essence, analysis as a function of time showed that the probability of radiation-related mental retardation is essentially zero with exposure before eight weeks from conception, is maximum with irradiation between eight and 15 weeks, and decreases between 16 and 25 weeks. After 25 weeks and for doses below 1 Gy, no case of severe mental retardation had been reported. On the assumption that the induction of the effect is linear with dose (as the data seemed to indicate), the probability of induction per unit absorbed dose was estimated at 0.4 per Gy at the time of the peak sensitivity and at 0.1 per Gy between 16 and 25 weeks from conception.

232. Using all the data available, the Committee attempted to derive quantitative risk estimates for the radiation effects for which there is positive evidence or, at least, reasonable presumption of induction. In addition to mental retardation, these effects include mortality and the induction of malformations, leukaemia and other malignancies. Under a number of qualifying assumptions, the Committee estimated that a dose to the conceptus of 0.01 Gy delivered over the whole pregnancy would add a probability of adverse health effects in the live born of less than 0.002. The normal risk of a non-irradiated live born carrying the same conditions is about 0.06. Information becoming available suggests that the risk estimates in the last two paragraphs may need substantial revision downward (particularly in the low-dose ranges). The Committee intends to review this in the near future.

C. DERIVATION OF RISK COEFFICIENTS

233. In the situations described in the Annexes, people are exposed to a range of types of radiation, and the resulting doses in their bodies are often non-uniform. In order to add the doses from groups of sources, e.g., natural sources, it is necessary to use a quantity that takes account of these different kinds of radiation and dose distributions in the body. The quantity used by the Committee is the effective dose equivalent. This quantity is obtained by weighting the absorbed dose in a tissue of the body, first by a factor to take account of the effectiveness of the type of radiation and then by a factor to take account of the different biological sensitivities of the tissues. The sum of these weighted absorbed doses is the effective dose equivalent.

234. The values of the two sets of weighting factors are those recommended by the International Commission on Radiological Protection. From time to time, the Committee has considered other systems of weighting, but has so far decided that the effective dose equivalent remains adequate for its purposes. The use of the effective dose equivalent is limited to assessments of long-term effects such as carcinogenesis. For assessing the early effects of high doses, the absorbed dose is an appropriate quantity.

235. When it uses the term "risk" (in a quantitative sense) the Committee means the probability of a harmful event, e.g., a radiation-induced death and often expresses this probability in per cent. The number of projected events in a population is expressed either as cases per thousand or cases per million. The term "risk coefficient" is used in a general way to indicate the risk per unit dose (risk per gray in the case of absorbed dose or risk per sievert in the case of effective dose equivalent). Since the relationship between dose and risk is not always proportional, it is sometimes necessary also to specify the dose or dose range for which the coefficient is valid.

236. In addition to estimating risk, the Committee has also estimated the projected number of years of life lost in an exposed population due to radiation-induced mortality. This quantity and also the projected number of cases or deaths in an exposed population are sometimes called measures of collective detriment.

1. Hereditary harm

237. Genetic risk coefficients may be defined to apply either to the gonad dose equivalent or the effective dose equivalent. It is also necessary to decide whether they should apply to genetically significant doses (i.e., doses to reproductive individuals) or average doses to the population at large. Opting for the latter might seem absurd from the scientific point of view, but sometimes only average doses or total collective doses are known; moreover, risk coefficients for cancer often apply to average doses.

238. In the UNSCEAR 1986 Report and in Annex E of this Report, "Genetic hazards", the Committee has reviewed the present body of knowledge of the hereditary effects of ionizing radiation. These reviews are summarized in section I.D.1. There are several customary ways of presenting the scientific information. One is to make the assessment for an equilibrium situation, wherein a stable population has been exposed over many generations, with each reproductive individual, male or female, receiving a unit gonad dose, and to estimate the fraction of the offspring who would then be expected to be affected by hereditary harm. Another way is to assess the affected number of offspring to a parent generation where the parent generation, males or females or both, have received a given collective dose.

239. In both cases, the information can be translated into a risk coefficient that expresses either the probability of a reproductive individual giving birth to a child affected by hereditary harm or the expected number of affected children, per unit individual or collective gonad dose to reproductive individuals. The risk coefficient may also be extended to include harm in all future generations.

240. Such risk coefficients can be applied directly to estimates of the genetically significant dose, such as those which have been made for various medical diagnostic x-ray procedures. However, they cannot be applied to effective dose equivalents unless there is uniform whole-body exposure. In other cases, the applicable genetic risk coefficient could range from zero (if the gonads are not exposed) to four times the risk coefficient that is applicable to the gonad dose (in the case that only the gonads are exposed), the organ weighting factor for the gonads being 1/4.

241. If the effective dose equivalent is assessed not for reproductive individuals but for average individuals in the population at large, then the relevant risk coefficient is only F/L of the genetic risk coefficient that would apply to reproductive individuals, F being the main reproductive age and L the life expectancy at birth. If F is about 30 years and L about 75 years, the genetic risk coefficient for the average individual becomes 40 per cent of the coefficient for reproductive individuals.

242. Table 8 summarizes the Committee's present estimates of genetic risk coefficients. Extensive information about the nature of the genetic risk is presented in the UNSCEAR 1986 Report.

243. A comparison with previous estimates (see Table 1) shows that present estimates are lower than those made in 1977. The 1977 estimates were used when the ICRP defined the effective dose equivalent. The risk coefficients refer only to the expected number of cases of quantifiable, severe, hereditary disease. What this means in terms of detriment is a question the Committee will continue to study.

2. Cancer

244. Cancer risk coefficients may be expressed either as (a) the site-specific individual probability of future radiation-induced cancer (death) per unit dose or (b) the collective detriment. The latter may be presented either as the expected number of cancer deaths (or cases) in the exposed population or as the number of person years lost because of cancer deaths per unit collective dose.

245. The new assessments in Annex F, "Radiation carcinogenesis in man", relate to the cancer risk at doses of 1 Gy at high dose rate of low-LET radiation. It has to be stressed, however, that statistically significant excess cancer mortality in Hiroshima and Nagasaki has been observed for the first time for some cancers and at several specific sites at doses between 0.2 and 0.5 Gy. Not only have the risks from nine types of cancer been assessed with reasonable confidence, but also the total risk from all other types of cancer has been independently assessed. The risk estimates include a projection into the future of observations on the exposed populations at Hiroshima and Nagasaki. The new estimates have taken into account the revised dosimetry. All of this has had the combined effect of making the risk estimates at these doses and dose rates higher than before.

(a) Site-specific individual risk

246. Table 9 shows the results of the Hiroshima-Nagasaki study with regard to the individual probability of death from site-specific radiation-induced cancer. Two sets of numbers are given: one is derived from projections based on the additive (absolute) risk model, the other from projections based on the multiplicative (relative) risk model.

247. The total cancer mortality risk coefficient for the average individual (averaged also over both sexes) is 4.5 per cent per gray on the additive risk model and 7.1 per cent per gray on the multiplicative risk model. These numbers may be compared with the 1977 estimate for high doses, which was about 2.5 per cent per sievert on the basis of the additive model (see Table 3). Further summary values of risk coefficients for populations of other ages and other circumstances are given in Table 10. These lifetime risks range from 4 per cent to 11 per cent per gray.

Table 8

Revised genetic risk coefficients a/
(per cent per Sv)

	For gonad dose equivalent		For effective dose equivalent	
	Reproductive population	Total population	Reproductive population	Total population
First two generations	0.3	0.1	0-1.2	0-0.5
All generations	1.2	0.5	0-5	0-2

a/ Risks from diseases of complex aetiology were not estimated. See also paragraph 186.

Table 9

Per caput lifetime excess cancer deaths probability following exposure to 1 Gy organ absorbed dose at high dose rate of low-LET radiation
(per cent)
(based on the population of Japan using an average age risk coefficient)

	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	0.97	0.93
All cancers except leukaemia	6.1	3.6
Bladder	0.39	0.23
Breast a/	0.6	0.43
Colon	0.79	0.29
Lung	1.5	0.59
Multiple myeloma	0.22	0.09
Ovary a/	0.31	0.26
Oesophagus	0.34	0.16
Stomach	1.3	0.86
Remainder	1.1	1.0
Total	7.1	4.5

a/ Value has to be divided by 2 to calculate the total and other organ risks.

Table 10

Estimates of projected lifetime risks for 1000 persons (500 males and 500 females) exposed to 1 Gy of high dose rate low-LET radiation

(based upon the population of Japan)

	Risk projection model	Excess fatal cases	Years of life lost
Total population	Additive	40- 50	950-1200
	Multiplicative	70-110	950-1400
Working population (aged 25-64 years)	Additive	40	880
	Multiplicative	80	970
Adult population (over 25 years)	Additive	50	840
	Multiplicative	60	640

248. The problems in deriving risk coefficients that are also applicable at low doses are the same as before. Such risk coefficients can only be inferred from the observed values at moderate to high doses. In 1977, when the total cancer risk coefficient at high doses was estimated to be about 2.5 per cent per sievert, the Committee pointed out some of the uncertainties; these included the fact that this estimate was an underestimate because no projection had been made into the future, but it was also an overestimate in the sense that the risk per unit dose at low doses was believed to be lower than the estimates for high doses.

249. In this Report, the problems in deriving risk coefficients at low doses and for low dose rates remain. The Committee agreed that there was a need for a reduction factor to modify the risks shown in Table 9 and Table 10 for low doses and low dose rates. The Committee considered that such a factor certainly varies very widely with individual tumour type and with dose rate range. However, an appropriate range to be applied to total risk for low dose and low dose rate should lie between 2 and 10. The Committee intends to study this matter in detail in the near future.

250. The Committee has not presented risk estimates for high-LET radiation in general in this Report (except for the exposure to radon of uranium miners). For low doses of external high-LET radiation it would be necessary to multiply the risks for low-LET radiation by an appropriate quality factor. No dose or dose rate reduction factor is considered necessary for high-LET external radiation at low doses.

(b) Collective detriment

251. The product of risk coefficients appropriate for individual risk and the relevant collective dose will give the expected number of cancer deaths in the exposed population, provided that the collective dose is at least of the order of 100 man Sv. If the collective dose is only a few man Sv, the most likely outcome is zero deaths.

252. The Committee has also assessed the person years lost per unit collective dose because of radiation-induced cancer mortality. The results at high doses and high dose rates of low-LET radiation are summarized in Table 10. The total loss amounts to about 1 person year per man Gy, with both projection models.

D. COMPARISON OF EXPOSURES

1. Previous UNSCEAR comparisons

253. The way in which to present radiation exposures from various sources has always been a problem for the Committee. In its 1958 Report, the Committee assessed the per caput mean marrow dose and the genetically significant dose to the world population from various sources and practices. At that time, the Committee even calculated the expected number of cases of leukaemia and hereditary harm from natural background radiation and nuclear explosions.

254. In the UNSCEAR 1962 Report, the Committee assessed the per caput doses from natural irradiation of the gonads, the bone surface layers and red bone marrow. It also calculated the dose commitments to the world population for the same organs. The genetically significant dose was assessed for medical and occupational exposures. However, in that Report the Committee felt that it had less confidence in the risk coefficients used in the UNSCEAR 1958 Report and that it was not able to assess any detriments. It stated, instead, that the estimated doses and dose commitments could be used for comparative risk assessments and gave this comparative risk in relation to natural background radiation, which was assigned the value of unity. This comparison was made for medical exposures and nuclear explosions with reference to leukaemia, bone tumours and hereditary effects. On the same basis, the Committee said, the detriment of various sources could be expressed in terms of exposure to natural background radiation that would give the same per caput dose or dose commitment.

255. In the UNSCEAR 1964, 1969 and 1972 Reports, the Committee continued to express the risk from nuclear explosions in terms of the equivalent period of exposure to natural background radiation. Until 1972 the Committee had calculated per caput doses or dose commitments for the whole world population. For a population of a given number, this implies an assessment of the collective dose from each source. In the UNSCEAR 1977 Report, the Committee for the first time explicitly presented collective dose assessments for various sources and practices. At the same time, however, it also drew comparisons on the basis of equivalent periods of natural background exposure. In the UNSCEAR 1982 Report, the Committee included more information on the ways in which individual exposures vary, and it assessed collective dose commitments. In the summary and conclusions, the collective dose equivalents were translated into equivalent periods of natural background radiation.

256. From this short review it can be seen that comparison with the natural background dose rate has always played an important role in the Committee's presentation of its assessments. When, in 1958, the Committee estimated the number of affected persons, it drew a comparison with the natural occurrence of cancer and hereditary disease. Since then, per caput and collective doses have been compared with the corresponding doses caused by natural radiation.

2. Purpose of comparisons

257. Comparisons usually have a purpose and may be presented in different ways depending on that purpose. Comparisons with doses or detriments caused by natural sources of radiation may help to clarify the relative radiological importance of man-made radiation sources, but they say little about justifiability or acceptability of these other sources. Information on where doses are low or high in relation to the natural background may help in determining whether there is a potential for meaningful epidemiological studies. Comparing the radiation doses or risks of alternative procedures for achieving one and the same objective, e.g., medical diagnostic information, may disclose what might be preferable from the radiation protection point of view, but it will not reveal other risks or disadvantages. Since the Committee has no use of its own for comparisons, it wishes to present its data in such form that they can be used for a number of different purposes.

3. Comparison of collective doses

258. If risk coefficients are known and if proportionality between dose and response can be assumed, radiation detriments, such as the expected number of cancer deaths, can be calculated from information on collective dose commitments. For relative comparisons, however, it suffices to compare collective doses or per caput doses (which amounts to the same thing) from the various sources, thereby eliminating the uncertainty in the risk coefficients. In such comparisons, the annual collective dose from natural sources of radiation may be taken as the reference; the contribution from other sources may be expressed in terms of the equivalent periods of natural background radiation, as has been the Committee's practice since 1962.

259. When collective doses from different sources are compared, it is important that the comparison be

on a relevant basis. This is simple for sources and practices aimed at achieving one and the same objective, such as energy production or medical diagnostic information. In other cases, one must be careful to find a common basis for comparison. For example, it is of doubtful relevance to compare collective doses to arbitrarily selected populations and time periods. However, although comparisons of collective doses from entirely different practices will often not be very meaningful, they may sometimes help in setting priorities for dealing with concerns of radiological consequences.

4. Comparison of individual doses

260. The radiation doses an individual receives from various man-made sources are normally compared with the dose he receives from natural sources of radiation. An extra dose that is small in relation to the background dose will not significantly affect an individual, i.e., it will not change his total exposure situation noticeably. While the individual might still wish to avoid such a small extra dose, he would know that it does not in itself present any substantial risk. This does not mean that the dose is acceptable just because it is small: rather, acceptability would depend on the total harm the source is likely to cause and on society's appraisal of that harm.

261. Comparing per caput doses in the case of an uneven dose distribution within a population may be misleading, since no individual may actually receive the per caput dose but instead will receive either higher or lower doses. In that case, comparing typical doses as well as extreme doses may be more appropriate.

5. Summary of dose comparisons

262. Table 11 summarizes the various estimates of radiation doses. As in previous Reports, the equivalent period of exposure to natural background radiation is given along with the collective dose commitments. In comparing these estimates with those in previous Reports, it should be remembered that the estimate of the annual dose from natural background radiation has increased, from less than 100 mrad (corresponding to about 1 mSv) in the 1977 Report to 2.4 mSv in the present Report. This increase came about for two reasons: (a) instead of giving a number of organ doses, the effective dose equivalent is now given and (b) the large contribution from radon daughter products has been recognized.

263. Table 11 is of necessity a considerable condensation of the available information. It is worth noting that about half of the natural background radiation is contributed by lung irradiation by radon daughters. Occupational exposures are experienced by those who work in the medical field as well as those who work in the nuclear power industry and in industrial radiography. Exposures from nuclear power production are due to radionuclides released from uranium mining and waste disposal activities, as well as from the operation of reactors to produce electric energy. About one third of the current exposures from nuclear power is attributable to radon emissions from mine tailings and another third to carbon-14 discharges from reactor operation, primarily heavy water reactors.

264. Of the collective effective dose equivalent commitment (other than from ^{14}C) from all atmospheric test explosions, 1.5 million man Sv have been contributed by short-lived radionuclides and 3.5 million man Sv

Table 11

Summary of estimates of effective dose equivalent

Source or practice	Present annual individual doses (mSv)		Collective dose commitments	
	Per caput (World population)	Typical (exposed individuals)	Million man Sv	Equivalent years of background
ANNUAL			Per year of practice	
Natural background	2.4	1 - 5	11	1
Medical exposures (diagnostic)	0.4-1	0.1 -10	2-5	0.2-0.5
Occupational exposure	0.002	0.5 - 5	0.01	0.001
Nuclear power production	0.0002	0.001-0.1	0.001 (0.03) a/	0.0001 (0.004) a/
SINGLE			Per total practice	
All test explosions together	0.01	0.01	5 (26) a/	0.5 (2.4) a/
Nuclear accidents			0.6	

a/ The additional long-term collective dose commitments from radon and carbon-14 for nuclear power production and carbon-14 for test explosions are given in parentheses.

represent contributions to present individual life-time doses primarily from strontium-90 and caesium-137. Because the Chernobyl accident led to doses mainly in Europe, the collective effective dose equivalent commitment rather than the global per caput dose is presented.

6. Direct comparison of detriments

265. In this Report, the Committee has reviewed the existing knowledge on radiation risks and has ventured to indicate the magnitude of the risk factors for low doses as well as for high doses. The Committee has also assessed the collective doses from various sources and practices. It is tempting to combine the estimates and calculate the expected number of cases of cancer and hereditary disease.

266. Many estimates of this type, with different degrees of reliability, depending on the risk coefficients assumed, and with widely different purposes on the part of those who made them, have been reported. The results have been very scattered, depending on the general assumptions. The Committee hesitates, for a number of reasons, to add its own detriment assessments to those already provided for the various sources of radiation.

267. First, the Committee needs to bear in mind the terms of reference under which it operates: its purpose is to evaluate doses, not to make value judgements or engage in setting standards. As is made clear by the discussion in section II.D.4, even those assessments of risk that purport to be scientific involve assumptions and decisions that are not, strictly speaking, scientific. Indeed, the physical quantities used by the Committee reflect such assumptions. For example, the effective dose equivalent, by definition, includes weighting factors that depend on subjective judgements as to what constitutes radiation-induced harm. For each further step in processing the basic information, non-scientific judgements are likely to be needed or implied.

268. Next, the way in which the basic scientific facts are presented influences the impression they give. For example, thousands of cancer deaths from a single accident would undoubtedly be a high number of deaths. However, since such deaths could be expected to occur over a long period of time, the annual incidence will be low. This means a very small increase of the normal incidence of cancer, an increase which is not expected to be noticeable in health statistics. This shows that it is possible, by selecting the form of presentation, to convey different impressions.

269. Lastly, there is the great uncertainty of such estimates. It was stressed in section II.C that the risk coefficients for cancer at low doses can only be inferred from observations at high doses and that the risk coefficients for hereditary effects are not even deduced from observations in man. Even though the Committee believes that its estimates are the best that can be given at the current state of knowledge, it must qualify them by drawing attention to the underlying assumptions and uncertainties. Unfortunately, any estimate of a finite number of cancer deaths is soon taken out of context and the qualifications forgotten.

270. For these reasons, the Committee prefers to follow its previous practice of comparing collective dose commitments from the main radiation sources rather than estimated detriments.

Appendix I

MEMBERS OF NATIONAL DELEGATIONS ATTENDING THE THIRTY-FIRST TO THIRTY-SEVENTH SESSIONS

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CANADA

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Wei Lüxin (Representative), Li Deping, Wu Dechang

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SWEDEN

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UNION OF SOVIET SOCIALIST REPUBLICS

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Appendix II

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Appendix III

REPORTS RECEIVED BY THE COMMITTEE

1. Listed below are reports received by the Committee from Governments between 19 April 1986 and 17 June 1988.

2. Reports received by the Committee before 19 April 1986 were listed in earlier reports of the Committee to the General Assembly.

Document	*Country*	*Title*
A/AC.82/G/L. 1732	United Kingdom of Great Britain and Northern Ireland	Environmental radioactivity surveillance programme: results for the UK for 1984
1733	Japan	Radioactivity Survey Data in Japan Number 72, March 1985
1734	Japan	Radioactivity Survey Data in Japan Number 73, June 1985
1735	United States of America	Environmental Measurements Laboratory: A compendium of the EML's research projects related to the Chernobyl nuclear accident
1736	United States of America	Environmental Measurements Laboratory: The high altitude sampling program: radioactivity in the stratosphere
1737	Japan	Radioactivity Survey Data in Japan Number 74, Sept. 1985
1738	Japan	Radioactivity Survey Data in Japan Number 75, Dec. 1985
1739	Union of Soviet Socialist Republics	Assessment of population doses from x-ray examination in the USSR (1970-1980)
1740	Union of Soviet Socialist Republics	Genetic effects of radionuclide decay
1741	Union of Soviet Socialist Republics	Acute radiation effects in man
1742	Union of Soviet Socialist Republics	Production and release of carbon-14 in nuclear power stations with RBMK reactors
1743	Union of Soviet Socialist Republics	Body burden of fallout caesium-137 in the inhabitants of Moscow 1980-1983
1744	Union of Soviet Socialist Republics	Radiation doses to the far north inhabitants
1745	Union of Soviet Socialist Republics	Occupational exposure of radiographic workers
1746	Japan	Radioactivity Survey Data in Japan Number 76, March 1986
1747	Japan	Radioactivity Survey Data in Japan Number 77, June 1986
1748	Japan	Radioactivity Survey Data in Japan Number 78, October 1987
1749	Japan	Radioactivity Survey Data in Japan Number 79, October 1987
1750	Union of Soviet Socialist Republics	Proposals for setting possible intake limits for transuranium radionuclides absorbed from the gastro-intestinal tract
1751	Union of Soviet Socialist Republics	The evaluation of non-stochastic effects in man from low doses of internal irradiation

Document	*Country*	*Title*
1752	Union of Soviet Socialist Republics	Tritium production in LWGR power plants and its release into the environment
1753	Union of Soviet Socialist Republics	Medical treatment in the case of uranium intoxication
1754	Union of Soviet Socialist Republics	Dynamics of effective dose equivalent from intake of strontium-90 and caesium-137
1755	Union of Soviet Socialist Republics	Specific activities of natural radionuclides in building materials used in the Soviet Union

back to first page

ANNEX A

Exposures from natural sources of radiation

CONTENTS

Introduction

1. The major contribution to the various radiation exposures received by mankind comes from natural sources. These include external sources, such as cosmic rays and radioactive substances in the ground and in building materials, and internal sources resulting from the inhalation and ingestion of naturally occurring substances in air and diet. One characteristic of natural irradiation is that it has been experienced by the whole population of the world at a relatively constant rate over a long period of time.

2. Exposures from natural sources of radiation have been reviewed by the Committee in its 1958, 1962, 1966, 1972, 1977 and 1982 Reports. Since such exposures do not, to a large extent, vary with time, the changes in the estimates of doses in successive Reports have reflected greater knowledge in the field of natural radiation, including wider surveys of the levels of radioactive materials in the environment and in human tissues, as well as changes in the dosimetric models and quantities used.

3. The Committee has repeatedly reviewed the exposures from natural sources of radiation for a number of reasons including: (a) natural background radiation represents a substantial fraction of the total radiation exposure of most individuals and is generally the most important source of radiation; (b) the knowledge of the doses from natural background is necessary as a basis for comparison with man-made sources of exposure; (c) some exposures vary substantially according to human practices and some of the natural sources of radiation may be easily controllable; and (d) owing to the large variability of doses from natural sources of radiation, some individual doses may be high enough to warrant the introduction of remedial measures.

4. The purpose of this Annex is to update information on exposures to all sources of natural radiation. The range of activity concentrations of natural radionuclides and the range of exposures of people can be very wide. The discussion in this Annex is focused on the exposures associated with the higher portions of the observed distribution, i.e., with elevated levels of radiation or of radioactive materials. These can sometimes be identified in a comparison with the world average value of the parameter being considered, for example, a comparison of the external dose rate from cosmic radiation at a high altitude with the average at sea level; in some instances it may be more appropriate to consider the average for a country or geographical region; for example, radon concentrations in air indoors in a part of a country compared with the average for that country.

5. Both occupational exposures and exposures of members of the public are considered. Not considered is any type of exposure arising from a technological activity expressly designed to make use of the nuclear or radioactive properties of a substance, such as exposures to consumer products containing naturally occurring radionuclides or exposures due to uranium mining and milling. The latter, however, are dealt with in Annex B, "Exposures from nuclear power production".

6. The first chapter presents an overview of the natural sources of radiation and indicates the changes that have been made since the UNSCEAR 1982 Report. Chapter II presents a discussion of the doses resulting from the inhalation of radon and its decay products, which constitute the most important exposure from natural sources of radiation. In chapter III, exposures resulting from industrial activities are considered in depth.

I. NATURAL SOURCES OF RADIATION

7. Natural radiation sources are classified into two categories: (a) External sources of extraterrestrial origin, i.e., cosmic radiation, and radiation of terrestrial origin, i.e., the radioactive nuclides present in the crust of the earth, in building materials and in air; (b) Internal sources, comprising the naturally occurring radionuclides that are taken into the human body.

8. Table 1 summarizes the contributions of natural sources to the radiation exposure of human populations living in areas of normal radiation background. The mean annual effective dose equivalent is estimated to be 2.4 mSv. This refers to exposures of adults in the populations.

9. Some of the contributions to the total exposure to natural radiation background are quite constant in space and time and practically independent of human practices and activities. This is true for doses from ingestion of ^{40}K, which is homeostatically controlled, and for doses from inhalation and ingestion of cosmogenic radionuclides, as such radioactive materials are to a first approximation homogeneously distributed over the surface of the globe. At the other end of the spectrum are exposures that depend strongly on human activities and practices and present a wide variability. Doses from indoor inhalation of radon and thoron decay products are typical: building design and practices, as well as the choice of building materials and of ventilation systems, influence indoor levels, thus implying a variation with time of the doses from radon as the techniques and practices evolve. Variability from one dwelling to another also stems from the wide range of radon entry rates from soil, which are to a large extent still unpredictable. In between those extreme types of exposures are several other types: (a) external doses from cosmic rays which, though affected by human practices and quite predictable, cannot be controlled except by moving to an area with a lower dose level; (b) doses from inhalation and ingestion of long-lived nuclides of the ^{238}U and ^{232}Th decay series, which represent a small contribution to the total dose from natural sources and which are relatively constant in space; (c) doses from external irradiation by terrestrial sources, which are also significantly altered by human activities and practices, especially through indoor exposure. Such doses, however, are, as a rule, smaller than those from inhalation of radon decay products and much less variable.

10. In comparison to the estimates given in the UNSCEAR 1982 Report, several changes have been made: (a) with respect to external exposures to cosmic radiation, the new estimates of the annual effective dose equivalents take into account the geographical distribution of the world population as a function of altitude and the shielding effect of building materials. As a result, the new estimates of the annual effective dose equivalents due to the ionizing and neutron components are higher by 20 μSv and 30 μSv, respectively; (b) regarding external exposure to terrestrial sources of radiation, the annual effective dose equivalents have been re-evaluated and increased by 60 μSv, as a result of better knowledge of the indoor gamma absorbed doses in air; (c) annual effective dose equivalents from internal exposure to primordial radionuclides have been reassessed slightly downwards for the ^{238}U and ^{210}Pb series, as well as for the decay products of ^{220}Rn, whereas those for the short-lived decay products of ^{222}Rn have been increased by about 300 μSv on the basis of more comprehensive results of nation-wide indoor surveys. The net effect of these corrections is a 20% increase in the estimate of the overall annual effective dose equivalent from natural sources of radiation. The various contributions to the annual effective dose equivalent are discussed in more detail in this chapter. As in the previous reports of the Committee, the estimates of exposures from natural sources of radiation are essentially based on measurements in temperate latitudes, and on dietary and living habits also of the populations in these regions. It is recognized that exposures to populations in tropical latitudes may differ substantially from those in temperate latitudes, because of differences in environmental concentrations, and also in living and dietary habits. An effort has been made to estimate the exposures to populations in tropical latitudes, but the data base available is too small to enable a good assessment to be made.

11. Table 1 shows clearly the magnitude of the inhalation of ^{222}Rn and its short-lived decay products, a topic discussed extensively in chapter II.

12. Exposures resulting from industrial activities that bring to the surface of the earth, or make available to the public, materials with enhanced concentrations of naturally occurring radionuclides do not significantly alter the picture presented in Table 1. These exposures, which may to a large extent be controlled, are dealt with in chapter III.

A. COSMIC RAYS

13. The high-energy radiation that enters the earth's atmosphere from outer space is known as primary cosmic rays. Upon interaction with the nuclei of atoms present in the air, neutrons, protons, pions and kaons (secondary cosmic rays) are produced, as well as a variety of reaction products (cosmogenic nuclides) such as ^{3}H, ^{7}Be, ^{10}Be, ^{14}C, ^{22}Na and ^{24}Na. The high-energy secondary cosmic rays thus formed react further with nuclei in the air to form more secondary particles (electrons and muons).

1. External irradiation

14. The doses from directly ionizing components of cosmic rays and from neutrons are examined separately.

(a) Ionizing component

15. In the lower atmosphere, the dose rates in air due to the ionizing component vary little with latitude but significantly with altitude, doubling approximately every 1,500 metres. Figure I shows the variation of absorbed dose rates in air, as derived from ionization chamber measurements aboard aircraft, for low and high latitudes and altitudes ranging from 0 to 12 km [H10]. Dose rates in air are about 30 nGy h^{-1} at sea level for any latitude and increase to about 4 μGy h^{-1} at an altitude of 12 km and high latitudes [H10].

16. The majority of the world's population lives at altitudes close to sea level. In the UNSCEAR 1982 Report (Annex B, paragraph 10), the absorbed dose rate in outdoor air from the ionizing component of cosmic rays was estimated to be 32 nGy h^{-1}; this value is taken to be numerically equal to the effective dose equivalent. Indoors, the doses are somewhat lower because of the shielding effect of building structures. Dose rates measured at the centre of various levels of

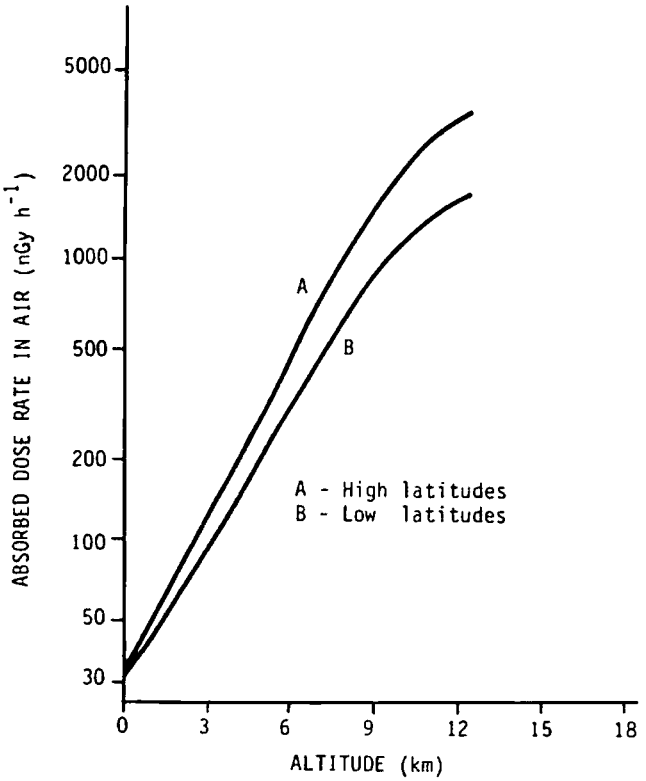

Figure I. Absorbed dose rates in air as a function of altitude and geomagnetic latitude. [H10]

a 12-storey building showed a fairly smooth decline with depth, i.e., from the top storey down to the basement of the building (Table 2) [M9]. The marked change from the roof to the twelfth floor (65% transmission) was because of the filtering out of the relatively soft component of the cosmic-ray flux at sea level. The value of the remaining hard component decreased more slowly with depth. These measurements, however, which were made in a massive building, are not representative of the average situation. A shielding factor of about 0.85 has been reported in the case of a 0.2 m thick concrete layer [L6]. In the Netherlands, the following shielding factors for cosmic rays were determined [J17]: 0.82 for single homes with wooden ceilings; 0.76 for row houses and office buildings with wooden ceilings and floors; 0.50 for dwellings of any kind with concrete ceilings and floors; 0.42 for apartment buildings. Somewhat higher values, varying according to the floor number, were obtained in the Soviet Union [F10]: 0.81-0.96 in wooden houses; 0.72-0.92 in old stone buildings; 0.54-0.86 in modern buildings. A mean shielding factor of 0.8 is assumed in this Annex. The average indoor absorbed dose index rate at sea level is thus estimated to be about 26 nGy h^{-1}.

17. Using a value of one for the quality factor of the ionizing component of cosmic rays and an indoor occupancy factor of 0.8, the annual effective dose equivalent is estimated to be about 240 μSv at sea level. The doses received by populations living above sea level are higher; they are discussed later in this section.

(b) Neutron component

18. The variation with altitude and latitude of the neutron component is similar to that of the ionizing component (see Figure II). At sea level, the neutron fluence rate is about 0.008 cm^{-2} s^{-1} [H10, H11]. On the basis of measurements of neutron energy spectra carried out in New York City in 1986 during a six-month experiment, Hajnal [H15] calculated an average absorbed dose rate of 0.8 nGy h^{-1} and a dose equivalent rate of 3 nSv h^{-1} for neutrons incident isotropi-

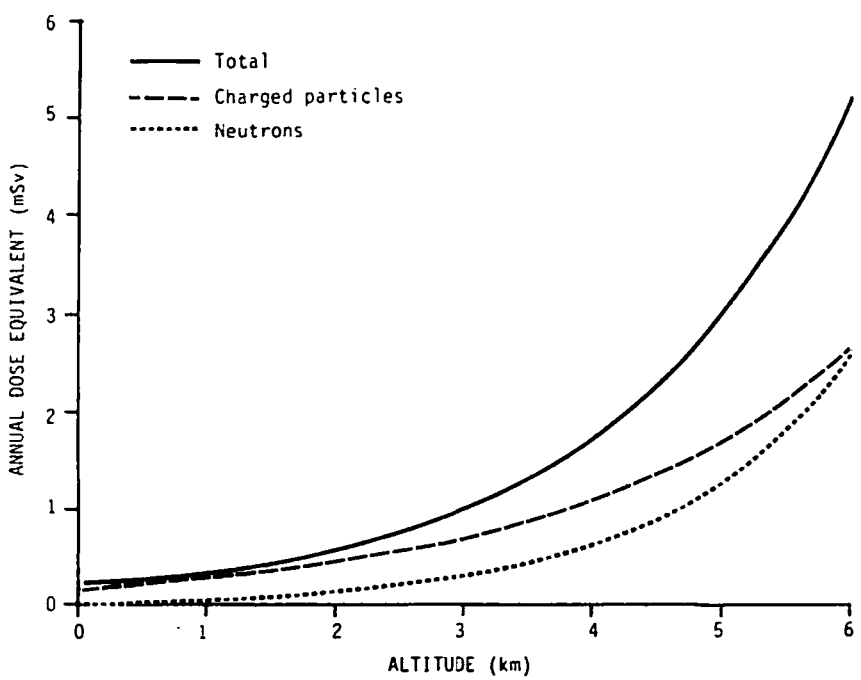

Figure II. Variation of the annual effective dose equivalent from the ionizing component and the neutron component of cosmic radiation as a function of altitude. (Based on [B38])

cally on both sides of a 30 cm thick tissue-equivalent slab. This translates into a quality factor of 3.8. In Japan, Nakamura et al. [N20] also derived from measured neutron energy spectra a dose equivalent rate of 3.3 nSv h^{-1}, which is an overestimate of the effective dose equivalent rate, as it was determined using the conversion factors from flux density to maximum dose equivalent given in ICRP Publication 21 [I14]. Nakamura et al. [N20] also indicated that the calculated spectra of O'Brien led to values of 2.2 nSv h^{-1} at the surface of a 30 cm body phantom and of 1.4 nSv h^{-1} on average over the phantom. These figures are consistent with the estimate of 2.4 nSv h^{-1} that was used in the UNSCEAR 1982 Report and reflect the variability associated with the choice of geometry used to calculate the effective dose equivalent. Neglecting the shielding effect of building structures, the annual effective dose equivalent for the neutron component is estimated to be about 20 μSv at sea level.

19. On the basis of radiobiological considerations, the ICRP issued in 1985 an interim recommendation according to which the value of the quality factor for neutrons was to be increased by a factor of 2 [I12]. This recommendation, however, has not been followed in most countries and has not been confirmed by the ICRP. The value of the quality factor for neutrons has not been modified in this report.

(c) Distribution of doses

20. The distribution of the effective dose equivalent from cosmic radiation over the globe was estimated by Bouville and Lowder [B38] using tabulated data on terrain heights on a 1° by 1° grid [G22], combined with population data [U7]. On the basis of information in [L14], [O3], [N20] and [H15], the variation of the annual effective dose equivalent from the ionizing component $\dot{H}_I$, in μSv, as a function of altitude z (km) was expressed as

$$\dot{H}_I(z) = \dot{H}_I(o)\,[0.205 \exp(-1.649z) + 0.795 \exp(+0.4528z)] \quad (1.1)$$

and, for the neutron component $\dot{H}_N$, as

$$\dot{H}_N(z) = \dot{H}_N(o) \exp(1.04z) \quad (1.2)$$

for $z < 2$ km, and as

$$\dot{H}_N(z) = \dot{H}_N(o)\,[1.98 \exp(0.698z)] \quad (1.3)$$

for $z > 2$ km. The value used in [B38] for $\dot{H}_I(o)$ is similar to the figure of 240 μSv a^{-1} adopted in this Annex, while the value for $\dot{H}_N(o)$ is the same.

21. For the purpose of this Annex, the distribution of the effective dose equivalent from cosmic radiation over the globe was recalculated, using the annual sea-level effective dose equivalents of 240 μSv for the ionizing component and 20 μSv for the neutron component. Figure II shows the variation of the annual effective dose equivalent with altitude obtained from equations (1.1), (1.2) and (1.3). It is worth noting that the dose equivalent from the neutron component, which is small at sea level, increases more rapidly than the dose equivalent from the ionizing component and becomes more important at altitudes above 6 km. The distribution of the collective effective dose equivalent as a function of altitude, presented in Figure III, indicates that although about one half of the collective dose equivalent is received by people living at altitudes below 0.5 km, a contribution to the collective dose equivalent of about 10% is received by populations living at altitudes above 3 km. There are large countries with mountains and sea borders, like the Soviet Union and the United States, where the population-weighted dose from cosmic rays differs only slightly from the dose at sea level because the bulk of the population in each of

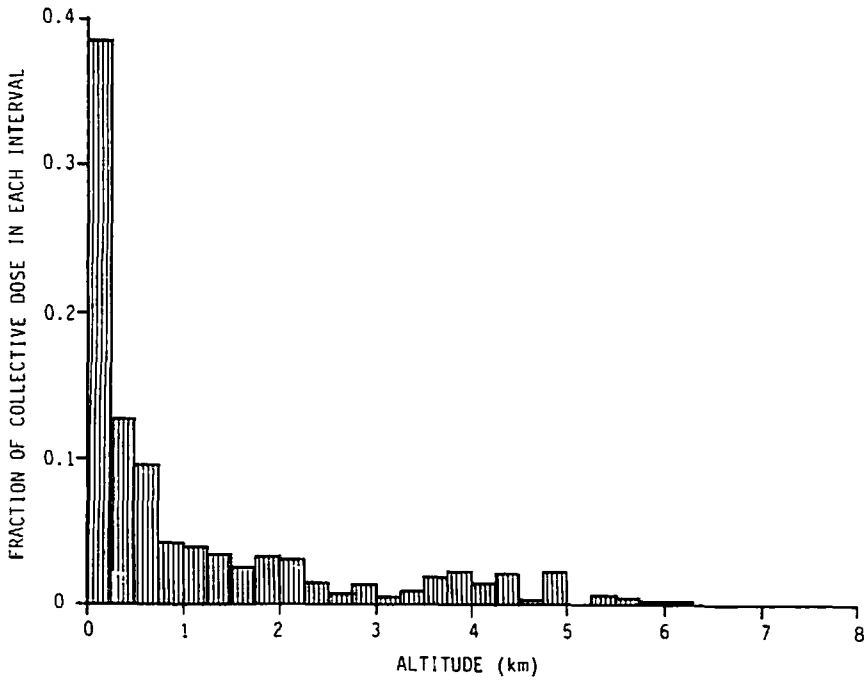

Figure III. Distribution of the collective effective dose equivalent from cosmic radiation as a function of altitude.
(Based on [B38])

those countries lives at low altitudes [F10, O12]. In countries like Ethiopia, Islamic Republic of Iran, Kenya or Mexico, however, large cities are situated on elevated plateaux, accounting for relatively high exposures. Figure IV presents the distribution of the collective dose equivalent and the variation of the per caput dose equivalent as a function of latitude. The population of the northern hemisphere accounts for about 90% of the total collective effective dose equivalent. The per caput effective dose equivalent for the world's population is found to be 355 μSv, the ionizing and neutron components contributing about 300 and 55 μSv, respectively.

(d) Elevated exposures

22. Elevated exposures result from prolonged presence at high altitudes. Populations living in high-altitude cities like Bogotá, Lhasa or Quito receive annual effective dose equivalents from cosmic radiation in excess of 1 mSv. Passengers and crew members aboard commercial aircraft are exposed to much higher dose rates, which vary according to the flight altitude and, to a smaller extent, the latitude and solar activity. Assuming that the average altitude of commercial subsonic flights is 8 km, the average dose equivalent rate would be about 2 μSv h^{-1} from equations (1.1) and (1.3), the neutron component contributing about 60% of the total. Taking the annual number of hours spent flying by crew members to be 600 h, the corresponding annual effective dose equivalent is about 1 mSv. A small fraction of commercial transport is conducted with supersonic aircraft, which fly at an altitude of about 15 km. Dose equivalent rates received during supersonic transport have been reported to be about 11 μSv h^{-1} on average for French aeroplanes [S64] and about 9 μSv h^{-1} on average for British aeroplanes, with a maximum of around 40 μSv h^{-1} [P14]. Annual dose equivalents received by the technical and cabin crew for the period 1979-1983 were on average about 2.5 mSv, with possible maxima of 15 mSv [P14]. For a given flight, doses received by passengers are about the same as in subsonic flights since the higher dose rates incurred during a supersonic flight are compensated for by the shorter travel time.

23. Using statistical data on air transportation, the collective dose equivalents incurred by the passenger and flight personnel of civil aviation may be estimated. The number of passenger kilometres flown in 1984, excluding Chinese airlines, was 1.3 10^{12} [I15]. Assuming an average speed of 600 km h^{-1} at an altitude of 8 km and an effective dose equivalent rate of 2 μSv h^{-1} at that altitude, the annual collective dose equivalent is found to be 4,300 man Sv. Published estimates [V1, W17] are 3,500 man Sv for the world population and no more than 1,600 man Sv for flights in the United States, while the collective dose equivalent to the flight crews would be up to 65 man Sv. Taking the ratio of the annual collective dose equivalent to the passengers and to the flight crews in the United States as a guide, the annual collective dose equivalent for the flight crews of the world would be about 170 man Sv.

24. While travelling in space, astronauts are subjected to primary cosmic-ray particles, radiation from solar flares and the intense radiation present in the two radiation belts. Radiation doses received by astronauts represent one of the most important constraints to long-term manned space activities [B17]. Reported dose equivalents (assumed to be effective dose equivalents) to astronauts from recent United States

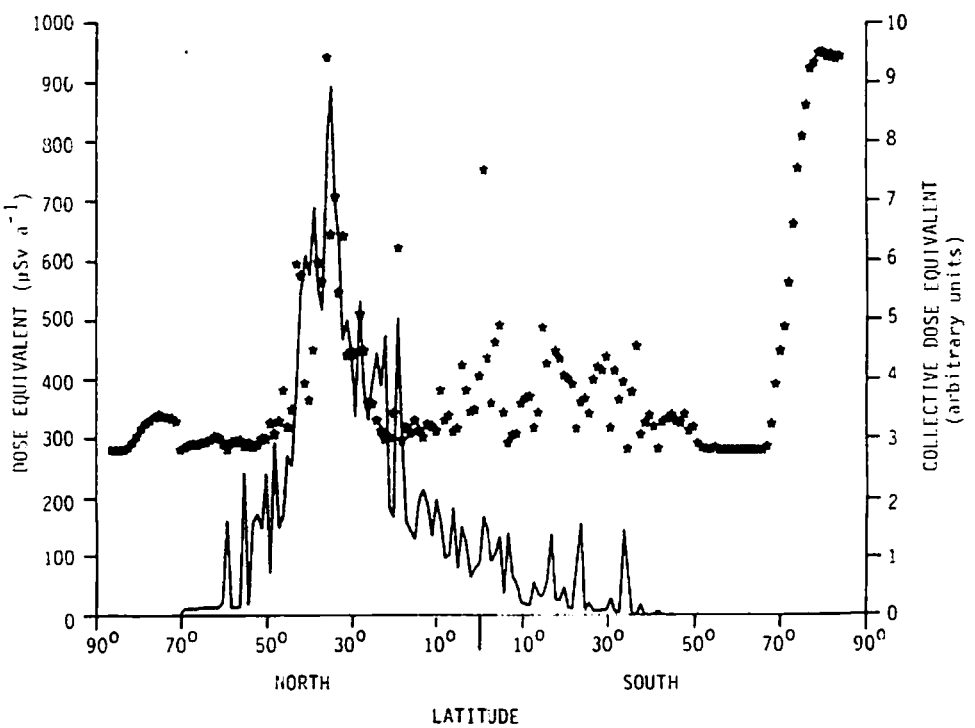

Figure IV. Distribution of the annual effective dose equivalent from cosmic radiation as a function of latitude.
(Based on [B38])

manned space missions range from 0.5 to 5 mSv [B18]. Similar information is available for Soviet missions [V1]. In a majority of cases, individual effective dose equivalents did not exceed 5 mSv per flight; however, for prolonged orbital flights, i.e., those lasting longer than one month, the effective dose equivalent exceeded 10 mSv and reached 55 mSv for expedition IV on board Salut-6, which lasted 175 days [V1].

2. Internal irradiation

25. A large number of cosmogenic radionuclides are produced in the stratosphere and upper troposphere by the interaction of cosmic rays with the nuclei of atoms present in the air (e.g., nitrogen, oxygen and argon). The production and distribution of these nuclides in the environment was reviewed by the Committee in the UNSCEAR 1977 Report [U2]. Only four cosmogenic radionuclides (^{3}H, ^{14}C, ^{7}Be and ^{22}Na) are of importance from the viewpoint of radiation doses to man. Together they deliver annual doses from internal irradiation ranging from 5 to 25 μGy in the various organs and tissues of interest. The annual effective dose equivalents are estimated to be 0.01 μSv for ^{3}H, 3 μSv for ^{7}Be, 12 μSv for ^{14}C and 0.2 μSv for ^{22}Na. Because of the relative homogeneity of the cosmic-ray flux over the earth's surface, the variability of the annual doses from the cosmogenic radionuclides is expected to be low.

B. TERRESTRIAL SOURCES OF RADIATION

26. Terrestrial sources of radiation are the very long-lived radionuclides that have existed within the earth since its formation several billion years ago and have not substantially decayed. The most important of these so-called primordial radionuclides are ^{40}K (half-life = 1.28 10^{9} a), ^{87}Rb (half-life = 4.7 10^{10} a), ^{238}U (half-life = 4.47 10^{9} a) and ^{232}Th (half-life = 1.41 10^{10} a). ^{238}U and ^{232}Th head series of 14 and 11 significant radionuclides, respectively, (Figures V and VI) that also contribute to the doses from terrestrial sources. Other radionuclides, such as those present in the ^{235}U decay series, have been neglected, as they contribute little to the total dose from natural background.

27. The ubiquitous presence of the primordial radionuclides and of their decay products in the environment (air, water, soil, rocks, foodstuffs) and in humans results in external and internal radiation doses.

1. External irradiation

(a) Exposure outdoors

28. Activity mass concentrations of primordial radionuclides in rocks are usually higher in igneous rocks than in sedimentary ones, while metamorphic rocks have concentrations typical of the rocks from which they are derived. There are, however, exceptions, as

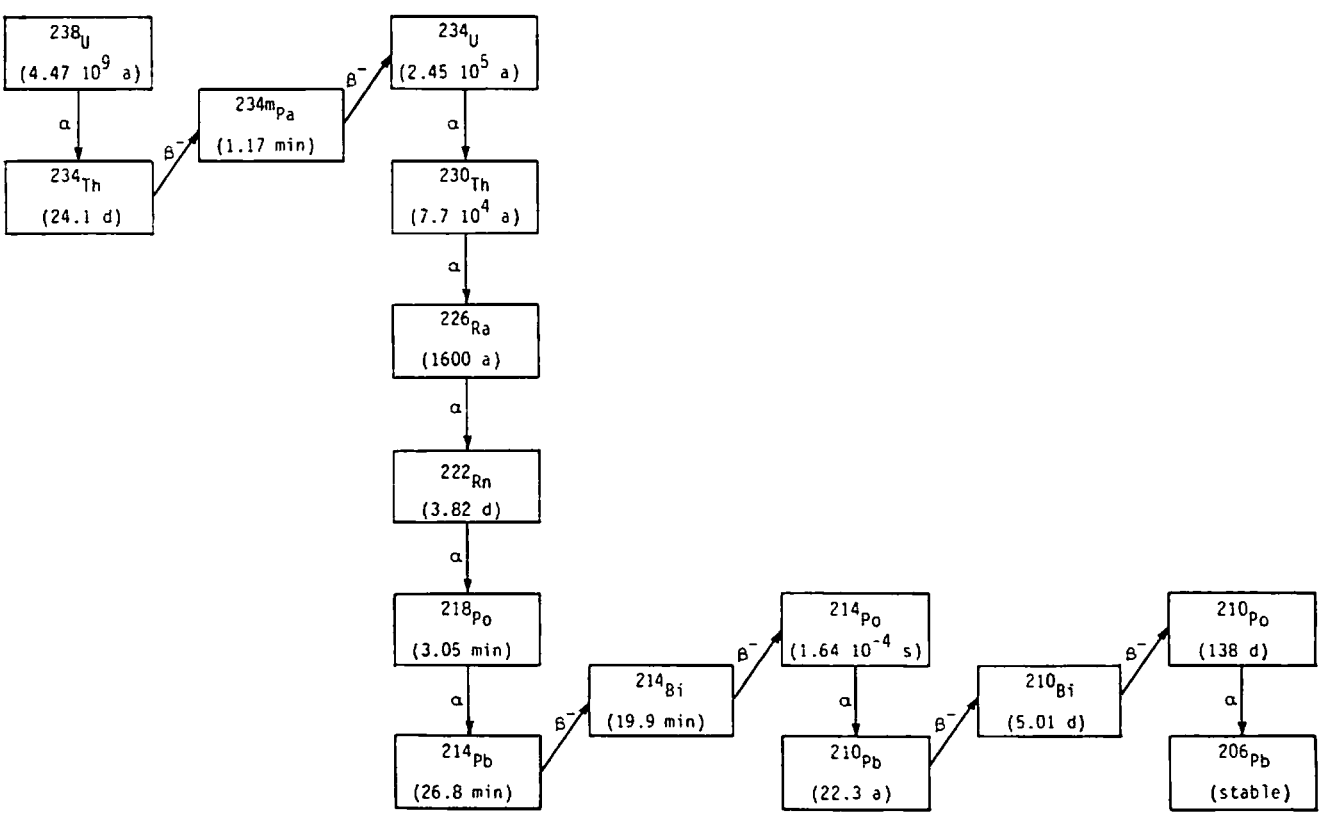

Figure V. Uranium-238 decay series.
[I10]

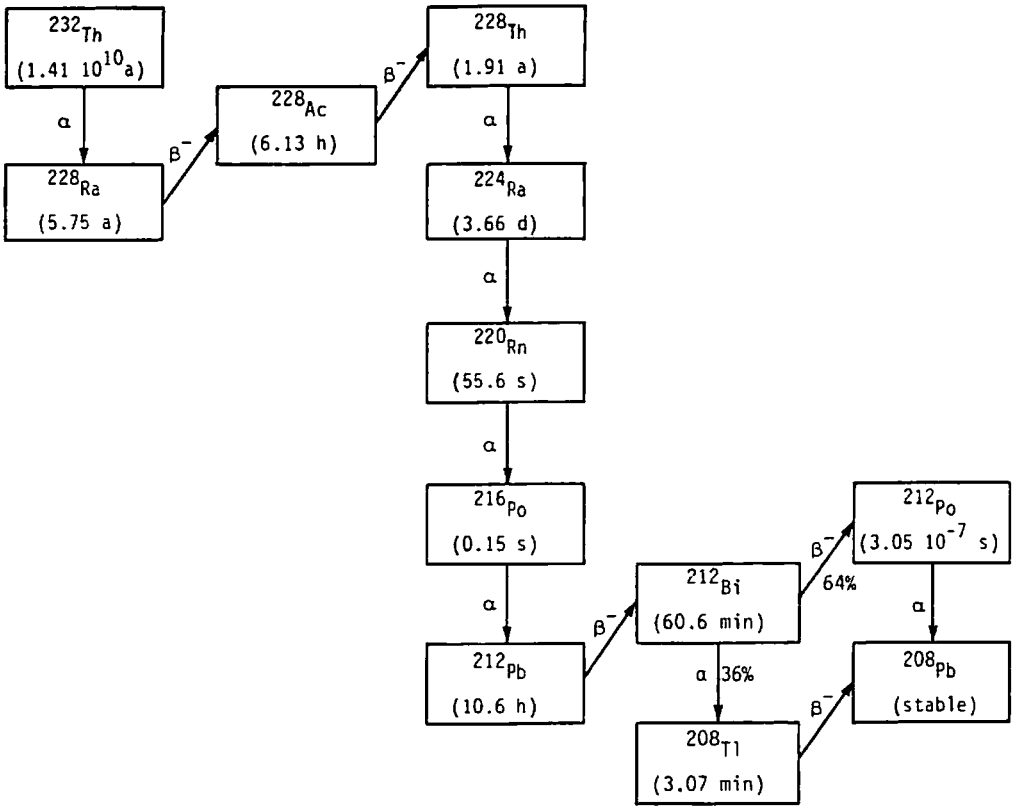

Figure VI. Thorium-232 decay series.
[I10]

certain sedimentary rocks, notably some shales and phosphate rocks, are highly active. The activity mass concentrations in soil, which are directly relevant to outdoor exposure, are thought to be largely determined by the activity mass concentrations in the source rock. The average activity mass concentrations of ^{40}K, ^{238}U and ^{232}Th in soil and the corresponding absorbed dose rates in air 1 m above the ground surface are given in Table 3 [B32]; the calculations are for a soil density of 1.6 g cm^{-3} and a water content of 10% and are based on the assumption that all decay products of ^{238}U and ^{232}Th are in radioactive equilibrium with their precursors. The average outdoor terrestrial absorbed dose rate in air from gamma-radiation 1 m above the ground surface is 44 nGy h^{-1}. The results of a large soil sampling programme recently carried out in the United States are in fairly good agreement with the activity concentrations given in Table 3 [M10].

29. Large-scale surveys, using different methods and types of instrumentation, have been or are being carried out in a number of countries in order to estimate average nation-wide exposures to outdoor external gamma-radiation. The results for 23 countries or areas, representing about one half of the world population, are summarized in Table 4. Country-averaged outdoor absorbed dose rates in air are found to range between 24 and 85 nGy h^{-1}, with an arithmetic mean of 55 nGy h^{-1}.

30. It is to be noted that the two most populated countries in the world, China and India, have recently published the results of extensive surveys. In China, about 40,000 outdoor measurements were carried out by the Ministry of Public Health from 1981 to 1985, using sodium iodide crystals and ionization chambers in 21 provinces, five autonomous regions and the cities of Beijing, Tianjin and Shanghai. Detailed preliminary results are found in a special issue of the Chinese Journal of Radiological Medicine and Protection [C19] and a summary has been prepared by Wang Qiliang et al. [W21]. Population-weighted means of the absorbed dose rates in air are presented in Table 5; the value for the country as a whole is reported to be about 80 nGy h^{-1}, which is in the upper range of the levels observed in the world. These results led to a large soil-sampling programme that showed that the average concentrations of ^{40}K, ^{238}U and ^{232}Th in soil in China are indeed higher than the estimates by UNSCEAR of the world averages by a factor of about 2 [W21]. Independent survey programmes of outdoor gamma-radiation have also been undertaken in China by the National Environmental Protection Agency [L18] and by the Ministry of Nuclear Industry [G29]. A comparison of the results obtained for the same areas [P22] shows discrepancies that may have arisen from the energy dependence of the various types of dosimeters used in the surveys and from the estimation of the cosmic-ray dose rates which were subtracted from the readings. The average absorbed dose rates in air in the cities, provinces and autonomous regions investigated by the Ministry of Public Health [W21] were found to be lower than those measured by the National Environmental Protection Agency and the Ministry of Nuclear Industry, the relative differences ranging from 11% to 63% [P22]. Since the survey of the Ministry of Public Health is the only one completed at the time of publication of

this Annex, its results have been incorporated without modification; however, it should be kept in mind that the gamma doses for China may be revised in the near future.

31. In India, 214 locations scattered all over the country were monitored with thermoluminescent dosimeters. The national mean value of the outdoor absorbed dose rate in air due to natural radiation (terrestrial sources of radiation and cosmic radiation) is estimated to be 785 μGy a^{-1} [N19]. Assuming a mean dose rate from cosmic rays of 300 μGy a^{-1} yields an average absorbed dose rate in air from the terrestrial component of 55 nGy h^{-1}.

32. In the UNSCEAR 1977 Report [U2], the Committee adopted a value of 50 nGy h^{-1} for the population-weighted average absorbed dose rate in air over the world and estimated that 95% of the world's population residing in areas of usual natural radiation live where the outdoor absorbed dose rate in air from the primordial radionuclides lies between 30 and 70 nGy h^{-1}. As shown in Table 4, the available data base is currently much larger than it was in 1977. The population-weighted average absorbed dose rate in air for the countries in Table 4 is 63 nGy h^{-1}. This is likely to be an overestimate because of the probable bias in the Chinese measurements. When the Chinese data are not taken into account, the population-weighted average absorbed dose rate in air is reduced to 53 nGy h^{-1}. An intermediate figure of 55 nGy h^{-1} is adopted in this Annex as the mean absorbed dose rate in air outdoor from terrestrial sources of radiation.

33. There are regions in the world where the usual range of variation of outdoor terrestrial radiation doses is substantially exceeded. The best documented of those regions are in Brazil and India. In the coastal areas of Kerala and Tamil Nadu in India, there are patches of sand containing monazite with thorium concentrations ranging between 8% and 10.5% by weight. The absorbed dose rates in air in the high radiation areas of Kerala vary between 150 and 1,000 nGy h^{-1}; in Tamil Nadu, these may reach about 6,000 nGy h^{-1} [S69]. In Brazil, outdoor absorbed dose rates of 130-1,200, 220-4,200 and 110-1,000 nGy h^{-1} were measured in Guarapari, Meaipe and Poços de Caldas, respectively [P24].

(b) Exposure indoors

34. Knowledge of radiation levels in buildings is important in the assessment of population exposures, as most individuals spend a large amount of time indoors. Large-scale surveys of indoor exposures to gamma external radiation have recently been conducted in several European countries as well as in China; the results are summarized in Table 6. An estimation of the world distribution of exposures may be derived from the results presented in Table 6 or, as in previous UNSCEAR Reports, from the indoor-to-outdoor ratios.

35. Estimates of average indoor absorbed dose rates in air are between 23 and 120 nGy h^{-1}, most values being in the narrow range of 60-95 nGy h^{-1} (Table 6). The arithmetic mean is about 70 nGy h^{-1}. The population-weighted mean for all countries except China is 72 nGy h^{-1} and becomes 110 nGy h^{-1} if China is included.

36. In previous UNSCEAR Reports, use was made of the indoor-to-outdoor ratio, under the assumption that the relationship between the indoor and the outdoor absorbed dose rates depends essentially on the type of building materials used and their origin. If the building materials are of local origin, it may be expected that the value of the indoor-to-outdoor ratio of the absorbed dose rates in air lies between 1 and 2, because of the change in source geometry and the presence of doors and windows. Calculations taking into account the thickness and the dimensions of the walls yield ratios of 1.35 for typical brick dwellings and 1.48 for concrete buildings [K11].

37. Country-averaged indoor-to-outdoor ratios, derived from the results presented in Tables 4 and 6, range from 0.8 to 2.0, with an average of 1.3. This figure, combined with an average outdoor absorbed dose rate in air of 55 nGy h^{-1}, yields an average indoor absorbed dose rate in air of 72 nGy h^{-1}, in close agreement with some of the values obtained by the first method. In this Annex, the mean indoor absorbed dose rate in air from terrestrial sources of radiation is taken to be 70 nGy h^{-1}.

38. Elevated external dose rates indoors may arise from high concentrations of natural radionuclides in building materials. These building materials may be of natural origin (concrete based on alum shale, granite, lithoid tuff) or result from industrial processes (phosphogypsum, red mud). As discussed in the UNSCEAR 1982 Report, the resulting exposures, calculated with pessimistic assumptions, range between 100 and 2,000 nGy h^{-1}.

(c) Annual effective dose equivalents from gamma terrestrial radiation

39. The value of the quotient of effective dose equivalent rate to absorbed dose rate in air is taken, as in the UNSCEAR 1982 Report, to be 0.7 Sv per Gy for environmental exposures to gamma rays of moderate energy. This value is assumed to apply equally to males and females and to the indoor and outdoor environments. Taking the outdoor occupancy factor to be 0.2, the annual effective dose equivalent from outdoor terrestrial gamma-radiation is found to be

$$55\ (\text{nGy h}^{-1}) \times 0.7\ (\text{Sv Gy}^{-1}) \times 8{,}760\ (\text{h a}^{-1}) \times 0.2 = 70\ \mu\text{Sv}$$

For indoor exposure, using an occupancy factor of 0.8, the annual effective dose equivalent is

$$70\ (\text{nGy h}^{-1}) \times 0.7\ (\text{Sv Gy}^{-1}) \times 8{,}760\ (\text{h a}^{-1}) \times 0.8 = 340\ \mu\text{Sv}$$

40. The total (outdoor plus indoor) annual effective dose equivalent from terrestrial radiation, averaged over the world's population, is 410 μSv. Using the information presented in Table 3 as a guide, the contributions of ^{40}K and the radionuclides of the ^{238}U and ^{232}Th decay series to the total annual effective dose equivalent from gamma terrestrial radiation would be 150, 100 and 160 μSv, respectively.

2. Internal irradiation

41. Inhalation and ingestion of naturally occurring radionuclides give rise to internal irradiation. As in the previous reports of the Committee, the absorbed doses and effective dose equivalents are derived from measured tissue concentrations, in so far as such data are available.

(a) Potassium-40

42. Potassium is an essential element that is under close homeostatic control in the body. The average mass concentration for an adult male is about 2 g of potassium per kg of body weight [A4, K12]. The isotopic ratio of ^{40}K is $1.18\ 10^{-4}$ and the average activity mass concentration of ^{40}K in the body is about 60 Bq kg^{-1}. The distribution of potassium in various tissues and organs of the body [K12] has been used to determine the concentrations of ^{40}K in those tissues and organs and the corresponding absorbed dose rates. The highest annual absorbed dose (270 μGy) is received in red bone marrow and the lowest in the thyroid (100 μGy). The annual effective dose equivalent is estimated to be 180 μSv. The variation of the concentration of potassium in the entire body reflects the amount of relatively potassium-free adipose tissue that is present in lean body mass at different ages: it is found to vary between about 1 and 2.5 g kg^{-1}.

(b) Rubidium-87

43. Very little is known about the behaviour of rubidium in the environment. Limited measurements in foodstuffs and humans seem to indicate that the human body retains rubidium more so than potassium [T12]. from the mass concentrations of rubidium reported on by ICRP [I3], the average activity mass concentration of ^{87}Rb in the body is 8.5 Bq kg^{-1}. The assumed distribution of rubidium in various organs and tissues of the body [I3] is used to calculate the resulting absorbed dose rates from ^{87}Rb. Bone lining cells receive the highest annual dose (14 μGy) and the thyroid the lowest (3 μGy). The annual effective dose equivalent is estimated to be about 6 μSv.

(c) Uranium-238 series

44. Uranium-238 is the head of a series of 14 principal nuclides (Figure V). This can be divided into subseries in which the activity of the precursor controls to a large degree the activities of the decay products: $^{238}U \rightarrow {}^{234}U$; ^{230}Th; ^{226}Ra; $^{222}Rn \rightarrow {}^{214}Po$; and $^{210}Pb \rightarrow {}^{210}Po$. For each subseries, the intakes by inhalation and by ingestion, as well as the concentrations in bone and in soft tissues, are estimated. The results are presented in Tables 7 and 8. The conversions from the activity concentrations in bone and in soft tissues to the absorbed doses are based on the models described in ICRP Publication 30 [I4]. Estimates of annual absorbed doses are shown in Table 9. Presented below is a brief discussion of the exposures with an indication of what new information has become available since the publication of the UNSCEAR 1982 Report.

(i) Uranium-238 subseries (^{238}U, ^{234}Th, ^{234m}Pa, ^{234}U)

45. In this Annex, uranium is assumed to consist of ^{238}U in radioactive equilibrium with ^{234}Th, ^{234m}Pa and ^{234}U, so that 1 kg of uranium contains 12 MBq of each of the four radionuclides. The contribution of ^{235}U and its decay products to the total dose from natural background has been neglected.

46. In the atmosphere, the main natural source of uranium, as well as of any other precursor of one of the radon isotopes, is likely to be the resuspension of dust particles from the earth [H24, H25, K21]. Taking a dust loading of about 50 μg m^{-3} in surface air of populated areas and assuming an average ^{238}U activity mass concentration in airborne dust as in soil of 25 Bq kg^{-1} (Table 3), the activity concentration in ground-level air is estimated to be about 1.2 μBq m^{-3}. The corresponding annual intake by adults through inhalation is approximately 0.01 Bq (Table 7). This result is applicable to all other radionuclides of the ^{238}U and ^{232}Th series that are precursors of gaseous ^{222}Rn and ^{220}Rn.

47. The annual dietary intake of ^{238}U has been found to be about 5 Bq in areas of normal natural activity (based on results from Japan, United Kingdom, United States). Recent measurements of uranium in the diet of New York City residents confirm the validity of this value [F12]. The contribution of drinking water to the total intake by ingestion varies over a wide range [H26]. According to the results of an extensive survey carried out in the United States, involving 90,000 measurements of domestic water supplies, average concentrations of uranium in ground water, for a given state, are only slightly higher (up to a factor of 4) than in surface water, and the ratio of the activity concentration of ^{234}U to that of ^{238}U usually ranges between 1 and 3, with an average value of 1.9 [D10, H20, H21]. It can be estimated from that survey that the population-averaged ^{238}U concentrations in domestic water supplies range from 0.1 Bq m^{-3} in several eastern states to 50 Bq m^{-3} in a number of western states, the nation-wide average in ground-water supplies being 25 Bq m^{-3} [D10, H21]. Uranium-238 concentrations in European mineral water are similar to those observed in United States ground water, with median values of 12 Bq m^{-3} for France and 24 Bq m^{-3} for the Federal Republic of Germany. If the average daily consumption of drinking water is taken to be 0.5 litre, the average annual intake from drinking water containing 25 Bq of ^{238}U per m^3 is about 5 Bq, which is the same value as that found for the intake from foodstuffs. This is an example of higher intake; however, the mean intake of uranium through ingestion of water is generally likely to be small in comparison with the mean dietary intake.

48. Measured values of the activity mass concentration of ^{238}U in bone of adults who have lived in areas with normal dietary levels are currently more abundant [F13, F16, F18, W19]; they lie in the range of 5-150 mBq kg^{-1} of ^{238}U in dry bone. Analyses of this wider data base indicates that a better value of the average activity mass concentration is 50 mBq kg^{-1}, which is lower by a factor of 3 than the previous

estimate by the Committee. The activity mass concentrations of ^{238}U in soft tissues (Table 8) are much smaller, with the exception of the lungs, and previously measured high values in kidneys have not been confirmed [F16, W19]. The estimated annual absorbed doses from the ^{238}U subseries are up to about 1 μGy in bone lining cells (Table 9); the corresponding annual effective dose equivalent is 5 μSv.

(ii) Thorium-230

49. The activity intake of ^{230}Th through inhalation, estimated in the same manner as that of ^{238}U, is about 0.01 Bq a^{-1}. The dietary intake of ^{230}Th has been measured in the United States for the first time; it is about 2 Bq a^{-1} [F12]. It can be derived from these values and ICRP models [I4] that inhalation accounts for about three quarters of the total uptake to the blood of ^{230}Th.

50. Thorium is a bone seeker having a long residence time in the skeleton and is assumed to remain on the bone surfaces [I4]. The distribution of ^{230}Th in human tissues has been investigated by Wrenn and his collaborators [I3, S22, S55, W9]. A typical activity of ^{230}Th in bone is 140 mBq, resulting in activity mass concentrations of approximately 20 mBq kg^{-1} in dry cortical bone and 70 mBq kg^{-1} in dry trabecular bone, assuming that the deposit of thorium is proportional to the bone area. In soft tissues, representative values of the activity mass concentrations could be 300 mBq kg^{-1} in lymph nodes, 20 mBq kg^{-1} in the lungs, 10 mBq kg^{-1} in kidneys, 7 mBq kg^{-1} in liver and 0.3 mBq kg^{-1} in other soft tissues. The corresponding annual absorbed doses (Table 9) range from 7 nGy to 7 μGy, depending on the organ or tissue considered. The annual effective dose equivalent is estimated to be about 7 μSv.

51. The doses in bone marrow and bone lining cells, as well as the effective dose equivalent, would be significantly lower if the activity of ^{230}Th were distributed uniformly over the mass of the skeleton instead of being concentrated on the bone surfaces. The activity mass concentration of ^{230}Th would then be about 30 mBq kg^{-1} in dry trabecular and cortical bone, while the calculated annual doses in bone marrow and bone lining cells would be 0.02 and 0.4 μGy, respectively, instead of 0.56 and 7.4 μGy (see Table 9). The annual effective dose equivalent would be reduced from 7 to 2 μSv.

(iii) Radium-226

52. Food is a much more important source of radium for intake and blood uptake than is inhalation. The Committee's previous estimate of the average annual dietary intake of ^{226}Ra in areas of normal radiation background of 15 Bq has been recently confirmed in the USSR [D11] and the United Kingdom [S56] and is to be compared with 0.01 Bq for inhalation. The contribution of drinking water to the total intake is generally small when the drinking-water supplies are drawn from surface water; however, in ground-water supplies, which in many countries serve a large portion of the population, ^{226}Ra concentrations vary widely and levels in excess of 200 Bq m^{-3} are not uncommon (see, for example, [C18] and [H20]). Concentrations in bottled mineral water from European countries range up to 1,800 Bq m^{-3} and have geometric means of 7, 44, 25 and 25 Bq m^{-3} in Italy, France, Austria and the Federal Republic of Germany, respectively [F17, G27, M28, R8]. In the United States, the reported geometric mean of the ^{226}Ra concentration in public water supplies is 22 Bq m^{-3}. This leads to an annual intake of 4 Bq, if a water consumption rate of 0.5 litre per day is assumed.

53. When radium is taken into the body, its metabolic behaviour is similar to that of calcium, and an appreciable fraction is deposited in bone [E5]. More than 70% of the radium in the body is contained in bone [I5, S57], the remaining fraction being distributed rather uniformly in soft tissues. Fisenne et al. [F4] have summarized the available data from 26 countries on measured activity mass concentrations of ^{226}Ra in human bone. The 26 countries sampled have 1.4 10^9 persons and thus represent about 30% of the world population. The population-weighted distribution was found to have a median of 850 mBq per kg of calcium (corresponding to 170 mBq per kg of dry bone and to 850 mBq in the skeleton) and a geometric standard deviation of 1.6. If the fraction of ^{226}Ra distributed in the soft tissues is taken to be 17%, as given in ICRP publication 20 [I5], the average activity mass concentration in human soft tissues is found to be 2.7 mBq kg^{-1}.

54. The annual absorbed doses in tissues have been calculated assuming that an average retention factor of 0.33 applies to ^{222}Rn in the skeleton (and also, conservatively, in soft tissues) and that the concentration of radium and its decay products is uniform over the total mass of mineral bone. The results, presented in Table 9, show average annual absorbed doses of less than 1 μGy in all organs and tissues, with the exception of bone lining cells. The annual effective dose equivalent resulting from ^{226}Ra intake in normal areas is found to be about 7 μSv.

55. Food samples grown in the high-radiation areas of India have been collected and analysed for several radionuclides, including ^{226}Ra. The ^{226}Ra concentrations were found to be higher in leafy and root vegetables than in fruits and fruit vegetables. The annual intake of ^{226}Ra by populations living in the high background areas was assessed to be about 200 Bq [L19]. A similar survey performed in the Araxa-Tapira region of Brazil showed that the annual ^{226}Ra intakes of the most exposed people ranged between 140 and 540 Bq [P25]. The corresponding annual effective dose equivalents are between 65 and 250 μSv.

(iv) Radon-222 and its short-lived decay products (^{218}Po, ^{214}Pb, ^{214}Bi, ^{214}Po)

56. Because of the current scientific interest in radon exposure, a detailed treatment is provided in chapter II.

57. Average annual intakes, activity mass concentrations in lung tissues and absorbed doses are presented in Tables 7, 8 and 9, respectively. The resulting annual effective dose equivalents are estimated to be, on

average, 70 μSv for outdoor exposure and 1,000 μSv for indoor exposure. The variability around these average figures is very high.

(v) Long-lived decay products of radon-222 (^{210}Pb, ^{210}Bi, ^{210}Po)

58. Radon-222 exhalation from the ground constitutes the main source of ^{210}Pb in the atmosphere. With respect to ^{210}Po, the volcanic output has been estimated to be about as important as ^{222}Rn exhalation [L10]. In the mid-latitudes of the northern hemisphere, the average concentration of ^{210}Pb in surface air is 0.5 mBq m^{-3}, while that of ^{210}Po is about 0.04 mBq m^{-3}, as shown in Table 10; this is lower than the value adopted in the UNSCEAR 1982 Report by a factor of 2.5. Assuming that the concentrations in air are the same indoors and outdoors, the annual intakes of non-smokers through inhalation would be 4 Bq of ^{210}Pb and 0.3 Bq of ^{210}Po. Cigarette smoking leads to an increase in the intake of ^{210}Pb and ^{210}Po [M13, P9]. A cigarette contains about 20 mBq of ^{210}Pb and 15 mBq of ^{210}Po [P9] and both nuclides are volatile at the burning temperature of tobacco. About 10% of the ^{210}Pb and 20% of the ^{210}Po contained in the cigarette will enter the lungs with the main smoke stream [P9]. Therefore, for a person smoking 20 cigarettes a day, the values of the estimated annual intakes are 15 Bq for ^{210}Pb and 20 Bq for ^{210}Po. In view of the short half-life of ^{210}Bi (5.01 d), its activity intakes are of no importance, as ^{210}Bi may be assumed to be in radioactive equilibrium with ^{210}Pb in the body tissues; the absorbed doses from ^{210}Bi arise mainly from the intake of ^{210}Pb and not from the intake of ^{210}Bi itself.

59. Consumption of food is usually the most important route by which ^{210}Pb and ^{210}Po enter the human body. Concentrations of ^{210}Pb and ^{210}Po are usually low in meat and milk, intermediate in cereals and vegetables, and relatively high in aquatic organisms. Annual intakes reflect the composition of the diet of the population in question and are approximately 20 Bq in the United States [H13, H14], 30 Bq in the United Kingdom [S56], about 40 Bq in the Federal Republic of Germany [G17], the USSR [L9], India [L13] and Italy [C12], and about 200 Bq in Japan [O7, T5]. More recent data from Japan, however, seem to indicate a much lower annual intake of 11 Bq [K18]. Concentrations of ^{210}Pb in drinking water are generally low [H20, G27] and do not usually contribute significantly to the total intake by ingestion.

60. A well-documented case of elevated intake is that of the tens of thousands of individuals living on reindeer or caribou meat in the Arctic and sub-Arctic regions of the northern hemisphere. Their main food is the meat of these animals, which contains unusually high concentrations of ^{210}Po because in the winter the animals graze on lichens, which accumulate ^{210}Pb and ^{210}Po. The annual intake of ^{210}Pb and ^{210}Po by the populations living on reindeer or caribou meat are about 140 Bq for ^{210}Pb and about 1,400 Bq for ^{210}Po [H14, P12].

61. Lead is a bone seeker that is found incorporated in mineral bone and has a long residence time in the skeleton. In continental areas, a typical activity mass concentration of ^{210}Pb in dry bone is about 3 Bq kg^{-1}, yielding a skeleton content of 15 Bq. The measured ratios of ^{210}Po and ^{210}Pb activity mass concentrations in bone are centred around 0.8, leading to a ^{210}Po skeleton content of 12 Bq.

62. About 30% of the body content of ^{210}Pb is found in soft tissues, with a relatively uniform distribution throughout the body. The degree of radioactive equilibrium between ^{210}Po and ^{210}Pb depends on the organ considered; it is about 0.5 in the lungs, 0.8 in red bone marrow, and definitely greater than 1 in the liver and kidneys. Additional intake due to smoking leads to increased concentrations, particularly in the lungs.

63. The absorbed doses from the ^{210}Pb subseries depend mainly on the highly energetic alpha particles of ^{210}Po, as the contribution from the beta emissions of ^{210}Pb and ^{210}Bi amounts to about 10% of the total. The estimated annual absorbed doses of non-smokers in areas of normal dietary intake (Table 9) are about 5 μGy in soft tissues and 36 μGy in bone lining cells. The annual effective dose equivalents arising from the total intake of ^{210}Pb, ^{210}Bi and ^{210}Po are about 120 μSv. The corresponding figure for the populations living on reindeer or caribou meat would be about 10 times higher.

(d) Thorium-232 series

(i) Thorium-232

64. Thorium-232 is the head of a series of 11 radionuclides (Figure VI). The ^{232}Th series has been divided into three subseries: ^{232}Th itself; ^{228}Ra → ^{224}Ra; and ^{220}Rn → ^{208}Pb. For each subseries, the intakes and concentrations are estimated as in the case of ^{238}U and the results are presented in Tables 7 and 8. Estimates of annual absorbed doses are shown in Table 9.

65. The activity mass concentration of ^{232}Th in soil is estimated to be on average 25 Bq kg^{-1}, the same as that of ^{238}U and its decay product ^{230}Th (Table 3). The annual intake from inhalation is estimated to be 0.01 Bq while that from ingestion, recently measured in the United States for the first time [F12], is about 2 Bq. Wrenn and his collaborators found the activity mass concentrations of ^{232}Th in the body to be lower than those of ^{230}Th by a factor of about 2 [S22, W9]. On the basis of their measurements, the body content of ^{232}Th would be about 80 mBq, 60% of which is in the skeleton. The activity mass concentrations adopted in this Annex and the resulting annual absorbed doses are presented in Tables 8 and 9, respectively. The annual effective dose equivalent is calculated to be about 3 μSv.

66. The dose calculations have assumed that ^{232}Th remains on bone surfaces. A volume distribution would yield an annual effective dose equivalent of about 1 μSv.

(ii) Radium-228 subseries (^{228}Ra, ^{228}Ac, ^{228}Th, ^{224}Ra)

67. Radium is much more available to plants and to animals than ^{232}Th, so that the activity concentrations

of ^{228}Ra in humans are mostly due to the dietary intake of ^{228}Ra itself and not to the decay of ^{232}Th in the body. Radium-228 can thus be considered to be the head of a subseries in which ^{228}Th (half-life: 1.91 a) and ^{224}Ra (half-life: 3.66 d), both alpha emitters, are the most important contributors to the dose.

68. The annual activity intake arising from inhalation is estimated to be 0.01 Bq, while that from ingestion of foods is considerably larger, about 15 Bq in areas of normal radiation background and about 2,000 Bq in the high background area along the Kerala coast in India. Radium-228 concentrations in drinking water are comparable to those of ^{226}Ra but not systematically correlated when individual water supplies are considered. They are low in surface water and extremely variable in ground water [H20]. The geometric mean of the ^{228}Ra concentrations in public ground-water supplies in the United States is reported to be 85 Bq m^{-3} [H21], corresponding to an annual intake of 15 Bq for a daily drinking-water consumption of 0.5 litre.

69. The Committee estimated the average ^{228}Ra activity mass concentrations in bone (dry weight) and in soft tissues (wet weight) of the human body to be 50 mBq kg^{-1} and 4 mBq kg^{-1}, respectively, in areas of normal background radiation [U1, U2]. Regarding ^{228}Th, Wrenn and Singh [W9] showed that approximately 80% of the body content (about 300 mBq) is in bone.

70. The annual absorbed doses in tissues have been calculated assuming that the ^{220}Rn activity arising from the decay of ^{224}Ra is retained in the body and that the concentrations of ^{228}Ra and of its decay products are uniform over the total mass of bone. The results presented in Table 9 show annual absorbed doses greater than 1 μGy in the lungs, kidneys and bone lining cells. The corresponding annual effective dose equivalent for the subseries is found to be about 13 μSv.

(iii) Radon-220 and its decay products (^{216}Po, ^{212}Pb, ^{212}Bi, ^{212}Po, ^{208}Tl)

71. As is the case of ^{222}Rn, inhalation is the major pathway through which humans are exposed to ^{220}Rn (thoron) and its short-lived decay products. In outdoor air, the few available measurements of thoron decay products [U1, Annex D, Table 23] point to an average ^{212}Pb/^{222}Rn activity concentration ratio of about 0.04. If the average ^{222}Rn concentration is taken to be 5 Bq m^{-3}, the average concentration of ^{212}Pb in outdoor air, representative of the equilibrium equivalent concentration of thoron decay products, would be 0.2 Bq m^{-3}. The equilibrium equivalent concentration (EEC) of thoron, or radon, is that activity concentration of thoron, or radon, in radioactive equilibrium with its short-lived decay products that has the same potential alpha energy concentration as the actual mixture of decay products. Ground-level air thoron concentrations in continental areas lie in the range of 2-10 Bq m^{-3} [F11].

72. The air exchange rate indoors is always much smaller than the radioactive decay constant of thoron (half-life = 55.6 s), so that the thoron concentration in room air is relatively insensitive to the value of the ventilation rate. The thoron concentration in room air is therefore mainly determined by the exhalation rate from the soil and building materials. As the diffusion length of thoron in these materials is of the order of 1 cm, it is expected that the nature of the surface layer covering the floor and the walls has a great influence on the thoron exhalation rate; however, measurements are needed to substantiate this assumption. The magnitude of the equilibrium factor between thoron and its decay products in room air has been theoretically estimated by Porstendorfer et al. [P4]. The difference between the concentrations of ^{212}Pb and ^{212}Bi is always found to be small but there is a factor of about 10-50, depending on the ventilation rate, between the concentrations of ^{220}Rn (or ^{216}Po) and ^{212}Pb (or ^{212}Bi), the concentrations of ^{220}Rn being higher. The unattached fraction of ^{212}Pb and ^{212}Bi is very small (less than 1%) [P4].

73. The number of indoor measurements of thoron decay products is small in comparison to that of radon decay products. Table 11 summarizes the available information on the potential alpha energy ratio of thoron and radon decay products measured simultaneously. The average values are around 0.5. Using this figure and assuming a typical equilibrium equivalent concentration of radon of 20 Bq m^{-3} in temperate latitudes (paragraph 140), the average equilibrium equivalent concentration of thoron (Tn) is tentatively estimated to be

$$(20\ \text{Bq m}^{-3})_{Rn} \times (55.4\ 10^{-10}\ \text{J Bq}^{-1})_{Rn} \times [(0.5\ \text{J})_{Tn}]/[(1\ \text{J})_{Rn}] \times [1\ \text{Bq}/(757\ 10^{10}\ \text{J})]_{Tn} \simeq 0.7\ \text{Bq m}^{-3}$$

in temperate latitudes.

74. For equatorial regions measurements are unavailable. Because of the different domestic conditions, the indoor concentrations of thoron decay products are expected to be lower than in temperate regions. To estimate the population-weighted world average of the indoor concentrations of thoron decay products, account must be taken of the fact that the population in tropical regions is about half that in temperate latitudes. The range of possible values for that mean concentration for the world is from 0.47 Bq m^{-3} (assuming that the indoor concentration in tropical latitudes is equal to zero) to 0.7 Bq m^{-3} (assuming that the indoor concentration in tropical regions is the same as that in the temperate zones). It is tentatively assumed that the population-weighted world average of the indoor concentration of thoron decay products is 0.5 Bq m^{-3}.

75. Regarding exposure-dose relationships, like radon and its decay products (see chapter II), thoron gas is considered separately from thoron decay products.

76. Inhaled thoron, as a noble gas, is constantly present in the air volume of the lungs at the concentration in the inhaled air; in addition, it is partly dissolved in the lung tissues. Due to its short radioactive half-life (55.6 s), however, the equilibrium solubility in tissues other than that of the lungs will not be reached. Thus, the decay of thoron and of its

very short-lived decay product ^{216}Po leads mainly to a dose in the lungs; the second decay product formed, ^{212}Pb, is mainly transferred to blood cells, kidneys, and bone surfaces [I2].

77. Values of annual absorbed doses per unit thoron concentrations have been derived from calculations by Jacobi and Eisfeld [J6]. They are presented in Table 12. For concentrations of thoron gas in air in the range of 2-20 Bq m^{-3}, the annual doses are up to 5 μGy in the lungs, 4 μGy in bone lining cells and 2 μGy in the kidneys. The corresponding annual effective dose equivalents are less than 20 μSv.

78. With respect to thoron decay products, the absorbed doses per unit inhaled activity have been adapted from calculations of Jacobi and Eisfeld [J11] to fit the value of 0.7 Sv J^{-1} used in the UNSCEAR 1982 Report [U1] for the effective dose equivalent per unit inhaled potential alpha energy. The results, presented in Table 12, show that the lungs, bone lining cells and kidneys are by far the most exposed organs and tissues. The absorbed doses per unit concentration, also shown in Table 12, have been estimated using a mean breathing rate of 0.8 m^3 h^{-1} indoors and 1 m^3 h^{-1} outdoors and an average occupancy factor of 0.8 indoors and 0.2 outdoors.

79. The dosimetric coefficients of Table 12 have been used to estimate average annual doses from inhalation of thoron decay products. Average annual doses in the lungs are 5 μGy outdoors and about 40 μGy indoors. Annual doses in other organs and tissues are lower. The annual average effective dose equivalent resulting from outdoor and indoor inhalation of thoron is estimated to be 160 μSv.

80. Because of the few data available, the distribution of individual exposures, as well as the reliability of the estimated mean value, is difficult to assess at present.

C. SUMMARY

81. A summary of the various contributions to the annual effective dose equivalent from natural sources of radiation is given in Table 1. In order of importance, inhalation of short-lived decay products of radon comes first, with the average annual effective dose equivalent estimated to be 1,100 μSv. A detailed discussion on the environmental behaviour of radon and of its dosimetry is provided in chapter II. The second most important pathway is external irradiation, accounting for nearly 800 μSv, divided approximately equally between cosmic radiation and terrestrial sources. Less significant are the ingestion of ^{40}K (180 μSv), inhalation of decay products of thoron (160 μSv) and internal irradiation from ^{210}Pb-^{210}Po (120 μSv). The other natural radionuclides contribute little to the total annual effective dose equivalent, which is estimated to be 2,400 μSv.

82. The variability around the mean dose from natural sources of radiation is dominated by the radon component, as the indoor radon concentrations span over four orders of magnitude.

II. RADON-222 AND ITS SHORT-LIVED DECAY PRODUCTS

83. In the UNSCEAR 1982 Report, it was estimated that inhalation of short-lived decay products of radon (^{222}Rn) accounts on average for about one half of the effective dose equivalent from all natural sources of radiation and may sometimes lead to doses high enough to cause concern for human health. The awareness of the potential health problems that could be caused by radon and other pollutants in the indoor environment has been growing steadily in the past few years so that a large number of scientific papers, books, reports and meetings have been devoted to questions related to radon exposures, especially indoors (see, for example [A17], [C24], [H27], [N24] and [N25]).

84. Following a brief overview of the outdoor situation, this chapter focuses mainly on indoor concentrations. The exposure-dose relationships for the outdoor and indoor conditions are then established, followed by an assessment of the annual doses.

A. OUTDOOR CONCENTRATIONS

85. Radon enters the atmosphere mainly by crossing the soil-air interface, but there are a number of other secondary sources, such as the ocean, ground water, natural gas, geothermal fluids and coal combustion. The atmospheric concentrations of radon at ground level are governed by the source term—the exhalation rate—and by atmospheric dilution processes, which are both affected by meteorological conditions. The degree of radioactive equilibrium between radon and its decay products in the atmosphere at ground level also depends to a large extent upon the meteorological conditions.

86. When ^{226}Ra decays in soil particles, the resulting atoms of ^{222}Rn must first escape from the soil particles to air-filled pores and move through these pores in order to enter the atmosphere. The escape from the soil particles to the air-filled pores is thought to be mainly the result of recoil of the radon atoms following the decay of ^{226}Ra [M1]; if they lie close to the surface of individual grains, they may be ejected into the pores between the grains. In comparison, the contribution from diffusion through the solid mineral grains is less important, as most of the radon atoms decay before escaping. The fraction of radon formed in the soil that escapes into the pores is known as the emanating power, coefficient, ratio or fraction; reported values range from about 1% to 80%.

87. In order to enter the free atmosphere, the radon gas must diffuse through the pores of the material, and a fraction of it will reach the surface before decaying. The diffusion path is tortuous and, of course, some radon atoms will be ejected into closed pores from which they cannot escape. Movement of radon atoms may be caused by diffusion or convection. Convective movements, induced by pressure differences created by meteorological conditions, vary

with time and cannot be readily quantified. The diffusion process is described mathematically by the definition of an effective diffusion coefficient that includes an allowance for the convoluted path. As in the UNSCEAR 1982 Report (Annex D, paragraph 58), the area exhalation rate, defined as the activity transfer rate per unit area at the soil-air interface, is expressed as

$$R = \lambda_{Rn} F_r C_{soil,Ra} \rho_{soil} L_{Rn} \qquad (2.1)$$

where R is the area exhalation rate in Bq m^{-2} s^{-1}; λ_{Rn} is the decay constant of ^{222}Rn (2.1 10^{-6} s^{-1}); F_r is the emanating power; $C_{soil,Ra}$ is the activity mass concentration of ^{226}Ra in soil (Bq kg^{-1}); ρ_{soil} is the soil density (kg m^{-3}); and L_{Rn} is the diffusion length of radon in soil (m), which can be expressed mathematically as the square root of $\Delta_{eff}/\lambda_{Rn}\rho_{soil,ps}$, where Δ_{eff} is the effective bulk diffusion coefficient (m^2 s^{-1}) and $\rho_{soil,ps}$ is the soil porosity.

88. Published information on experimental values of R, F_r, Δ_{eff} and $\rho_{soil,ps}$ is summarized in Table 13. Using the representative values given in Table 13 for F_r, Δ_{eff} and $\rho_{soil,ps}$ and assuming a ^{226}Ra activity mass concentration in soil of 25 Bq kg^{-1} (Table 3) and a soil density of 1.6 10^3 kg m^{-3}, the diffusion length of radon L_{Rn} is approximately 1 m and the area exhalation rate is 1.7 10^{-2} Bq m^{-2} s^{-1}, in agreement with the estimated area-weighted average per unit area for soil, based on direct measurements, of 1.6 10^{-2} Bq m^{-2} s^{-1} [W1]. Over the ocean, the area exhalation rate is about two orders of magnitude lower than that for soil.

89. The concentrations of radon in air vary depending on the place, time, height above the ground and meteorological conditions. Because the source of radon is the soil and radon has a rather short physical half-life, the radon concentration is in general constantly decreasing with height. The geographical location is important: radon concentrations are as a rule lower over locations, such as islands and Arctic areas, which have less soil capable of emanating radon than over continental temperate regions.

90. Detailed information on the variations of radon concentration at ground level over time was obtained from a six-year record of hourly radon measurements made at Chester, New Jersey, United States [F7]. The arithmetic mean for the six years of operation was found to be 8 Bq m^{-3} and the hourly data and three-hour averages of radon concentrations were log-normally distributed. A seasonal pattern of a summer maximum and a winter minimum was observed over the period 1977-1982 with little variation from year to year [F7]. The seasonal maximum in August was a factor of 3 higher than the February minimum (Figure VII). The diurnal variation (Figure VIII) shows a maximum in the night and a minimum at noon. The diurnal maximum is a factor of 2 greater than the minimum. The three-hour average radon concentrations were tested for correlation with five meteorological parameters measured at the site—temperature, dew-point temperature, wind speed, atmospheric pressure and precipitation—but no significant correlation was found [F2].

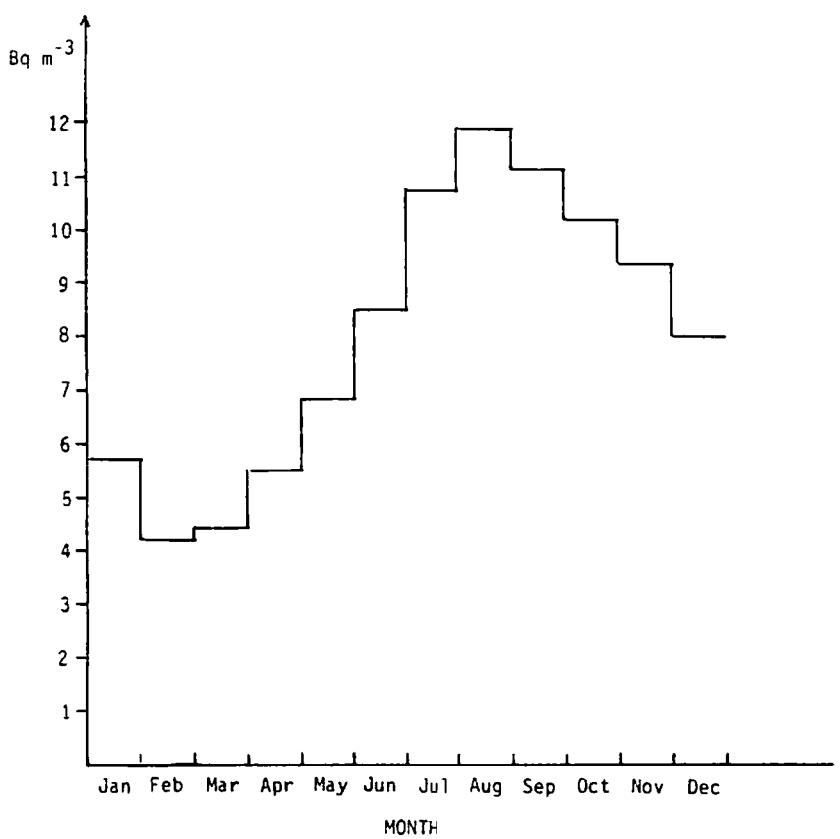

Figure VII. Seasonal variation of outdoor radon concentration at Chester, New Jersey (1977-1982 average).
[F1]

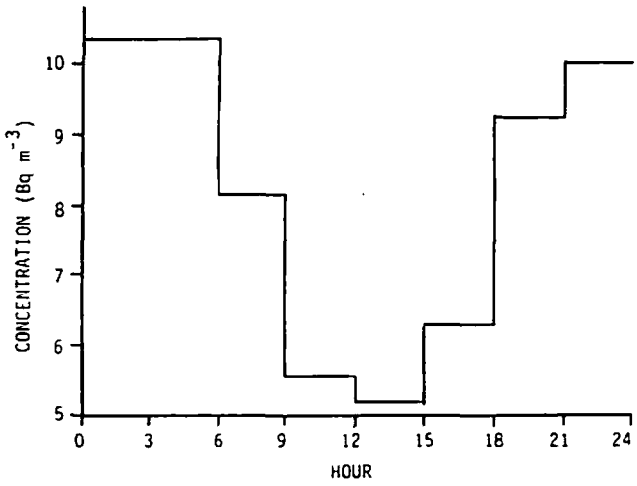

Figure VIII. Diurnal variation of outdoor radon concentration at Chester, New Jersey (1977-1983 average of three-hour data). [F7]

91. The compilation obtained at Chester represents the most complete available set of data on outdoor radon concentrations at ground level. Less extensive series of measurements were reviewed in the UNSCEAR 1982 Report and by Gesell [G1]. With regard to the seasonal variations, other data from the United States showed lower values in the spring than in summer and autumn [W2, C1, G3, L2]. Data from Hungary [G9] are in agreement with those obtained at Chester. Other seasonal patterns, however, were observed at Livermore, California [L1], Bombay, India [M2], Rio de Janeiro, Brazil [L2], and Japan [I8, M3, S47]; this may be due to the fact that those locations, being islands or coastal cities, are more affected by the annual wind rose.

92. The average value of outdoor atmospheric radon concentration for normal areas of the United States was estimated by Gesell [G1] to be about 9 Bq m^{-3} after the diurnal radon cycle was taken into account. This is probably representative of the continental areas in the temperate latitudes. Values lower by a factor of 2-4 are generally observed on islands and at coastal sites [C4, F3, I8, S47, W14]. In this Annex it is tentatively estimated that the annual average of the population-weighted radon concentration in outdoor air is 5 Bq m^{-3}. A lower value, 3 Bq m^{-3}, was adopted in the UNSCEAR 1982 Report.

93. The equilibrium factor F between radon and its short-lived decay products is defined as the ratio of the equilibrium equivalent radon concentration to the radon activity concentration:

$$F = (\chi_{eq,Rn})/\chi_{Rn} \qquad (2.2)$$

$$\text{with } \chi_{eq,Rn} = 0.105\,\chi_1 + 0.516\,\chi_2 + 0.379\,\chi_3$$

where χ_1, χ_2 and χ_3 represent the activity concentrations of polonium-218, lead-214 and polonium-214, respectively. A few studies were devoted to the assessment of the equilibrium factor F between radon and its short-lived decay products. At Chester, New Jersey, George [G2] found an average value of 0.85 at 1 m above ground. In the Federal Republic of Germany, Jacobi obtained an equilibrium factor of 0.77 at 1-10 m above ground. Cox et al. [C1], at Cincinnati, Ohio, reported an average value of 0.87, and measurements in New Jersey and New York at 0.3-1 m above ground gave an equilibrium factor of 0.79. It seems that 0.8 may be a representative value of the average equilibrium factor F at 1 m above ground. This is higher than the value of 0.6 assumed in the UNSCEAR 1982 Report. Combining the annual average of the population-weighted radon concentration in outdoor air of 5 Bq m^{-3} with an average equilibrium factor of 0.8 yields an average equilibrium equivalent concentration of radon of 4 Bq m^{-3}. Annual averages of this value vary depending on the location; a typical range, excluding extreme values, is from 1 to 10 Bq m^{-3}.

B. INDOOR CONCENTRATIONS

94. There is no basic difference in the physical behaviour of radon and its decay products in a room and in the open air; however, the different nature of some of the sources and the magnitude of the indoor exposures warrant a separate discussion. In this section, the various sources of indoor radon are first discussed and quantified in order to estimate an average and a typical range of the rate of entry of radon into a hypothetical reference house. The principal mechanisms of transport and removal of radon and radon decay products are then considered in order to estimate average radon and radon decay product concentrations in the hypothetical reference house. In the last part of this section, the results of large-scale indoor surveys of radon or radon decay products are reviewed.

1. Sources of indoor radon

95. Radon enters buildings from different sources, such as the soil or rock under or surrounding the buildings, building materials, water supplies, natural gas and outdoor air. Characterizing the radon entry rate may require consideration of the rate at which radon is generated in source materials, of the modes of radon transport through various materials and, finally, of the manner in which radon actually enters indoor air. Several reviews of indoor radon sources have recently been prepared [N1, B25, G24, N13].

96. As in the UNSCEAR 1982 Report, reference will be made to a hypothetical reference house; its dimensions and relevant parameters are given in Table 14. In the UNSCEAR 1982 Report, the radon source in the reference house was estimated to be 74 kBq d^{-1}, corresponding to a radon entry rate of about 15 Bq m^{-3} h^{-1}. Figure IX presents the cumulative frequency distributions of radon entry rates determined in dwellings in several countries as the product of the simultaneously measured ventilation rate and radon concentration [N8]. It appears that the radon entry rates are approximately log-normally distributed and that there is in most countries a considerable spread between the minimum and the maximum values. The median radon entry rates presented in Figure IX range from 5 Bq m^{-3} h^{-1} in the Federal Republic of Germany

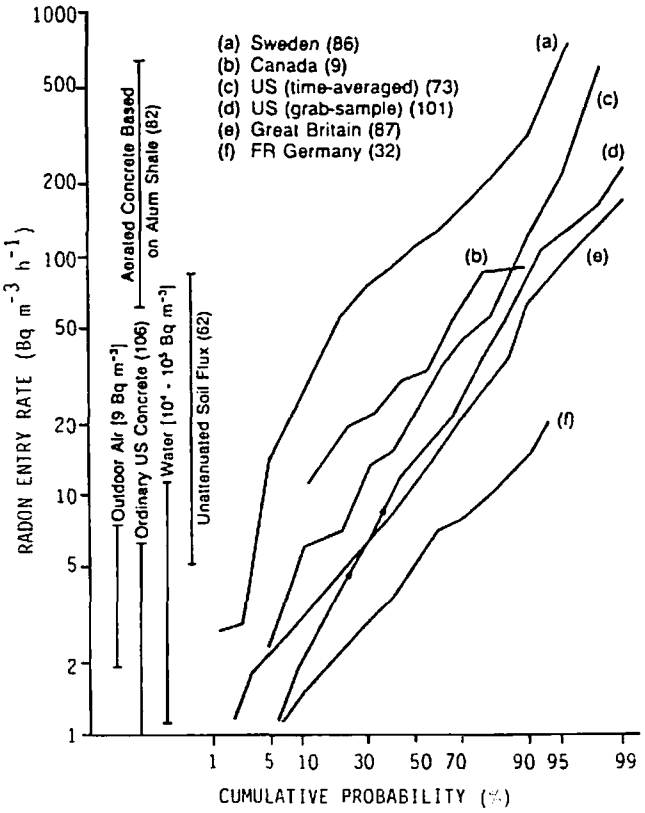

Figure IX. Cumulative frequency distributions of radon entry rate in buildings in several countries determined from the product of simultaneously measured ventilation rate and radon concentration. (The number of residences in each sample is indicated in parentheses.)
[N8]

[W6] to 102 Bq m^{-3} h^{-1} in Sweden [H16]: the corresponding arithmetic means, assuming log-normal distributions, are 7 and 190 Bq m^{-3} h^{-1}, respectively. Intermediate values have been obtained in Canada [S41], the United Kingdom [C4] and the United States [N8]. Figure X shows the histogram of the radon entry rates determined from the British survey [C4]. It should be pointed out that the number of houses considered was relatively small in each of the surveys. The various sources of indoor radon are considered next.

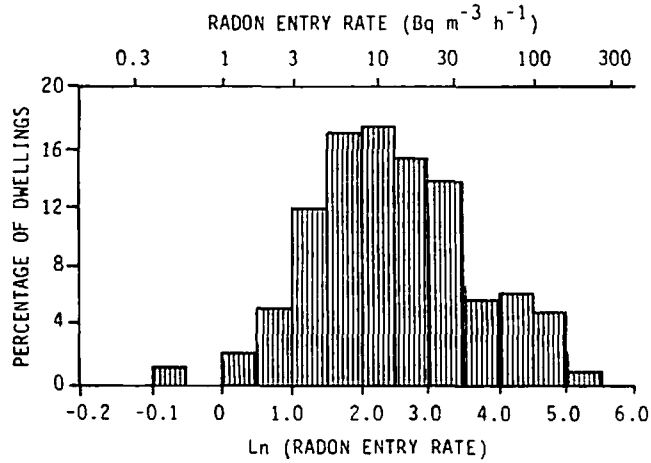

Figure X. Distribution of radon entry rates in rooms from a survey in the United Kingdom.
[C4]

(a) Underlying soil

97. Radon can be transported into a building from the underlying soil via diffusion or via the pressure-driven flow of air through the structural elements or through openings in the structural elements.

98. *Diffusion.* For an uncracked slab of concrete of thickness L lying on the ground, the area exhalation rate transmitted by diffusion from the underlying soil through the slab can be expressed as [N1, C3]

$$R_T = R/[\cosh(\frac{L_c}{L_{Rn,slab}}) + \frac{F_{soil,ps}\, L_{Rn,soil}}{F_{slab,ps}\, L_{Rn,slab}} \sinh(\frac{L_c}{L_{Rn,slab}})] \qquad (2.3)$$

where R is the area exhalation rate from uncovered soil (equation 2.1). Other formulations can be found in the literature on the subject (see, for example, [B26]). Taking the values already given in preceding paragraphs for the various parameters yields

$$R_T = 1.7\ 10^{-2}\ \text{Bq m}^{-2}\,\text{s}^{-1} / [\cosh(\frac{0.2}{0.15}) + \frac{0.15 \times 1}{0.2 \times 0.15} \sinh(\frac{0.2}{0.15})] = 1.2\ 10^{-3}\ \text{Bq m}^{-2}\,\text{s}^{-1} \qquad (2.4)$$

Thus, only a small percentage of the activity exhaled from the soil is transmitted through 0.2 m of uncracked concrete, and this exhaled activity represents about one half of that due to the concrete itself. The corresponding radon entry rate in the reference house can be estimated as

$$U_T = R_T N S_F / V \qquad (2.5)$$

or, in numerical terms,

$$1.2\ 10^{-3}\ \text{Bq m}^{-2}\,\text{s}^{-1} \times 3600\ \text{s h}^{-1} \times 100\ \text{m}^2/250\ \text{m}^3 = 1.7\ \text{Bq m}^{-3}\,\text{h}^{-1}$$

99. The presence of cracks in the slab may considerably increase the transmission of the diffusive flux from the soil. Using a mathematical model of a cracked slab, Landman [L3] determined that 25% of the flux from uncovered soil would penetrate the slab by diffusion if a gap of 1 cm existed for every metre of slab.

100. *Pressure-driven flow.* The pressure-driven flow of radon-bearing air through structural elements is believed to be an important mechanism for radon entry [N1]; it is often the predominant source of radon in dwellings with elevated concentrations (see, for example, [A10], [E7], [N8], [W13] and [W8]), especially if the house is in direct contact with the ground [S65]. Flow through an intact concrete slab is likely to be negligible in comparison to flow through cracks, holes and other penetrations. In dry soil with a density of 1.6 10^3 kg m^{-3}, porosity of 20%, ^{226}Ra activity mass concentration of 25 Bq kg^{-1} and emanating power of 20%, the calculated equilibrium radon concentration in soil gas is 40 kBq m^{-3}. If it is assumed that 0.25 m^3 of soil gas enters a building per hour, representing about 0.1% of the total air exchange rate, the radon entry rate from that source is 40 Bq m^{-3} h^{-1}.

101. Much attention has been given to the question of radon entry through convective flow in countries

such as Sweden [S7, S8], the United States [N1], Canada [E1] and the United Kingdom [O2], where a number of dwellings have been found to have unusually high radon concentrations. The radon entry rate through convective flow R_c is simply the product of the radon concentration in soil gas χ_s and the infiltration rate from the soil Q_s. The radon concentration in soil gas depends on the activity mass concentration of radium in soil, its emanating power and the porosity, permeability and moisture content of the soil. Investigations of radon concentrations in soil gas in Sweden, the results of which are presented in Table 15, show that there is usually no long-distance transport of radon in the soil and that the radon concentrations in soil gas can be explained assuming an emanating power of 10-30% [A10].

102. The infiltration rate from the soil Q_s depends on the degree to which indoor air is coupled to the soil, which, in turn, depends on the design and construction of the building structure, on meteorological parameters influencing radon movements in the soil and on the living habits of the occupants, which can affect the air exchange rate in the building. Mechanisms of radon transport from soil into a house having a vented crawl space are illustrated in Figure XI [N10]. The two pressure-driven flows considered are those due to the indoor-outdoor temperature difference, including a stack effect in winter, and wind.

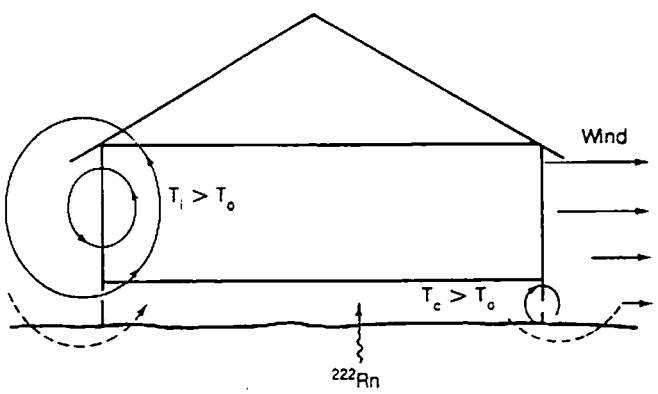

Figure XI. Schematic diagram of mechanisms related to radon transport from soil, through a vented crawl space and into a house.
[N10]

103. Experimental investigations in a house with a vented crawl space showed that a higher temperature difference, including an increasing infiltration rate, corresponded to a higher indoor radon concentration, suggesting that the increased infiltration rate is more than compensated for by an increase in the radon entry rate [N10]; they also suggested that the infiltration rate from the soil into the house was, in this case, more important than the infiltration rate from the walls. A high wind speed tended to reduce the indoor radon concentration, presumably by increasing the cross-ventilation of the crawl space and the infiltration rate of the living space. For this particular house, as well as for two other houses with vented crawl spaces that were investigated in the study, it was found that perhaps 50% or more of the radon released into the crawl space from the soil beneath the house entered the living space [N10]. This figure cannot, however, be applied to all types of construction: the floor of a crawl-space house, which is usually built of wood, is likely to have greater infiltration rates than the floor and walls of a basement, which are usually built of poured concrete and concrete blocks.

104. Another extensive investigation of radon entry through the soil was conducted in a single-family house with a basement; real-time measurements of the indoor radon concentration, air exchange rate, source-related parameters and meteorological factors were performed for a period of five months [N4]. The radon entry rate was calculated from the measured radon concentration and air exchange rate. Figure XII shows the results obtained during a week of that experiment with regard to the radon entry rate and the radon concentrations in the soil adjacent to the house and in the basement sump, which collects rainwater via a drain tile system surrounding the house at a level near that of the basement floor. The good correlation between the sump activity and the radon entry rate suggests that the sump is an important pathway for radon entry into the house, although it cannot account for the total radon activity [N4]. The main conclusion derived from this study is that the radon entry rate may have two components: one may be independent of the air exchange rate, acting in a way similar to entry by diffusion; the other may be proportional to the air exchange rate, behaving like pressure-driven entry [N4]. The corresponding radon entry rates in the house were calculated to be about 2 Bq m^{-3} h^{-1} for the diffusion process and about 60 Bq m^{-3} h^{-1} for the pressure-driven force [N1].

105. Computer modelling of radon movement of soil gas resulting from pressure differences between the underlying soil and indoor air has been carried out in Canada [E7, S9]. In a wind-tunnel study, variations in wind speed and direction, and not the wind speed or direction itself, were found to cause large variations in the radon entry rate. The average radon entry rate was found to be mainly a function of the soil permeability and only secondarily a function of the area and resistance of the house used in the computer model to soil connections. The effect of seasonal differences on radon entry rate was approximately 4 to 1 from winter to summer. Stack-effect pressure differences due to temperature differences increased by a similar factor over this period. Because the ventilation rate of the house depends largely on the stack effect, these results suggest that summer and winter concentrations should be similar unless doors and windows are opened in summer to increase the ventilation rate. For the same geometry and weather, decreasing the soil permeability from 10^{-4} cm^2 to 10^{-7} cm^2 (a factor of 1,000) decreased the average radon entry rate into the house by a factor of about 25. This suggests that the radon entry rate is roughly proportional to the square root of soil permeability [D12]. Considerably more theoretical and experimental work is likely to be required to determine entry routes and transport mechanisms from underlying soil with some degree of confidence.

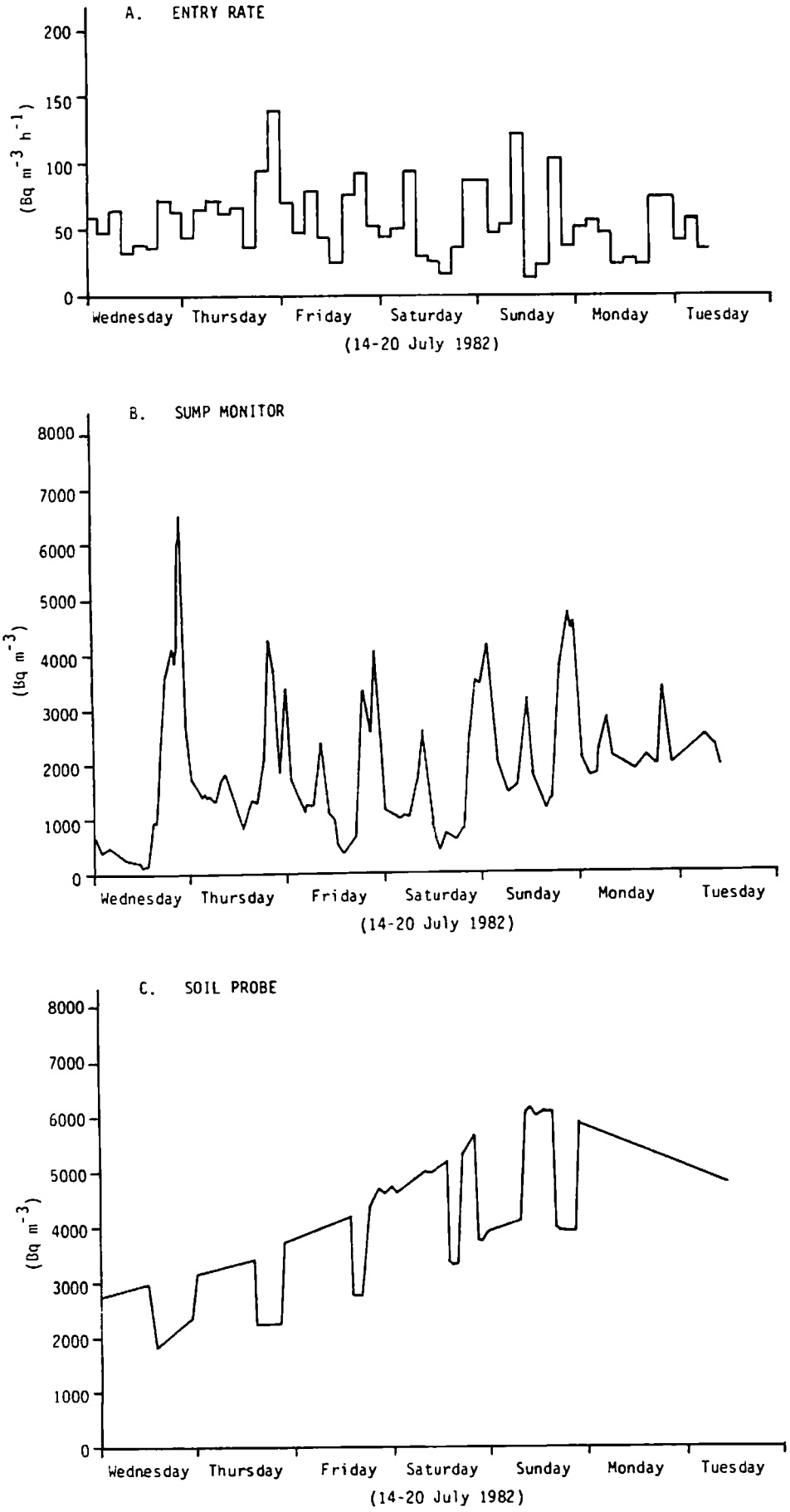

Figure XII. Variation with time in a single-family house with a basement of (a) radon entry rate; (b) the radon concentration in the basement sump; and (c) in the soil adjacent to the house.
[N4]

(b) Building materials

106. A fraction of the radon activity produced by decay of ^{226}Ra in building materials enters buildings by diffusion. The area exhalation rate can be expressed as

$$R = \lambda_{Rn}\rho_{build}C_{build.Ra}F_rL_{Rn}\tanh(L_h/L_{Rn}) \quad (2.6)$$

which is an equation very similar to that related to soil (equation 2.1), the only difference being the introduction of a hyperbolic term to account for the fact that diffusion takes place in a medium of finite thickness. In equation (2.6), R is the area exhalation rate (Bq m^{-2} s^{-1}); F_r is the emanating power; ρ_{build} is the density of the building material (kg m^{-3}); $C_{build.Ra}$ is the activity mass concentration of ^{226}Ra in the building material (Bq kg^{-1}); L_{Rn} is the diffusion length (m); and L_h is the half-thickness of a slab of building material (m).

107. The mass activity exhalation rate, expressed in Bq kg^{-1} s^{-1} and defined as

$$R_m = \lambda_{Rn}C_{build.Ra}F_r \quad (2.7)$$

is the quantity usually determined in laboratory measurements [J1]. The area exhalation rate from a wall or a floor made of building material of half-thickness L_h, diffusion length L_{Rn} and density ρ_{build} can then be expressed as

$$R = R_m\rho_{build}L_{Rn}\tanh(L_h/L_{Rn}) \quad (2.8)$$

108. Information on the mass exhalation rate, the diffusion length, the emanating power and the ^{226}Ra activity mass concentration in building materials is presented in Table 16. Concrete and brick are probably the two most widely used building materials, at least in the temperate latitudes. Table 16 shows that even though the ^{226}Ra concentrations in concrete seem to be lower than those in brick, the ^{222}Rn mass exhalation rates appear to be higher for concrete than for brick. This is because of the high values of the emanating power for concrete, with or without fly ash, in comparison to those for brick. The results presented for gypsum (Table 16) are difficult to analyse because the few measurements available give either high or low values of the emanating power. Further measurements are necessary to clarify this point.

109. Some data on the emanating power of the concrete components are given in Table 16. Cement and fly ash have low emanating powers, probably due to their crystalline nature. Differences in the moisture content of the materials studied may also account in part for the wide disparity in the results of exhalation measurements from different countries [N1]. It is worth noting that the exhalation rate from concrete should not be derived from the emanating power of its constituents, as chemical changes occur during manufacturing.

110. The adopted reference values for the parameters of interest in the calculation of the area exhalation rate from concrete and brick are indicated in Table 16. For building material with a thickness of 0.2 m, the reference area exhalation rates would be 2 mBq m^{-2} s^{-1} and 0.3 mBq m^{-2} s^{-1} for concrete and for brick, respectively. The rate of entry of radon resulting from exhalation from building materials may be expressed as

$$U_{bm} = N/V\,(2R_cS_F + R_bS_w) \quad (2.9)$$

where N is the number of seconds per hour; V is the volume of the reference house (250 m^3); S_F is the surface area of its floor or ceiling (100 m^2); S_w is the surface area of its external walls (100 m^2); and R_c and R_b are the area exhalation rates from concrete and brick, respectively.

111. The rate of entry of radon from the floor and the ceiling of the reference house would amount to about 6 Bq m^{-3} h^{-1}, while the contribution from the external walls would be about 0.4 Bq m^{-3} h^{-1}. The contribution from radon exhalation from the building materials to the radon concentration in the reference house is thus estimated to be 6.4 Bq m^{-3} if the air exchange rate is taken to be 1 h^{-1}.

112. Considerably greater values of the rate of entry of radon are expected to be obtained when building materials with high ^{226}Ra concentrations and normal emanating power are extensively used. Examples of such building materials are granite, Italian tuff and alum-shale lightweight concrete. Among these materials, Swedish alum-shale lightweight concrete has the highest ^{226}Ra concentrations (about 1,300 Bq kg^{-1} on average) [S19, S20] and probably the highest ^{222}Rn mass exhalation rate (440 μBq kg^{-1} s^{-1} in Table 16). Assuming a density of $2\,10^3$ kg m^{-3} and a diffusion length of $7.4\,10^{-2}$ m (as measured in [J2]), a 0.2 m thick slab of 100 m^2 would lead to a rate of entry of radon of about 80 Bq m^{-3} h^{-1} in the reference house.

113. Techniques for reducing the radon entry rate due to exhalation from building materials have been investigated. In Sweden, aluminium foil has been applied to the walls of houses built with aerated concrete based on alum shale [S19]; the results showed a 50% reduction in the radon entry rate. In the United States, various radon sealants have been tested under conditions representative of normal construction conditions [E2]. The radon exhalation rate was reduced from 20% to 80%, depending on the surface coating used. Because of the trapping of radon decay products in the wall, there is a relatively small increase in the external gamma dose rate [E2, M8]. It was also noted that any cracks that later developed in a sealant such as paint may lead to leaks that negate a large portion of the sealing effectiveness of the paint [M19]. Similarly, the radon exhalation rate from any unpainted areas may be increased as they offer to radon a path of least resistance in comparison with painted areas [A11].

(c) Outdoor air

114. Air exchange between the outdoor and the indoor environments brings some outdoor radon into buildings. Air exchange arises from natural ventilation through open doors and windows, mechanical ventilation and infiltration, the uncontrolled leakage of air through cracks in the building envelope. Unless otherwise stated, air exchange in this Annex is

assumed to consist of infiltration only. Outdoor radon can play a significant role in the indoor radon entry rate if the house is poorly sealed. The radon entry rate resulting from the infiltration of outdoor air may be written as

$$\dot{U}_{out} = \chi_{out} a \lambda_v \qquad (2.10)$$

where χ_{out} is the radon concentration in outdoor air (Bq m^{-3}); λ_v is the air exchange rate (h^{-1}); and a is the fraction of the air exchange rate involving outdoor air, which in all cases is close to 1. Assuming an outdoor concentration of 5 Bq m^{-3} (see paragraph 92), an air exchange rate of 1 h^{-1} and a value of 1 for a, the radon entry rate would be 5 Bq h^{-1} m^{-3}. It is worth noting that the radon entry rate resulting from the infiltration of outdoor air is proportional to the air exchange rate.

(d) Water

115. Radon contained in water is to some extent transferred into room air as a result of agitation or heating. Radon concentrations are as a rule much lower in surface water than in ground water. In water-saturated soil with a density of 1.6 10^3 kg m^{-3}, porosity of 20% and emanating power of 20%, a ^{226}Ra activity mass concentration of 25 Bq kg^{-1} yields at equilibrium a ^{222}Rn concentration in ground water of

25 Bq kg^{-1} × 1.6 10^3 kg m^{-3} × 0.2/0.2 = 4 10^4 Bq m^{-3}

In surface water, radon concentrations are expected to be similar to those of ^{226}Ra, that is, about 10 Bq m^{-3}.

116. The release of radon from water to air depends upon the circumstances in which the water is used, as the degassed fraction increases considerably with temperature [G5]. Studies of radon transfer from tap-water to indoor air reported a use-weighted transfer efficiency of 0.5-0.6 and an average water use of 0.2-0.4 m^3 per day and per person [G5, P2]. Degassing of radon from tap-water has been found to lead to elevated indoor radon concentrations in Canada [M7], Finland [C2] and the United States [G6]. Taking the air-to-water concentration ratio to be typically 10^{-4} [C6], an indoor air concentration of 400 Bq m^{-3} is obtained if the radon concentration in water is 4 MBq m^{-3}. High concentrations in water are usually associated with deep wells drilled in granitic areas; the highest reported concentrations in water are 14 MBq m^{-3} in Canada [M7], 77 MBq m^{-3} in Finland [S68] and about 20 MBq m^{-3} in the United States [G6, S18]. The use of aeration can reduce the radon concentration in water by a factor of about 100 [C2].

117. Measurements of radon concentrations in water have mostly been undertaken in regions where high levels were suspected. Measurements intended to estimate the weighted average radon concentration in water for a country or a community are rare. Results of that nature are only available for Finland, Sweden and the United States. In Finland, the population-weighted average has been estimated to be 25 kBq m^{-3} for drinking water distributed by water-supply plants [A2, C2], and about 60 kBq m^{-3} when the contribution of private wells is included [K4]. The corresponding value in Sweden has been found to be 38 kBq m^{-3} [K24]. In the United States, the geometric mean radon concentrations in municipal ground-water supplies in the central part of the United States and in Texas were found to be about 4 kBq m^{-3} [P3, W3] and 5 kBq m^{-3} [W3], respectively. It was tentatively estimated in the UNSCEAR 1982 Report (Annex D, paragraph 163) that between 1% and 10% of the world's population consumes water containing radon concentrations of the order of 100 kBq m^{-3} or higher, drawn from relatively deep wells. For the remainder, who consume water from aquifers or surface sources, the weighted world average concentration is probably less than 1 kBq m^{-3}.

118. The radon entry rate due to water degassing may be expressed as

$$\dot{U}_w = \chi_w \dot{Q}_w \varepsilon / V \qquad (2.11)$$

where χ_w is the radon concentration in water (Bq m^{-3}); $\dot{Q}_w$ is the amount of water used per unit time (m^3 h^{-1}); ε is the degassing efficiency; and V is the volume of the reference house (m^3). Assuming a radon concentration of 1,000 Bq m^{-3} in tap-water, a degassing efficiency of 0.5, a water use rate of 0.07 m^3 h^{-1}, corresponding to 0.4 m^3 per day for each of the four persons living in the reference house, the radon entry rate is estimated to be about 0.1 Bq m^{-3} h^{-1}; the corresponding mean radon concentration is 0.1 Bq m^{-3} in the reference house, with a very uneven distribution within the dwelling, the highest concentrations being expected in the rooms where radon is released (bathrooms and kitchen). In any case, average radon concentrations in tap-water do not bring a significant contribution to the total radon entry rate in buildings, except, however, when the water contains high concentrations of radon. Figure XIII shows the variation of concentrations in indoor air for a house with a radon concentration in tap-water of about 2 MBq m^{-3} [G6]; the peaks are associated with periods of high use of water in the house. The integrated radon concentrations represented by each of the peaks in Figure XIII are 200-400 Bq h m^{-3}. Assuming that the characteristics of the surveyed house are the same as those of the reference house yields a water use of 0.05-0.1 m^3 per peak.

(e) Natural gas

119. Natural gas is sometimes mentioned as a potential significant source of indoor radon. The radon concentration in natural gas at production wells varies from undetectable values to levels of about 50 kBq m^{-3} [G7, H2, J3, W4]. The industrial processing of natural gas involves the removal of impurities and separation of hydrocarbons. Some of these hydrocarbons are bottled under pressure for sale as liquefied petroleum gas (LPG) while others may be used for fuel. Some of the radon activity contained in the processed natural gas decays during the transit time between processing and use, or while it is stored in the bottles. When natural gas is burned in houses for cooking or space heating the radon that is released may enhance the radon level indoor if the appliances are unvented. If the combustion products are vented outside the house, this radon source is negligible.

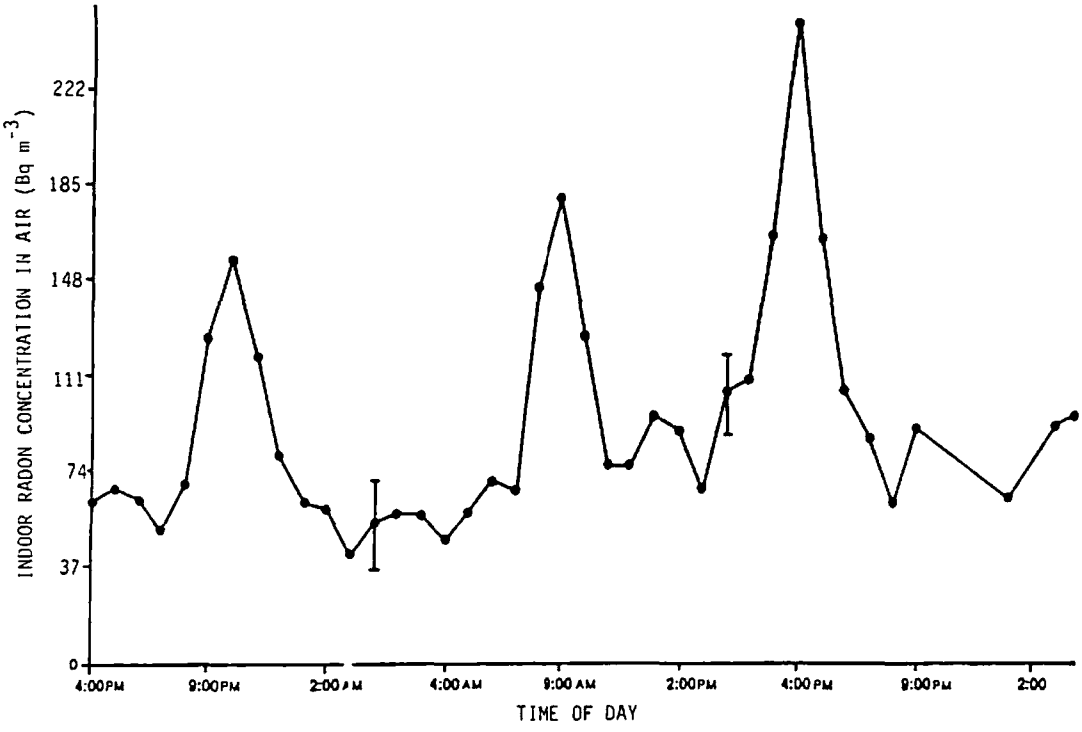

Figure XIII. Variation of the radon concentration in indoor air of a house with radon concentration in water of about 2 MBq m^{-3}.
[G6]

120. The radon entry rate from the use of natural gas may be expressed as

$$U_{ng} = \chi_{ng} \dot{Q}_{ng} / V \qquad (2.12)$$

where χ_{ng} is the radon concentration in natural gas (Bq m^{-3}); $\dot{Q}_{ng}$ is the amount of natural gas used per unit time (m^3 h^{-1}); and V is the volume of the reference house ([3]). Radon concentrations in natural gas and LPG have mainly been measured in the United States, where the average radon concentrations in either natural gas or LPG have been found to be about 1,000 Bq m^{-3} [G8, J3]. For a consumption rate of 2 m^3 d^{-1} in unvented appliances, the radon entry rate in the reference house is estimated to be about 0.3 Bq m^{-3} h^{-1}. The corresponding contribution to the radon concentration is 0.3 Bq m^{-3}.

(f) Summary

121. Table 17 summarizes information on the estimates of radon entry rates in the reference house. The estimated arithmetic mean value obtained is 50 Bq m^{-3} h^{-1}, in agreement with arithmetic means of experimental surveys [N8]. High values can be traced to a substantial flow of soil gas from the underlying soil, the use of alum shale as a building material or elevated radon concentrations in tap-water. It should be noted, however, that all other contributors remaining the same, the relative importance of the flow of soil gas is less in apartment buildings because the ratio of volume to ground-floor surface is much larger for them than for the reference house. Its relative importance is also less if dwellings are built in such a way that the contribution of the radon entry rate from underlying soil to the total radon entry rate in living areas is very small, because either the infiltration rate from the soil is negligible or the ventilation system in the basement is separate from that of the living areas of the building.

2. Indoor behaviour of radon and radon daughters

122. Radon entering indoors accumulates as a function of the air exchange rate in the building. The activity of radon that will accumulate within a building is limited by its removal through ventilation, normally expressed as the air exchange rate (measured in air changes per hour). Radon decay product concentrations are similarly limited; in addition, they can also be subject to plate-out mechanisms, not directly connected with room ventilation rate, which will keep their concentration below that of radon.

(a) Radon

123. Assuming an instantaneous and homogeneous mixing of radon in the building, the variation of the indoor radon concentration can be described by

$$[d\chi_a(t)]/dt = \dot{U}(t) - \chi_a(t)\,(\lambda_{Rn} + \lambda_v) \qquad (2.13)$$

where $\chi_a(t)$ is the radon activity concentration at time t in Bq m^{-3}; $\dot{U}(t)$ is the radon entry rate at time t in Bq m^{-3} h^{-1}; λ_{Rn} is the decay constant of radon, 7.6 10^{-3} h^{-1}; and λ_v is the air exchange rate at time t in h^{-1}.

124. If the radon entry rate ($\dot{U}$) and the ventilation rate (λ_v) are constant with time, the radon concentration at equilibrium is given by

$$\chi_a = \dot{U}/(\lambda_{Rn} + \lambda_v) \qquad (2.14)$$

If the contributions from outdoor air and pressure-driven flows through the soil are small, the indoor radon concentration at equilibrium is, as a first approximation, inversely proportional to the air exchange rate. This is illustrated in Figure XIV, which presents data from an experiment in which a balanced mechanical system—that is, one with equal intake and exhaust flows—was used to vary the air exchange rate λ_v without affecting the pressure-driven flow from the soil [N5].

125. Equation (2.14) can only be used as a first approximation of the mean indoor radon concentration, as it is based on assumptions that are usually not strictly valid. They are as follows: (a) the radon concentration within the building is assumed to be homogeneous. There is in fact essentially universal agreement that the radon concentrations decrease as the storey numbers of a multi-storey building increase, especially between the basement, first and second floor [G1, G9, G10, S10]. In the upper storeys of high-rise buildings, the radon concentration does not always decrease as the storey number increases. The presence of direct routes, such as lift shafts and service ducts between the basement, or even the sub-basement level, and higher storeys can distort the radon concentration gradient; (b) the air exchange rate is assumed to be constant with time. Actually, it is subject to important variations, as it is a function of human activities (opening or closing doors or windows, turning ventilation systems on or off) and of meteorological conditions (wind speed and direction, temperature, pressure). Figure XV shows the measured variation of the air exchange rate in an occupied house over a few days, together with the wind speed observed at a weather station 20 km away from the house [N6]. The air exchange rate is shown to vary from 0.5 h^{-1} to more than 4 h^{-1} in a matter of a few hours; there is in that particular case a good correlation between the wind speed and the air exchange rate, as predicted by the model of Grimsrud et al. [G25]; (c) the radon entry rate is assumed to be constant with time. In fact, as shown in Figure XII, the entry rate may experience significant changes in the course of one day owing to variation with time of the different radon sources discussed in the previous section.

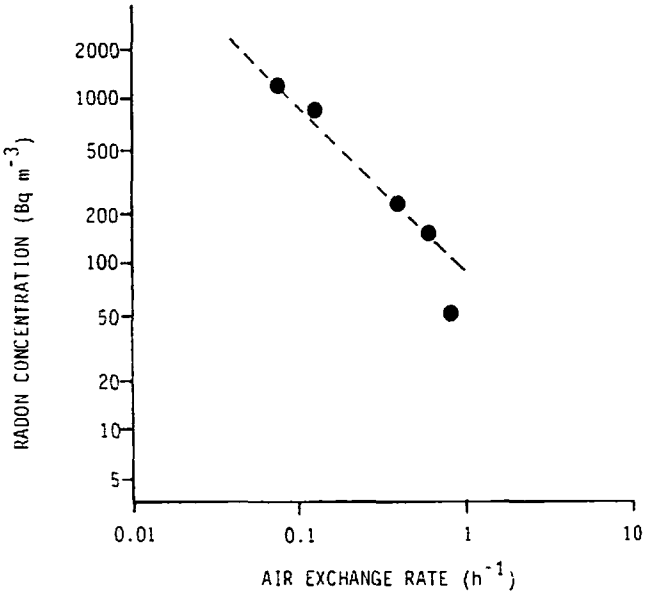

Figure XIV. Average steady-state radon concentration in a residence as a function of ventilation rate. (The dashed line indicates a constant radon source of 90 Bq m^{-3} h^{-1}).
[N5]

126. According to equation (2.14), there is a clear relationship between the radon concentration and the air exchange rate. However, simultaneous measurements of the air exchange rate and of the instantaneous radon concentration have been performed in three surveys involving 98 houses, including both energy-efficient and conventional houses. The surveys failed to reveal such a correlation between the indoor radon concentration and the air exchange rate, although each parameter varied over a wide range [N3]. Similar results were obtained from time-averaged measurements [D2] (Figure XVI).

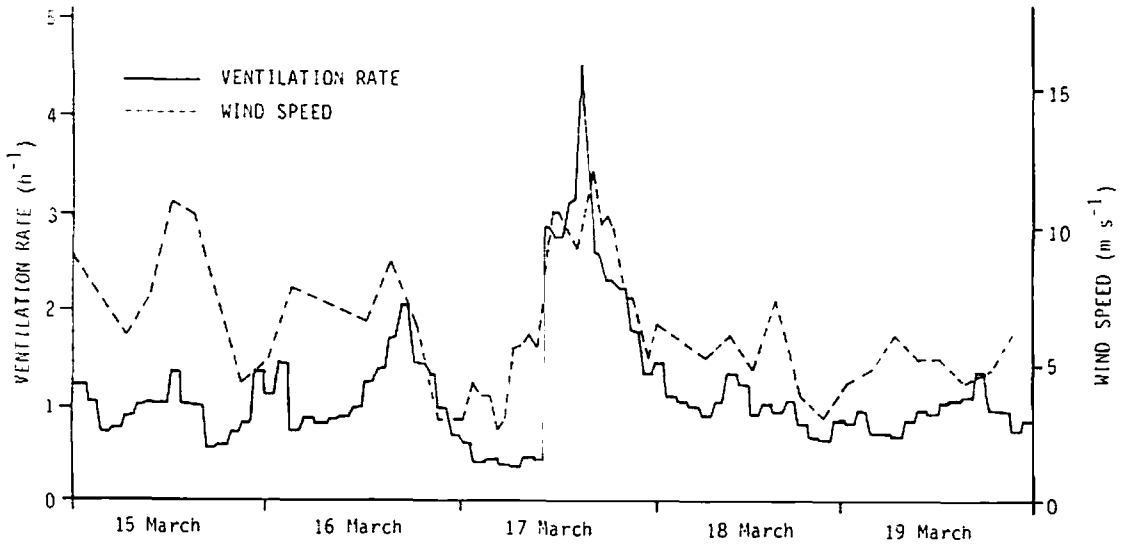

Figure XV. Variation of the ventilation rate in an occupied house. The wind-speed data represent observations at a weather station 20 km away from the house.
[N6]

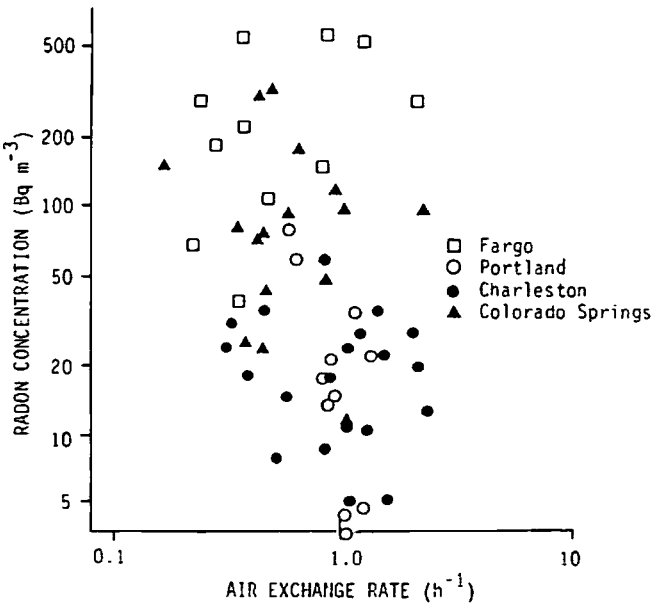

Figure XVI. Scatter plot of radon-222 concentrations versus air exchange rate for 58 houses in four cities. Measurements were made during a four- to five-month period between November 1981 and May 1982.
[D2]

127. Even though equation (2.14) is very simple and can be used to derive a typical value of the mean indoor radon concentration (for example, $\chi_a = 55$ Bq m^{-3} with the radon entry rates given in Table 17 and $\lambda_v = 1$ h^{-1}), it is clear that the prediction of the actual radon concentration (and of its variation as a function of time) in a given building cannot at this stage be made with accuracy. Figure XVII shows that the variation of the indoor radon concentration over one week has no consistent pattern [G11]. Further theoretical and experimental work (as in [B26]) is required to derive the values of χ_a, U and λ_v from the measurements of environmental and technological parameters.

(b) Radon decay products

128. When an atom of ^{218}Po is formed through the decay of radon, it is a free ion. Molecules of water vapour or trace gases coalesce almost immediately around the ion, forming a molecular cluster of 2-20 nm in diameter (Figure XVIII). The ion and the cluster are usually referred to as free, uncombined or unattached decay products. Unattached ^{218}Po is highly mobile and, after 10-100 s, it attaches to an aerosol particle (normal size in the range of 50-500 nm: see Figure XIX); the other options offered to unattached ^{218}Po are to plate out on the indoor surfaces, to be transported outdoors with the outgoing air or to decay into unattached ^{214}Pb. Attached ^{218}Po is relatively immobile and its plate-out on indoor surfaces is sometimes ignored. The various processes are illustrated in Figure XIX. The attached and the unattached decay products are often distinguished by their environmental behaviour and deposition pattern in the respiratory system. In a series of air samples, the position and size of the peak related to the unattached fraction may vary for reasons not yet completely elucidated [K16].

129. Upon alpha decay of attached ^{218}Po, the decay product ^{214}Pb thus created may remain on the aerosol or indoor surface or become unattached as the result of its recoil energy. Subsequently, the behaviour of ^{214}Pb is similar to that of ^{218}Po. Upon the decay of attached ^{214}Pb, the ^{214}Bi created typically remains attached, since the recoil energy from beta decay is not sufficiently large to promote detachment. The indoor behaviour of the radon decay products is illustrated in Figure XX, where subscripts 1, 2 and 3 relate to decay products ^{218}Po, ^{214}Pb and ^{214}Bi, respectively, and superscripts fr, a and s designate the unattached state, the attached state and the presence on indoor surfaces [B1]. Dotted lines denote the processes that have been neglected in this treatment. This model of indoor behaviour of radon decay products was first proposed by Jacobi [J4] and has subsequently been extended and modified by a number

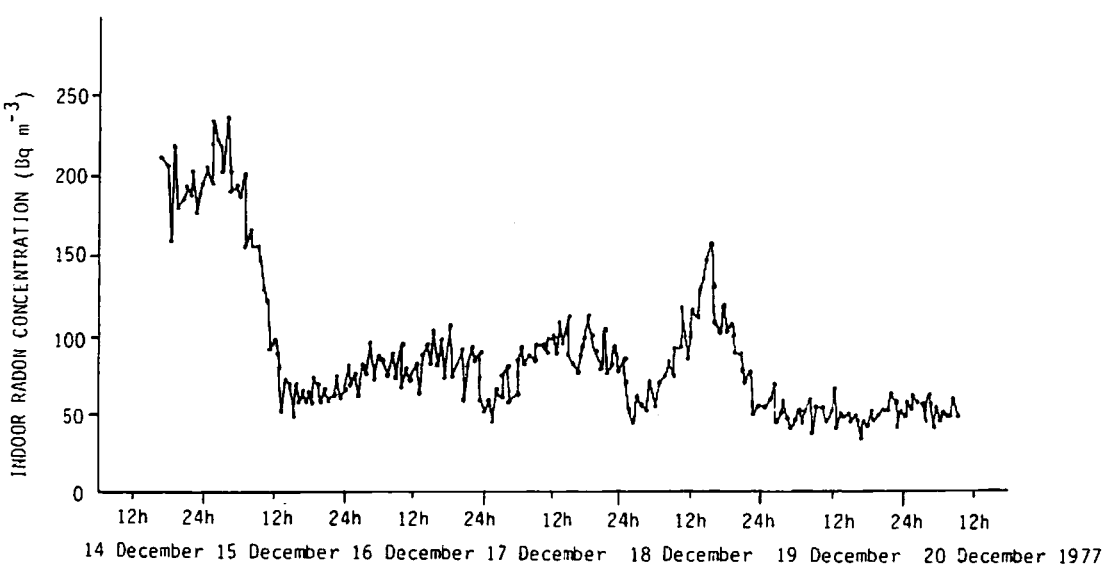

Figure XVII. Variation with time of indoor radon concentrations.
[G11]

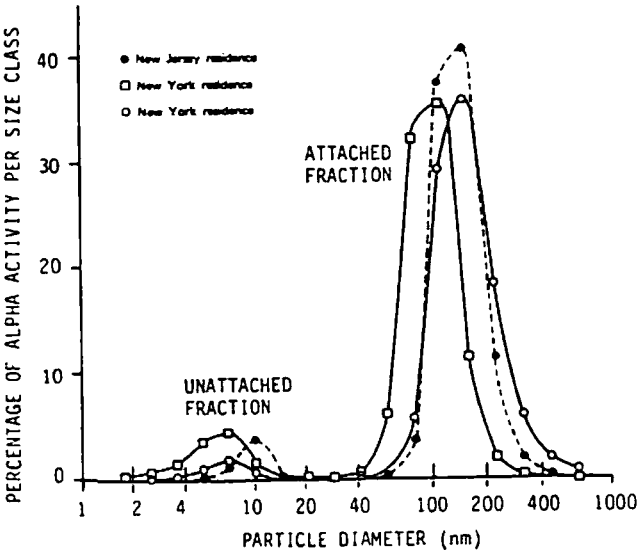

Figure XVIII. Histograms of size distribution of radon decay products.
[G11]

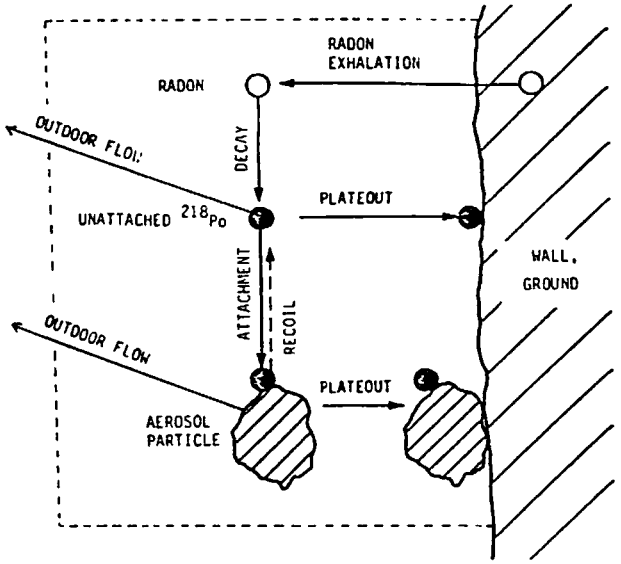

Figure XIX. The basic processes influencing the activity balance of radon decay products.
[P5]

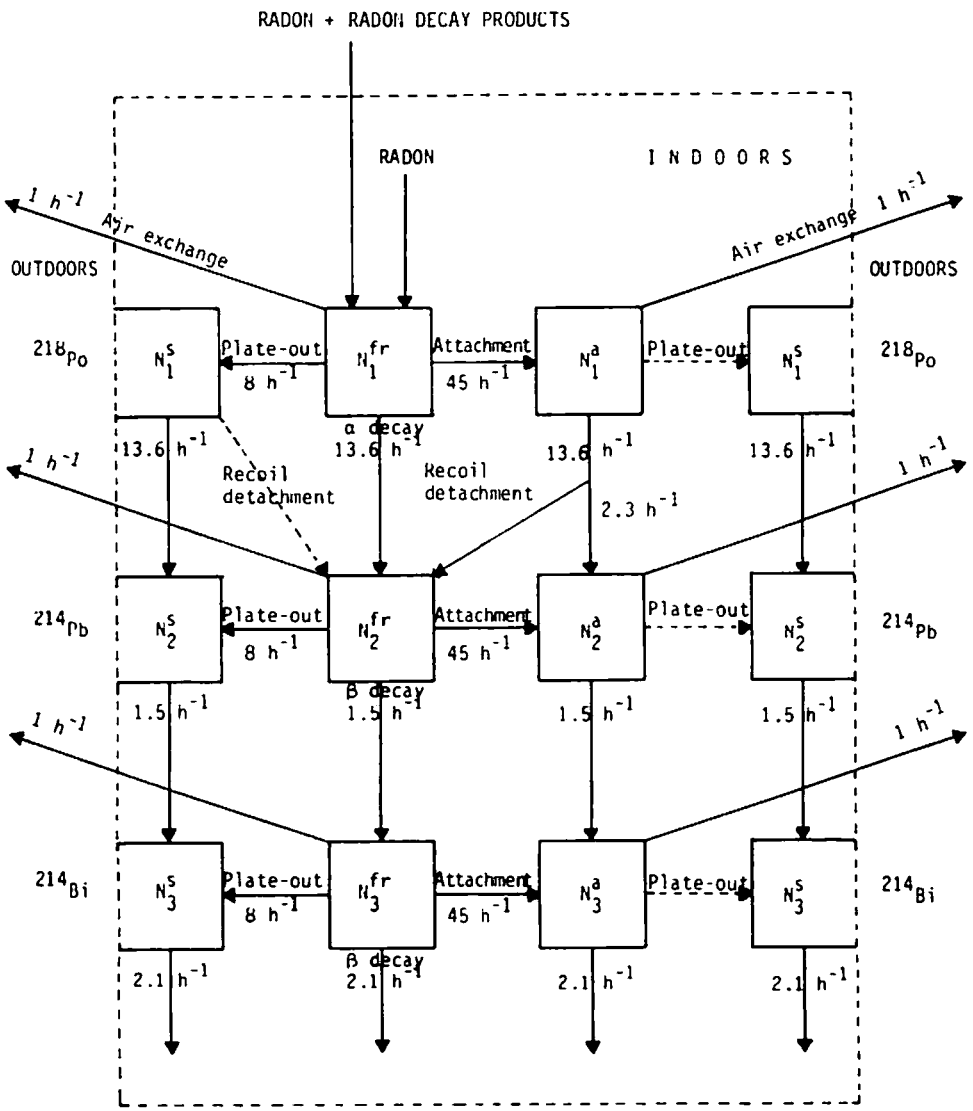

Figure XX. Flow chart of radon decay product behaviour under average conditions.
[B1]

of researchers, notably Porstendörfer et al. [P4, P5], Shimo et al. [S46, S48, S49] and Bruno [B1]. The plate-out rate constant of unattached radon decay products can be expressed as $\lambda_{po} = v_g S/V$, where v_g is the deposition *velocity* ($m\ h^{-1}$), S the total surface area in the room and V the volume of the room. The deposition velocity is about $2\ m\ h^{-1}$ for unattached daughters in still air [G12, K5, P5]. The surface to volume quotient S/V is estimated to range from 3 to $6\ m^{-1}$ in rooms of dwellings if the furniture is taken into account [P5]. The plate-out rate constant would therefore be about $10\ h^{-1}$ in still air. In ventilated rooms, the deposition velocity increases with the air speed so that the plate-out rate constant is expected to range from 10 to $30\ h^{-1}$ in dwellings. Values reported in the literature on the subject range from $2\ h^{-1}$ to $200\ h^{-1}$ [I7, I9, J4, K5, K17, P4, S11, S44, W5].

130. The deposition velocity for attached daughters is approximately two orders of magnitude less than that for unattached decay products, leading to plate-out rate constants of $0.1\text{-}0.3\ h^{-1}$ [K5, P5]. Consequently, plate-out of attached decay products is negligible as a removal mechanism relative to decay and ventilation.

131. The attachment rate X to aerosol particles has been shown by Porstendörfer and Mercer [P6] to be a linear function of the particle concentration

$$X = \beta Z \qquad (2.15)$$

where β is the attachment coefficient in $cm^3\ h^{-1}$ and Z is the particle concentration in cm^{-3}. Reported values of the attachment coefficient range from $1.8\ 10^{-3}\ cm^3\ h^{-1}$ to $7.4\ 10^{-2}\ cm^3\ h^{-1}$ [P5]. For indoor air, an average attachment coefficient of $5\ 10^{-3}\ cm^3\ h^{-1}$ may be assumed [P5]. The aerosol concentration indoor depends mainly on the outdoor particle concentration, the ventilation rate and the indoor sources of particles. Figure XXI presents the measured variation of aerosol concentrations indoor and outdoor for different ventilation rates [P5]. For high ventilation rates, the particle concentration ranges between 10^4 and $10^5\ cm^{-3}$ and is about the same indoors and outdoors. For low ventilation rates, the indoor particle concentration typically ranges from $2\ 10^3$ to $2\ 10^4\ cm^{-3}$ [P5]. Therefore the attachment rate X is expected to vary from $10\ h^{-1}$ to $500\ h^{-1}$, according to the indoor particle concentration. Average values between 20 and $50\ h^{-1}$ may be expected in closed rooms with low ventilation rates [P5].

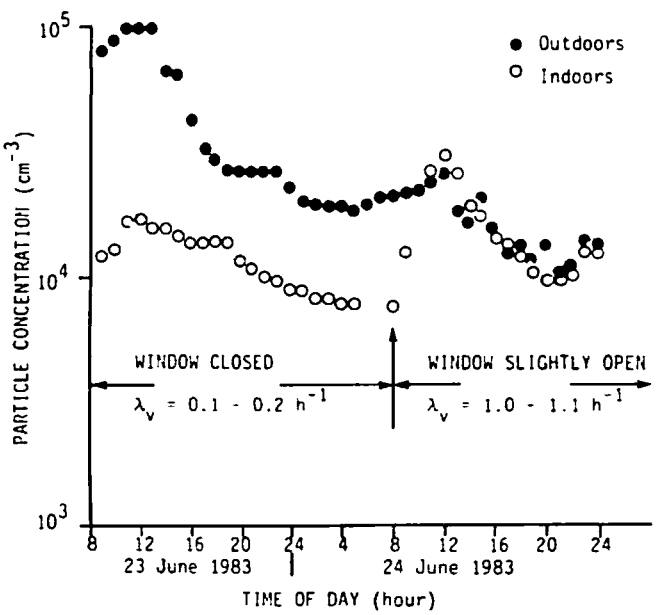

Figure XXI. Diurnal variation of aerosol concentrations outdoors and indoors of a building for different ventilation rates. [P5]

132. The probability for recoil detachment from aerosol particles upon alpha decay, also called recoil factor, has been studied from the theoretical and experimental point of view by Mercer [M4]. The recoil energy possessed by a daughter product after alpha decay is about 100 keV, corresponding to a range of about $0.1\ \mu m$ in solid material. For a typical aerosol of median diameter $0.2\ \mu m$, the recoil factor is about 0.8 [M4]. Upon beta decay, the recoil energy is only a few electron volts, yielding a recoil factor of 0.01-0.02 [P4]. The detachment from walls and other surfaces has not been examined in detail in any work to date; it would be expected that 25-50% of deposited activity could re-enter the room air mainstream upon alpha decay [B1, M1].

133. As already indicated, the equilibrium factor F is defined as the ratio of the equilibrium equivalent radon activity concentration to the radon activity concentration:

$$F = (\chi_{eq,Rn})/\chi_{Rn} \qquad (2.16)$$

with

$$\chi_{eq,Rn} = 0.105\,\chi_1 + 0.516\,\chi_2 + 0.379\,\chi_3$$

where χ_1, χ_2 and χ_3 represent the activity concentrations of polonium-218, lead-214 and polonium-214, respectively. The unattached fraction f_p is usually defined as the ratio of the equilibrium equivalent radon concentration in the unattached state and of the total:

$$f_p = (\chi_{eq,Rn})fr/(\chi_{eq,Rn}) \qquad (2.17)$$

It is also, however, sometimes defined as the ratio of the equilibrium equivalent radon concentration that is in the unattached state and of the radon concentration:

$$f_p = (\chi_{eq,Rn})fr/\chi_{Rn} \qquad (2.18)$$

The fact that two different definitions may be used for the same quantity leads to a certain amount of ambiguity in the interpretation of the results reported in the literature when the definition used is not clearly specified. The same ambiguity exists when the unattached fraction of a given decay product of radon is reported, as the denominator of the equation can be the radon activity concentration, the total equilibrium equivalent radon activity concentration, or the equilibrium equivalent radon activity concentration of the decay product under consideration. In this Annex, it is assumed in all cases that the denominator of the equation consists of the equilibrium equivalent radon concentration, as, for example, in equation (2.17).

134. Bruno [B1] used the results of the survey conducted by George et al. [G11] on long-term

average concentrations of radon and radon decay product in 21 houses in the New York area to derive the best fit for the attachment rate and the plate-out constant rate. Assuming a ventilation rate of 1 h^{-1}, an equilibrium factor of 0.5 and a relatively high unattached fraction of 0.07, Bruno obtained $\lambda_{po} = 8\ h^{-1}$ and $X = 45\ h^{-1}$, as shown in Figure XX, and indicated that the model would be more accurately validated with the help of simultaneous measurements of ventilation rate, radon concentrations and concentrations of attached and unattached ^{218}Po [B1].

135. Using another approach, Porstendörfer [P5] analysed the sensitivity of F and f_p to the variations of some influencing parameters. He concluded that F and f_p are only slightly influenced by the outdoor concentrations of radon daughters (for $\lambda_v < 1\ h^{-1}$), the value of the recoil factor, the radon exhalation rate and the plate-out rate of attached decay products; however, F and f_p are greatly influenced by the plate-out rate of the unattached daughters and by the aerosol concentration in the room (Figure XXII). The theoretical curves shown in Figure XXII conform well to the experimental data for $\lambda_{po} = 20\text{-}30\ h^{-1}$. Since in most cases the aerosol concentrations in dwellings with low ventilation rates ($\lambda_v < 0.3\ h^{-1}$) vary between $5\ 10^3$ and $10^4\ cm^{-3}$ (corresponding to attachment rates of 25-50 h^{-1} [P5]), F values between 0.25 and 0.4 and f_p values between 0.05 and 0.1 can be expected. This is in agreement with the experimental results of Wicke [W6] and Keller et al. [K6], who measured median F values of 0.37 and 0.33, respectively, in buildings in the Federal Republic of Germany. In another study, however, it was claimed that the model tends to underestimate the equilibrium factor, as measured values of the deposition rates of unattached radon decay products in occupied buildings are found to be smaller than those obtained in laboratory experiments [S44].

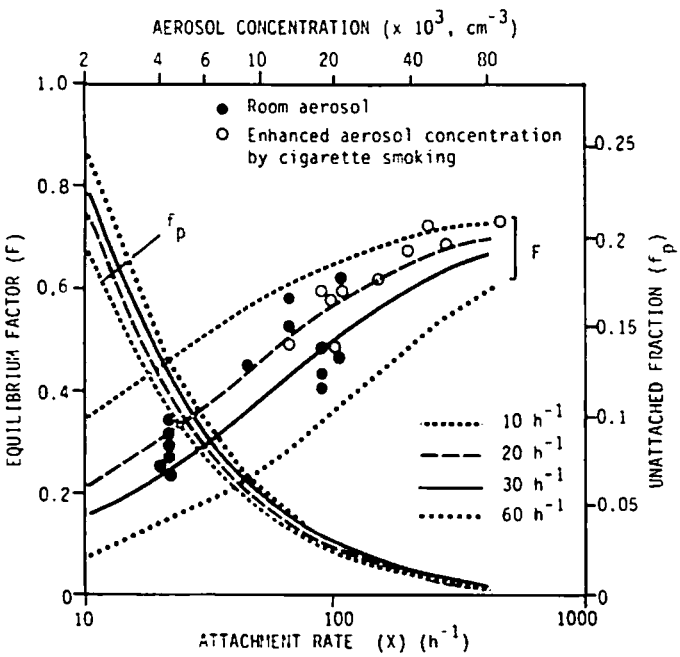

Figure XXII. Equilibrium factor and unattached fraction for radon decay products as a function of aerosol concentration. [P5]

136. The influence of the particle concentration on the equilibrium factor is further illustrated in Figure XXIII [S43], which shows the results of a study in which the equilibrium factor increased from 0.1 to 0.8 as the particle concentration varied from 1,000 to 25,000 cm^{-3}, a range typical of the indoor environment. Because of the great influence of the aerosol concentration in buildings on the equilibrium factor and on the unattached fraction, it is recommended that more simultaneous measurements of the equilibrium factor and the aerosol concentration be conducted.

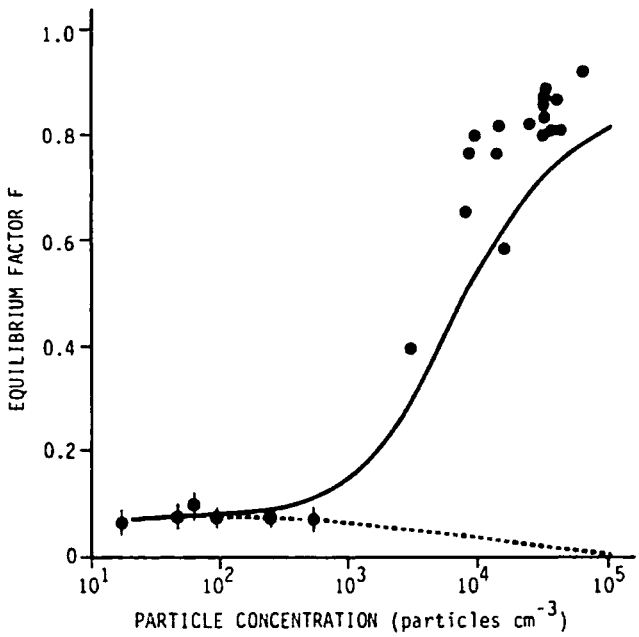

Figure XXIII. Equilibrium factor F versus particle concentration. Measured data and representative uncertainties are indicated by the solid circles and error bars. The solid line is based on calculated values for total radon decay products, while the dashed line indicates calculated values for unattached radon decay products. [S43]

137. In summary, the basic processes influencing the concentrations of the radon decay products in indoor environments are qualitatively well known. Quantitatively, it seems that the largest uncertainty lies in the determination of the plate-out rate of unattached decay products λ_{po}. More simultaneous measurements of the equilibrium factor and of the unattached fraction, together with the aerosol concentration and the ventilation rate, are necessary in order to obtain a clear and consistent picture.

138. Information on the equilibrium factors measured in several surveys is given in Table 18. In the Swedish survey [S12], simultaneous measurements of the air exchange rate and of the equilibrium factors were performed; the equilibrium factor was found to increase as the air exchange rate decreased (F = 0.33 for $\lambda_v > 0.6\ h^{-1}$; F = 0.51 for $\lambda_v < 0.3\ h^{-1}$). The average values obtained in the surveys listed in Table 18 lie between 0.3 and 0.8, most of the values being on the low side of this range. In this Annex, a value of 0.4 is adopted as the average equilibrium factor for radon

decay products in dwellings. This is slightly lower than the figure of 0.5 selected in the UNSCEAR 1977 and 1982 Reports [U2, U1].

3. Results of indoor surveys

139. Large-scale surveys, usually involving more than 100 dwellings, have recently been completed or are in progress in several countries (Table 19). The purposes of these large-scale surveys are to study the distribution of population exposures, search for problem areas, or suggest remedial action to reduce elevated natural levels or man-made enhancements. An analysis of the results presented in Table 19 shows that (a) in all cases, the radon and radon decay product concentrations fit as a first approximation, a log-normal distribution. As an example, Figure XXIV shows the histogram of indoor radon concentrations obtained in a survey in Sweden [M30, S8], which conforms reasonably well to a log-normal distribution with a geometric standard deviation of 3.4. The geometric standard deviations reported in Table 19 range from 1.6 to 5.2. The medians of the equilibrium equivalent concentrations of radon range from less than 4 Bq m^{-3} in Poland [G13] to about 30 Bq m^{-3} in Sweden [S8] and Switzerland [B4]; in the tail of the distribution, relatively high concentrations of the order of 100 Bq m^{-3} (EEC) or more were obtained; (b) the highest radon concentrations were caused by a high influx of radon from the soil, a high exhalation rate from building materials, a high radon concentration in tap-water or a combination of the above factors, depending on environmental characteristics and the building design. In houses, a large fraction of the highest radon concentrations could be consistently attributed to high radon entry rates from the soil; in apartments, the influence of building materials was more important; (c) in most of the older surveys radon and/or radon decay products were sampled under conditions leading to high concentrations (basements or unventilated rooms). In more recent surveys, the sampling has been carried out over periods lasting from a few weeks to a year in order to average out the temporal variations; the spatial variations could also be taken into account by installing the measuring devices in two or three locations (living-room, bedroom, basement). Thus, the intention in recent surveys was to attempt to assess the exposures in buildings under realistic as opposed to worst-case conditions; and (d) owing to the large influence of the radon influx from the soil, radon concentrations tended on average to be greater in houses than in apartments. This was clearly shown in the Swedish survey [S8], as well as in the survey from the Federal Republic of Germany [W15], and may explain the relatively low radon concentrations in Poland [G13] and Austria [S16, S17], where a large proportion of the dwellings are apartment buildings.

140. In the UNSCEAR 1982 Report (Annex D, paragraph 203) it was estimated that the mean indoor equilibrium equivalent concentration of radon was 15 Bq m^{-3} in the temperate regions of the world. The results presented in Table 19, although preliminary for a few surveys, make it possible to reassess this value. The population-weighted mean of the indoor radon concentrations obtained in the nation-wide surveys was calculated assuming a log-normal distribution and a geometric standard deviation of 2.5 for countries that reported only the median concentration. The countries considered in the assessment are in the temperate and high latitudes and represent about 750 million people. The population-weighted mean indoor concentration is found to be 51 Bq m^{-3} while the arithmetic mean of the country means is 55 Bq m^{-3}. This is in agreement, perhaps as a coincidence, with the mean radon concentration of 50 Bq m^{-3} obtained

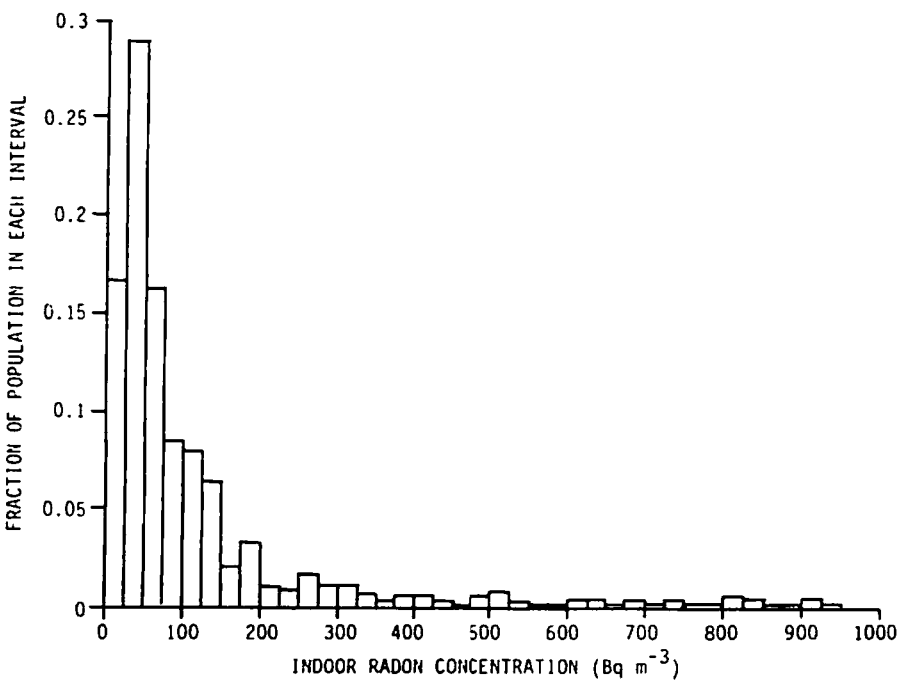

Figure XXIV. Distribution of indoor radon concentrations in Swedish homes. [M30, S8]

for the reference house with a radon entry rate of 50 Bq h^{-1} m^{-3} and an air exchange rate of 1 h^{-1}. Using that round value of 50 Bq m^{-3} and an average equilibrium factor between radon and its decay products of 0.4 yields an average indoor equilibrium equivalent concentration of radon of 20 Bq m^{-3} in the temperate regions of the world; the geometric standard deviation of the distribution is expected to be about 2.5.

141. There is little information on the indoor radon concentrations in the tropical regions. At Bombay, India, an indoor radon concentration of 19 Bq m^{-3} and an equilibrium factor of 0.43 are reported as typical for normal areas [M29], while a mean radon concentration of 34 Bq m^{-3} may be derived from indoor measurements at Hong Kong [T13], which is not thought to be representative of tropical regions. It is safe to assume that the mean indoor radon concentrations in tropical regions are smaller than in temperate latitudes but the data base available is too small to allow a reliable assessment to be made. Possible values of the population-weighted world average of the indoor concentrations of radon gas, taking into account that the population in tropical regions is about half that in temperate latitudes, range from 33 Bq m^{-3} (assuming that the radon concentration in tropical regions is equal to zero) to 50 Bq m^{-3} (assuming that the mean radon concentration in tropical regions is the same as that in the temperate zones). It is tentatively assumed in this document that the population-weighted world average of the indoor radon concentrations is 40 Bq m^{-3}. The same method can be used for the equilibrium equivalent radon concentration; the range of possible values is from 13 to 20 Bq m^{-3} and the mean population-weighted world average of the indoor equilibrium equivalent concentrations of radon is tentatively assumed to be 15 Bq m^{-3}.

C. EXPOSURE-DOSE RELATIONSHIPS

1. Inhalation

(a) Relationships from the UNSCEAR 1982 Report

142. Because of their different physical properties, radon gas and radon decay products are considered separately. Inhaled radon, being a noble gas, is constantly present in the air volume of the lungs at the concentration in air and is partly dissolved in soft tissues. Taking the solubility factor for soft tissues to be 0.4 and assuming that the short-lived decay products decay in the same tissue as radon gas, the following relationship for soft tissues other than the lungs was derived from [I2]:

$$\dot{D}_{\text{soft tissues}}\,(\text{nGy h}^{-1}) = 0.005\,\chi_{\text{Rn,air}}\,(\text{Bq m}^{-3}) \quad (2.19)$$

In the case of the lungs, in addition to the dissolved radon, the radon content of air in the lungs must be taken into account. Assuming the air volume in the lungs to be $3.2\,10^{-3}$ m^3 for Reference Man and assuming further that the short-lived decay products will stay in the lungs, the dose rate due to alpha-radiation was obtained as [I2]:

$$\dot{D}_{\text{lung}}\,(\text{nGy h}^{-1}) = 0.04\,\chi_{\text{Rn,air}}\,(\text{Bq m}^{-3}) \quad (2.20)$$

Taking a quality factor of 20 for alpha-radiation and applying a weighting factor of 0.12 for the lungs and of 0.88 for the other tissues, the effective dose equivalent rate was calculated as

$$\dot{H}_{\text{eff}}\,(\text{nSv h}^{-1}) = 0.18\,\chi_{\text{Rn,air}}\,(\text{Bq m}^{-3}) \quad (2.21)$$

143. For the short-lived decay products of radon, use was made of the potential alpha-energy intake by inhalation during time period T, which is given by

$$I_{\text{pot}} = E/A\; I_{\text{in}}\; T\; \chi_{\text{eq,Rn}} \quad (2.22)$$

where I_{pot} is expressed in joules; $E/A = 55.4\,10^{-10}$ J Bq^{-1} is the potential alpha energy per unit of inhaled equilibrium equivalent activity; I_{in} is the mean breathing rate, taken to be equal to 0.8 m^3 h^{-1} indoors and 1 m^3 h^{-1} outdoors; T is the time period in hours; $\chi_{\text{eq,Rn}}$ is the equilibrium equivalent concentration of radon daughters in Bq m^{-3}.

144. The dosimetry must consider two target tissues in the lung: the tracheo-bronchial basal cell layer (T−B) and the pulmonary epithelium (P), the latter including the alveolar region and the non-ciliated terminal bronchioles. In the UNSCEAR 1982 Report (Annex D, paragraphs 140-144), the conclusions of an expert group of the Nuclear Energy Agency (NEA) of OECD [N15] were adopted after taking into consideration various dosimetric models [H3, H4, J5, J6, J7]. The Committee indicated that from the dosimetric models, it follows that the conversion factors between the inhaled potential alpha-energy intake I_{pot} of the decay product mixture and the committed dose equivalent H to the tracheo-bronchial basal cell layer and to the pulmonary epithelium show nearly linear relations with the unattached fraction f_p of the total potential alpha energy of the decay product mixture:

$$H_{T-B}/I_{\text{pot}} = a_{T-B} + b_{T-B}\,f_p \quad (2.23)$$

and

$$H_P/I_{\text{pot}} = a_P\,(1-f_p) \quad (2.24)$$

where the term a gives the conversion coefficient for the attached decay products, which depends on the activity median diameter (AMD) of the carrier aerosol. It should be noted that the calculated value of I_{pot} applies only to the specified breathing rate.

145. Taking $f_p = 0.025$ and AMD = 0.2 μm for the indoor environment, the different dosimetric models yield

$$D_{T-B}/I_{\text{pot}} = 1.2 - 1.7 \text{ Gy J}^{-1}$$

and

$$D_P/I_{\text{pot}} = 0.13 - 0.4 \text{ Gy J}^{-1}$$

For the purpose of dose estimation, values of $D_{T-B}/I_{\text{pot}} = 1.5$ Gy J^{-1} and $D_P/I_{\text{pot}} = 0.2$ Gy J^{-1} were selected. Following the ICRP recommendations [I2], the Committee decided to split the weighting factor for the total lung and to apply a weighting factor w = 0.06 to each of the two target tissues in the lungs. As the doses to tissues other than the lungs can be

neglected, the effective dose equivalent per unit of inhaled potential alpha-energy intake of ^{222}Rn decay products was found to be in the range of

$$H_{eff}/I_{pot} = 1-3 \text{ Sv J}^{-1}$$

A reference value of 2 Sv J^{-1} was adopted in the UNSCEAR 1982 Report for indoor exposures.

146. For the exposures in outdoor air, AMD was taken to be 0.1 μm, leading to a dose equivalent per unit intake higher by a factor of 1.5 than those for indoor exposures:

$$D_{T-B}/I_{pot} = 2.2 \text{ Gy J}^{-1}$$

$$D_P/I_{pot} = 0.3 \text{ Gy J}^{-1}$$

147. Assuming steady-state conditions, the dose rates, in nGy h^{-1}, are, for indoor exposures, approximately equal to

$$\dot{D}_{T-B} = 7\,\chi_{eq,Rn} \text{ and } \dot{D}_P = 0.9\,\chi_{eq,Rn}$$

corresponding to effective dose equivalent rates, in nSv h^{-1}, of

$$\dot{H}_{eff} = 9\,\chi_{eq,Rn}$$

148. For outdoor exposures, the dose rates, in nGy h^{-1}, are found to be

$$\dot{D}_{T-B} = 12\,\chi_{eq,Rn} \text{ and } \dot{D}_P = 1.7\,\chi_{eq,Rn}$$

while the effective dose equivalent rate, in nSv h^{-1}, is

$$\dot{H}_{eff} = 17\,\chi_{eq,Rn}$$

As indicated later (paragraph 153), this is an overestimate.

149. All the dosimetric coefficients given above refer to adult members of the public. Correction factors should be applied for infants and children, to account for the age-dependent change in lung mass and breathing rate. The UNSCEAR 1982 Report (Annex D, paragraph 151) estimated on the basis of the NEA expert report [N15] that the effective dose equivalent for the age group of up to ten years might on the average be a factor of 1.5-2 higher than for adults.

(b) Consideration of other information

150. NCRP [N14] estimated the variability of the bronchial dose from inhalation of radon decay products due to the uncertainties attached to the many physical and biological parameters which are involved in the calculation of absorbed doses per unit concentration of radon decay products. The physical characteristics include the fraction of unattached ^{218}Po in the atmosphere, the daughter product equilibrium, particle deposition models, the particle size distribution in the atmosphere and the physical dose calculation. Among the biological characteristics are the breathing pattern (including nasal deposition), bronchial morphometry, the mucociliary clearance rate, the location of target cells and the mucus thickness.

151. The dosimetric model used by NCRP to derive the bronchial dose is different from that used in the UNSCEAR 1982 Report but the parameters are the same and the uncertainties should be qualitatively similar. The results obtained by NCRP are given in Table 20 as per cent changes from the nominal annual dose for males. The standard deviation of the annual dose to an individual is estimated to be 120%, if the various factors other than the radon concentration are unspecified, and less than 100%, that is, better than a factor of 2, if some of the parameters are measured or reasonably well estimated [N14].

152. According to Table 20, the estimate of bronchial dose is very sensitive to the particle size spectrum and to the location of target cells in the tracheo-bronchial tree. The per cent variation in annual dose for a very large shift in the median particle size from an average of 0.125 μm would range from +100% for the 0.05 μm particle to −20% for the 0.17 μm particle.

153. The values of AMD adopted in the UNSCEAR 1982 Report for the calculation of doses in the respiratory system from inhalation of radon decay products were 0.1 μm and 0.2 μm for outdoor and indoor exposures, respectively. Reported indoor AMDs are 0.125 μm in the United States [G11] and 0.17 μm ($\sigma_g = 3.5$) in the Federal Republic of Germany [B30, P5]. The doses calculated by the Committee for indoor exposures may thus be slightly underestimated. Regarding the situation outdoors, a significant shift to large particle sizes with a mean AMD of 0.39 μm and a mean σ_g of 2.3 [P5] was observed in the Federal Republic of Germany [B30]; a similar shift had also been measured in the United States [S45]. In another series of measurements in the United States, the AMD was found to range from 0.09 to 0.37 μm with a mean of 0.16 μm [P23]. In the case of outdoor exposures, the doses calculated in the UNSCEAR 1982 Report and shown in paragraph 148 may thus be overestimated by a factor of about 2. If the dose coefficients related to outdoor exposures are divided by two, the resulting values are very similar to the dose coefficients for indoor conditions. In this Annex, the same dosimetric coefficients, in terms of absorbed dose rates per unit equilibrium equivalent radon concentrations, have been used for indoor and outdoor conditions. This implies similar values of the AMD, about 0.2 μm for both indoors and outdoors.

154. The dose factor of 9 nGy h^{-1} per Bq m^{-3}, as derived in paragraph 147, is based on a median particle size of 0.2 AMD for both indoor and outdoor exposures and on breathing rates of 0.8 m^3 h^{-1} indoors and 1.0 m^3 h^{-1} outdoors. Other investigators have developed factors based on other reasonable sizes and rates, as well as other deposition models, without markedly changing the derived dose factors.

155. In the NCRP model [N14], based on the work of Harley and Pasternack [H3], the doses are calculated for the shallow basal cells lying 22 μm below the epithelial surface of the fourth generation of the bronchial tree that was found to experience the maximum deposition density. The deposition model was based on the Weibel lung morphometry [W16] as used by several investigators. The average dose factor for adults was 6 mGy h^{-1} per WLM or 10 nGy h^{-1} per Bq m^{-3} (EEC). Inclusion of later information on morphometry has tended to smooth out the maximum

dose over the entire bronchial tree. Harley and Cohen [H23] measured deposition in a bronchial cast and found it to be very uniform. The dose factor, averaged over all branch airways below the trachea, is 5 mGy per WLM or 8 nGy h^{-1} per Bq m^{-3} (EEC).

156. Calculations have been performed in Canada of the bronchial dose per unit EEC radon concentration for various indoor aerosol conditions [D14]: the relative range of the bronchial dose per unit EEC radon concentration is from 1 to 5 when indoor aerosol conditions change as a result of seasonal air circulation practices, but is only from 1 to 1.6 when the bronchial dose per unit radon concentration is considered. The bronchial dose rate per unit radon concentration was calculated to range from 2.5-4 nGy h^{-1} per Bq m^{-3} for adult males.

157. In Belgium, Vanmarcke et al. [V4] measured indoor aerosol characteristics, as well as radon and radon decay product concentrations, under various conditions in several different rooms and calculated the resulting bronchial dose rates per unit radon gas and radon EEC concentration, using the models considered by OECD [N15]. With respect to dose calculations, Vanmarcke et al. [V4], as in [D14], obtained a narrower range when the bronchial dose was related to the measured radon gas concentration rather than to the measured equilibrium equivalent concentration, a typical value being 5 nGy h^{-1} per Bq m^{-3} of radon gas.

158. James [J19], in a review of all the factors involved in the dosimetry of radon decay products, suggested that it would be appropriate to consider secretory cells as well as basal cells as target cells, implying that the bronchial dose should be calculated for the entire depth of the bronchial epithelium rather than for the basal cells lining the basement membrane alone. This change, combined with the use of a revised deposition model, led to an effective dose equivalent rate per unit EEC radon concentration of 24 nSv h^{-1} per Bq m^{-3} [J19].

(c) Conclusion

159. The value of bronchial dose rate per unit EEC radon concentration adopted in this Annex for indoor and outdoor conditions is 7 nGy h^{-1} per Bq m^{-3} (see Table 21). If the equilibrium factor is assumed to be 0.4, the bronchial dose rate per unit radon gas concentration is about 3 nGy h^{-1} per Bq m^{-3}, which is in the lower part of the range of values recently published [D4, J19, N22, V4]. The corresponding effective dose equivalent rate is about 10 nSv h^{-1} per Bq m^{-3} (EEC) including the contribution from the pulmonary region. This is the mean dose in the specified target tissues. It is recognized that the dose factors may vary within a factor of about 3 according to the target cells considered in the dosimetry. Also, recent studies seem to indicate that for indoor conditions, the bronchial dose is better related to the radon gas concentration than to the equilibrium equivalent concentration. These results, however, need confirmation. The dose factors adopted in this Annex are only slightly modified in comparison to those used in the UNSCEAR 1982 Report.

2. Ingestion

160. Ingestion of water containing dissolved radon results in doses in the body tissues from the radon gas and the radon daughters in the water. Much of the dissolved radon is released from water during cooking. The only significant radon intake comes from the drinking of water. The main part of the ingested radon is eliminated from the body very rapidly through the lungs. The largest dose is received by the stomach; estimates of stomach doses per unit activity of radon ingested vary between about 2 and 10 nGy Bq^{-1} [A3, B2, B3, C20, H7, K7, S15]. In this Annex, an average value of 5 nGy Bq^{-1} has been adopted.

161. Assuming an average consumption of 0.5 litre of water (taken from the tap, without processing) per day and per person, the annual stomach dose equivalent per unit ^{222}Rn concentration in water would be 0.9 nGy per Bq m^{-3}. Using a weighting factor of 0.06 and a quality factor of 20 for alpha-radiation, the annual effective dose equivalent per unit radon concentration in water is thus about 1 nSv per Bq m^{-3}.

3. Summary

162. Table 21 summarizes the dosimetric coefficients adopted in this Annex for inhalation and ingestion of radon and radon decay products. It is to be noted that these dosimetric coefficients apply only to adults in the general population.

D. DOSES

163. The annual absorbed doses resulting from indoor and outdoor inhalation of radon short-lived decay products have been derived from the estimated mean concentrations and dose coefficients discussed previously, using occupancy factors of 0.8 and 0.2 for indoor and outdoor exposures, respectively. The population-weighted world means of the annual absorbed doses in the lungs and in tracheo-bronchial cells add up to about 100 μGy and 800 μGy, respectively. The corresponding annual effective dose equivalent amounts to approximately 1,100 μSv, the contribution of outdoor exposure being 70 μSv and that of indoor exposure 1,000 μSv.

164. It should be stressed that these values apply to adults in the general public and are averaged over large population groups. Very high individual doses can be obtained, especially from indoor exposures at the tail of the log-normal distribution. The situations in which these elevated exposures arise have been discussed in this section.

165. A refined methodology could distinguish the indoor concentrations in dwellings from the indoor concentrations in working places and public buildings; it could also take into account the daily variations of the indoor concentrations and of the occupancy factors.

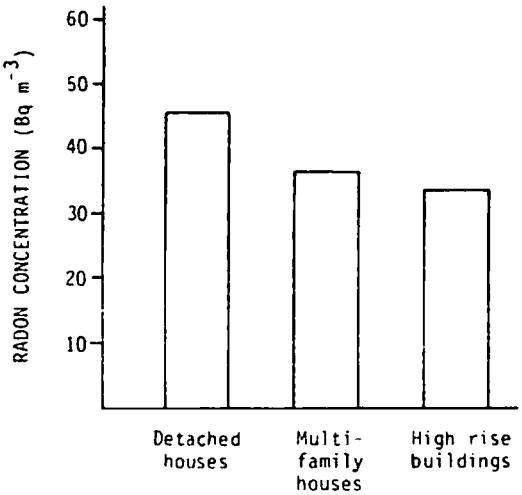

Figure XXV. Median radon concentration levels in different types of houses.
[W15]

166. Some information is available on the dependence of indoor radon concentration upon the type of building (see, for example, Figure XXV [W15]). Further surveys of this type should be encouraged.

167. There is abundant information on the daily variation of indoor radon concentrations, which could be reviewed and used in the refined methodology. Figure XXVI [C16] gives an example of measured diurnal variation of indoor radon concentrations.

168. A survey of diurnal variation of indoor and outdoor occupancy factors has been published in the United Kingdom [B23]; it is presented in Figure XXVII. Occupancy factors reported for populations in China [B24] are similar to those obtained in the United Kingdom.

169. Information of the type presented in Figures XXV, XXVI and XXVII can be combined to estimate refined annual absorbed doses from inhalation of

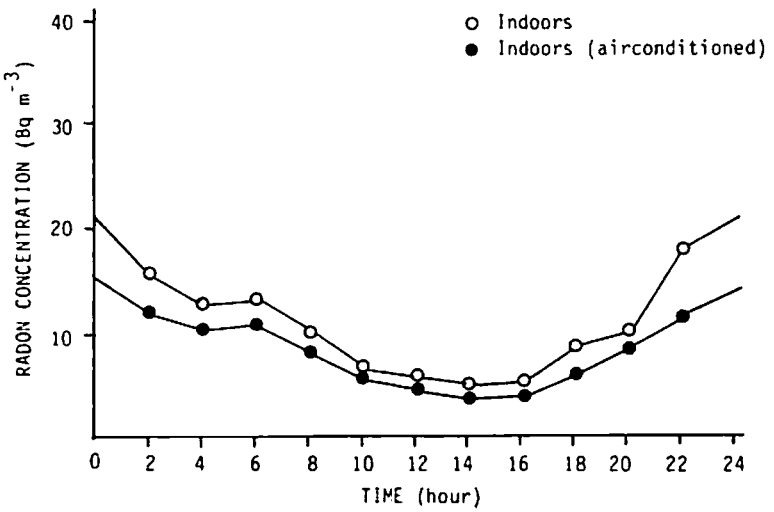

Figure XXVI. Diurnal variation of indoor radon-222 concentrations.
[C16]

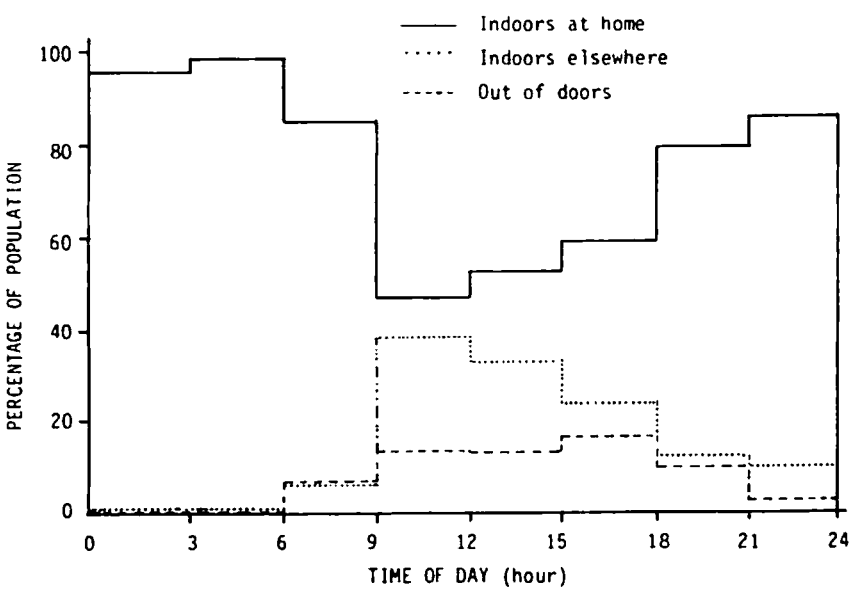

Figure XXVII. Average occupancy over a whole year in the United Kingdom.
[B23]

radon decay products. Further refinements, which could not be implemented at present because of insufficient information, would take into account the age distribution of the populations exposed, urban versus rural population distribution, as well as seasonal variations and the latitudinal distribution of the radon concentrations.

III. INDUSTRIAL ACTIVITIES

170. The industrial processes considered are those that bring to the surface of the earth materials containing above average concentrations of the natural radionuclides, such as geothermal work or phosphate mining, and those which treat material containing average or above average amounts of the natural radionuclides and concentrate them in one or more products or by-products, such as coal burning or the production of fertilizer from phosphate rock. In these industrial processes, the hazard from radiation is generally small compared to that from other chemical substances; therefore, radiation is not systematically monitored. Assessment of such exposures is usually based on sketchy information derived from isolated surveys. The only assessment at the national scale that was available to the Committee is that of the Netherlands [P26]. In this, the naturally occurring radioactive materials discharged to the environment by non-nuclear industries in the Netherlands was estimated using a crude model based on radionuclide concentrations in raw materials, mass flow, and information on the technologies used in industrial processes. The annual effective dose equivalents received by members of the public in the Netherlands were estimated to be about 40 μSv on average, and about 0.7 mSv for individuals of the critical group consisting of consumers of large amounts of fish [P26].

171. This chapter reviews the information available world-wide on radiation exposures from industrial activities, classified into: (a) combustion of coal; (b) other energy production; (c) use of phosphate rock; and (d) metal mining and processing. When possible, both occupational exposures and exposures of members of the public are considered. Collective effective dose equivalent commitments resulting from atmospheric discharges of radioactive materials are estimated using the crude models described in the UNSCEAR 1982 Report ([U1], Annex C, paragraphs 24-27).

A. ENERGY PRODUCTION FROM COAL

172. Coal, like most materials found in nature, contains trace quantities of ^{40}K and of ^{238}U, ^{232}Th and their decay products. By burning coal, the activities of these naturally occurring radionuclides are redistributed from underground into the biosphere. Expressed in coal equivalent for energy purposes, the world production of coal was 3.1 10^{12} kg in 1985 [U15], the main producers being China, the USSR and the United States. Table 22 presents the amounts of coal produced in various countries and the measured activity mass concentrations of natural radionuclides.

173. Although the reported activity mass concentrations range over two or three orders of magnitude, the averages from the various countries are in fairly good agreement. Figure XXVIII shows, for example, the ^{238}U levels measured in 800 samples of coal mined in the United States [W12]; they range from 1 to 1,000 Bq kg^{-1}. In this Annex, as in the UNSCEAR 1982 Report, it will be assumed that the average activity mass concentrations of ^{40}K, ^{238}U and ^{232}Th in coal are 50, 20 and 20 Bq kg^{-1}, respectively, and that the decay products of ^{238}U and ^{232}Th are in radioactive equilibrium with their precursor.

174. The uses of coal in OECD countries are listed in Table 23, together with the amounts involved in

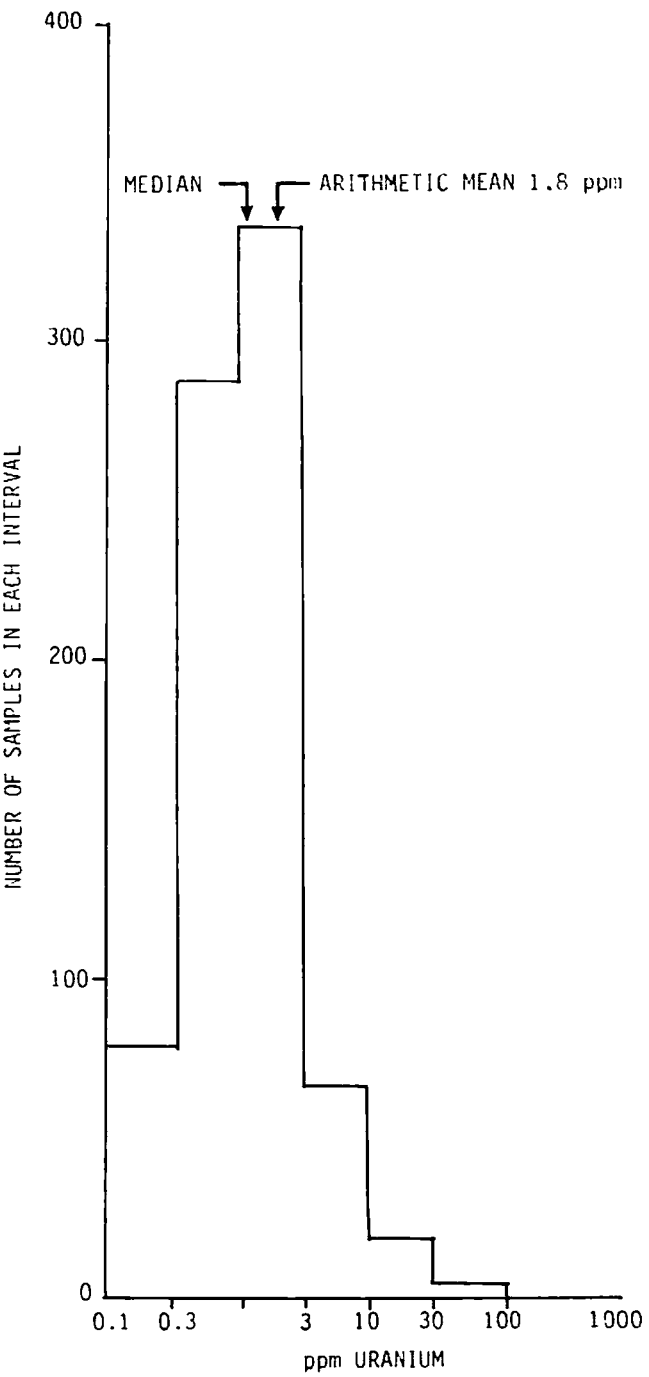

Figure XXVIII. Distribution of uranium-238 in 800 coal samples from the United States. [W12]

several countries [O6]. Most of the coal is used in electric power stations and in coke ovens; even though the use of coal for space heating is not as important as it was years ago, it is still significant in some countries. It will be assumed that, on a world-wide basis. 70% of the coal produced is burned in electric power stations, 20% in coke ovens and 10% in houses.

175. Radiation exposures occur throughout the coal fuel cycle, which consists of (a) coal mining; (b) use of coal; and (c) use of fly ash. Occupational exposures and exposures of members of the public are estimated, in so far as data are available. It should be emphasized, however, that the contribution of radiation hazards to the overall health and environmental impact resulting from the exploitation of coal is generally estimated to be quite small (see, for example, [B39], [U16] and [M17]).

1. Coal mining

176. Coal miners are exposed to coal dust and the decay products of radon and thoron. Coal mines are usually well ventilated to keep the methane and dust levels low; consequently, the radon concentrations are also relatively low. Table 24 summarizes published measurements from several countries. Radon concentrations in coal mines are comparable to those observed in dwellings. From measurements in British coal mines, a maximum individual effective dose equivalent of 1 mSv per year, and an upper limit for the normalized collective effective dose equivalent of 3 man Sv per GW a have been calculated [C14, R4]. Unquantified additional exposures may result from the activity present in mine water [T8].

177. The global production of electrical energy by coal burning was about 600 GW a in 1980, resulting in an upper estimate of the total collective effective dose equivalent to coal miners of 2,000 man Sv. This figure is taken to represent the collective effective dose equivalent for the world for one year of mining coal, including the mining of coal for purposes other than electrical energy production.

178. Members of the public are exposed to the radon activity present in the exhaust air of coal mines. There are currently no measured data on emission of radon from coal mines. It can be assumed, as in [S32], that 20% of the radon contained in the ore is released during the mining process and that the radon release rate from the exposed ore surface is a factor of 2 greater than that resulting from mining. Taking the mass of coal required to produce an electrical energy of 1 GW a to be $3\ 10^9$ kg and a mean radon concentration in coal of 20 Bq kg^{-1}, the normalized radon release from coal mines would be 36 GBq per GW a. Another method consists in scaling the radon releases from coal mines to the radon releases from uranium mines, taking into account an average factor of 1,000 between the average activities of ^{226}Ra per unit mass of uranium ore and of coal [J15]. This method leads to a normalized radon release from coal mines of 600 GBq per GW a. The normalized collective effective dose equivalent commitments corresponding to the two values of radon releases are $6\ 10^{-4}$ and 10^{-2} man Sv per GW a.

179. Using the two methods described in the previous paragraph, the activity of radon released per year from coal mining all over the world is estimated to be 30 TBq and 800 TBq, resulting in collective effective dose equivalent commitments of about 0.5 and 10 man Sv, respectively. Although differing by a factor of 20, the latter values are small compared to other radiation exposures from the coal fuel cycle and do not deserve further investigation.

2. Use of coal

180. The uses of coal that are considered here are electrical energy production, carbonization and space heating in dwellings.

(a) Coal-fired power plants

181. In the production of electric power, coal is burned in furnaces operating at temperatures of up to 1,700° C. About $3\ 10^9$ kg of coal is required to produce 1 GW a of electrical energy. In the combustion process, most of the mineral matter in the coal is fused into a vitrified ash. A portion of the heavier ash, together with incompletely burned organic matter, drops to the bottom of the furnace as bottom ash or slag. The lighter fly ash, however, is carried through the boiler, together with the hot flue gases and any volatilized mineral compounds, to the stack, where, depending on the efficiency of emission control devices, most is collected while the rest (escaping fly ash) is released to the atmosphere. Table 25 presents the ash content of coal burned in the United Kingdom in power stations and for other uses, together with the amounts of ash produced; it is clear that coal combustion in coal-fired power plants accounts for the bulk of the total ash production. The corresponding information on the atmospheric emissions of particulates (Table 26), however, indicates that the power stations are a small contributor to the total emissions of particulates, implying that the efficiency of the emission control devices used in power plants is rather high.

182. The radionuclides included in the non-combustible mineral matter of coal are partitioned between the bottom ash and fly ash, except for the gases and volatilized minerals, which are incorporated directly into the flue gases. In large power stations, there is about 20% bottom ash and, therefore, 80% fly ash. Owing mainly to the elimination of the organic component of the coal, there is approximately an order of magnitude enhancement of the activity concentrations from coal to ash. Consequently, the natural radionuclide concentrations in ash and slag from coal-fired power stations are significantly higher than the corresponding concentrations in the earth's crust. The arithmetic averages of the reported activity mass concentrations in escaping fly ash are 265 Bq kg^{-1} for ^{40}K, 200 Bq kg^{-1} for ^{238}U, 240 Bq kg^{-1} for ^{226}Ra, 930 Bq kg^{-1} for ^{210}Pb, 1,700 Bq kg^{-1} for ^{210}Po, 70 Bq kg^{-1} for ^{232}Th, 110 Bq kg^{-1} for ^{228}Th and 130 Bq kg^{-1} for

^{228}Ra ([U1], Annex C, paragraph 11). There is an enrichment by a factor of about 3 for ^{210}Pb and 5 for ^{210}Po in relation to the rest of the ^{238}U decay series. This is probably because of the volatilization of ^{210}Pb and ^{210}Po during the combustion and a condensation of these radionuclides down the flue line on the finer fly-ash particles.

183. The activities of natural radionuclides discharged to the atmosphere from a power plant per unit electrical energy produced depend on a number of factors such as the activity concentration in coal, the ash content of the coal, the temperature of combustion, the partitioning between bottom ash and fly ash, and the efficiency of the filtering system. Marked differences should therefore be expected between the measured activities discharged per unit energy produced from different power plants. Most of the available information on the normalized atmospheric discharges is presented in Table 27. Apparently, there are two types of coal-fired power plants used throughout the world: older ones, which release about 10% of the fly ash produced; and more modern ones, which are equipped with sophisticated retention devices and, as a result, release only about 0.5% of the fly ash. Table 27 presents estimates of normalized discharged activities for such older and more modern plants. They have been derived on the following assumptions: the combustion of 3 10^9 kg of coal is required to produce an electrical energy of 1 Gw a; fly ash accounts for 80% of the total ash production; the activity mass concentration in coal is 20 Bq kg^{-1} for the radionuclides of the ^{238}U and of the ^{232}Th decay series; and there is an enrichment factor of 3 for ^{210}Pb and ^{210}Po. Releases of ^{222}Rn and of ^{220}Rn must be estimated separately because radon is not collected by the particulate control devices. The activities of ^{222}Rn and of ^{220}Rn released per GW a have been assessed at 60 GBq on the basis of the conservative assumption that all of the radon activity contained in the coal is discharged to the atmosphere.

184. The main pathways through which the populations living around coal-fired power plants are exposed to enhanced levels of natural radionuclides are inhalation during the cloud passage and external irradiation and ingestion following deposition of activity on the ground. The collective effective dose equivalent commitments due to those pathways, normalized to the generation of 1 GW a of electrical energy, have been estimated using the crude models described in the UNSCEAR 1982 Report ([U1], Annex C, paragraphs 24-27). Tables 28 and 29 present the results obtained for the reference older and more modern coal-fired power stations, respectively; they amount to about 6 and 0.3 man Sv per GW a. Assuming that, on a worldwide scale, two thirds of the electrical energy provided by coal-fired power stations is due to old plants (and one third to modern plants), the average normalized collective effective dose equivalent commitment is approximately 4 man Sv per GW a, while the overall figure for the world for one year of practice is about 2,000 man Sv.

185. The release of ^{14}C-free carbon dioxide emitted by the coal-fired power plants will have the effect of diluting the natural concentration of ^{14}C in the biosphere. The reduction in the natural ^{14}C collective effective dose equivalent commitment resulting from the release of activity-free carbon dioxide is estimated to be about 60 man Sv per GW a assuming that: (a) the combustion of about 3 Tg of coal is required to produce an electrical energy of 1 GW a; (b) the coal contains 85% carbon by mass; (c) the natural activity mass concentration of ^{14}C in coal is approximately 226 Bq kg^{-1}; and (d) the collective effective dose equivalent commitment per unit release is 110 man Sv per TBq. This reduction in the natural effective dose equivalent commitment would be over thousands of years.

186. Annual individual effective dose equivalents resulting from inhalation during the cloud passage have been estimated. Assuming an effective stack height of 100 m and a uniform wind rose, the annual average of the ground-level air concentration per unit release rate is estimated to be about 4 10^{-8} Bq m^{-3} per Bq s^{-1} at approximately 1 km from the stack. For an individual of the critical group living near a coal-fired power plant producing annually 1 GW a of electrical energy, the resulting annual effective dose equivalent would be about 1 μSv for a modern plant and about 20 μSv for an old plant. The annual effective dose equivalents from ingestion and external irradiation would be of about the same magnitude after 20-30 years of operation. Calculated doses from ingestion are very sensitive to the assumptions made about food-chain transfers. Annual individual doses are, in any case, small; it is worth noting that recent measurements in the environment surrounding coal-fired power plants show no statistically significant increases in the activity concentrations of natural radionuclides in samples of air [T7], precipitation [T7], soil [B15, S30, T7], vegetation [S30, T7] or cattle organs [B16, S30, S31] that could be attributed to the operation of power stations.

187. Only atmospheric discharges have been considered so far, as coal ash is not usually directly discharged into the aquatic environment. It may be stored in ash ponds, from where some activity may leach into the aquifer. Ground water may then carry the leachates into the nearest river. The leachability of the ash is quite low [S27], however, so aquatic discharges are not likely to pose a radiation problem, at least in the short term.

188. Radiation exposures of workers in coal-fired power plants are thought to be mainly caused by inhalation of airborne fly ash. Effective dose equivalents of 150 μSv per year have been estimated in the United Kingdom for the most exposed groups of power station workers on the basis of exposure to a concentration of respirable fly ash in air of 0.5 mg m^{-3} [C14]. In Yugoslavia, significant concentrations of ^{210}Pb in the urine of individuals working in a coal-fired power plant burning high activity coal have been reported [B12, B22].

189. Values of collective effective dose equivalent received by workers in coal-fired power plants are not available. An upper estimate can be derived from the

individual effective dose equivalent of 150 μSv per year calculated in the United Kingdom, assuming that a labour force of 500 persons is required to produce an electrical energy of 1 GW a per year. The normalized annual collective effective dose equivalent would be at most 0.1 man Sv per GW a. The global production of electrical energy by coal burning was about 600 GW a in 1980, resulting in an upper estimate of the total collective effective dose equivalent to workers in coal-fired power plants of 60 man Sv.

(b) Domestic use

190. Another major use of coal is domestic cooking and heating. No information has been found in the literature on the environmental discharges of natural radionuclides from this source. The use of coal for cooking and heating in private homes may, however, be estimated to result in high collective dose commitments since chimneys are generally low and not equipped with ash removal systems and the population densities around sources of emission are generally high. Figure XXIX [U16] presents trends in urban smoke concentrations in the United Kingdom and shows that domestic sources, which have decreased in importance since 1960, still remain substantial. It is estimated that in a simple open fire burning coal, some 3-4% of the coal is emitted as tarry smoke while in smoke-reducing appliances, which have only been marketed since the early 1970s, only 0.3-0.6% of the coal burned is emitted as smoke [U16]. Smoke is a mixture of soot, tarry matter and inorganic substances, produced when bituminous coal is incompletely burned. Measured activity mass concentrations of natural radionuclides in smoke have not been found, but they can be expected to be between those in coal and ash (without enrichment) and are likely to be closer to those in coal. Assuming that the activity mass concentrations in smoke are equal to those in coal and that 3.5% of the coal is emitted as smoke, the annual world-wide atmospheric releases caused by domestic burning of coal are estimated to be 0.7 TBq of ^{40}K and 0.3 TBq of each of the radionuclides of the ^{238}U and ^{232}Th series (radon and thoron excepted); these figures become 20 times greater if it is assumed that the activity mass concentrations in smoke are equal to those in ash and that the coal burned has a 5% ash content. Taking the average population densities around the houses to be 10^3 km^{-2} leads to collective effective dose equivalent commitments resulting from a yearly world-wide use of coal in the range of 2,000-40,000 man Sv. This estimate is highly uncertain, as it is not supported by any discharge or environmental data.

(c) Coke ovens

191. Coal is also used in vast amounts in coke ovens; however, relevant information on the releases of natural radionuclides from coke ovens has not been found.

3. Use of fuel ash

192. Large quantities of coal ash (fly ash and bottom ash combined) are produced each year throughout the world. The global production of coal equivalent in 1985 was 3.1 10^{12} kg (Table 22); assuming that coal burned in power stations represents 70% of the total

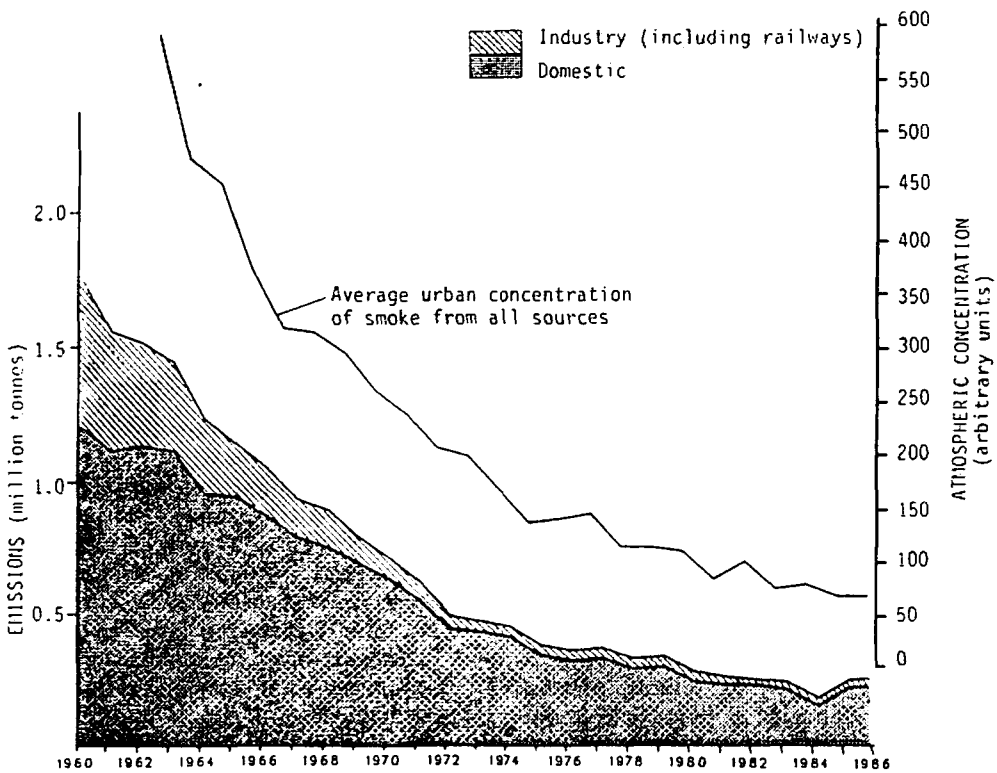

Figure XXIX. Smoke: trends in emissions and average urban concentrations in the United Kingdom. [U16]

consumption and that its ash content is 13%, about 280 million tonnes of coal ash are produced annually in coal-fired power stations. Taking the average activity mass concentrations of natural radionuclides in coal to be 50 Bq kg^{-1} for ^{40}K and 20 Bq kg^{-1} for ^{238}U and for ^{232}Th, the average activity mass concentrations of ^{40}K, ^{238}U and ^{232}Th in coal ash are estimated to be 400, 150 and 150 Bq kg^{-1}, respectively. Compared with normal soil, the average activity mass concentration of ^{40}K is similar, but those of ^{238}U and ^{232}Th are six times higher.

193. Coal ash is used in a variety of applications, the largest of which is the manufacture of cement and concrete. It is also used as a road stabilizer, as road fill, in asphalt mix and as fertilizer. Data on the various uses of coal ash in several countries have been reported [G20]. It can be estimated that about 5% of the total ash production from coal-burning power stations is used for constructing dwellings; this represents an annual usage of 14 million tonnes.

194. The use of coal ash in building materials may result in changes in the indoor doses owing to external irradiation and to inhalation of radon decay products. With respect to external irradiation, Siotis and Wrixon [S5], assuming that the use of concrete containing fly ash for constructing dwellings would result in an additional activity mass concentration of ^{226}Ra in concrete of 13 Bq kg^{-1}, calculated that the additional effective dose equivalent from indoor exposure would amount to 20 μSv per year. Stranden [S28], on the basis of additional activity mass concentrations of 10 Bq kg^{-1} of ^{226}Ra and 8 Bq kg^{-1} of ^{232}Th, estimated that the additional absorbed dose in air would be 90 μGy per year in concrete houses and 40 μGy per year in wooden houses. These figures would be reduced to 70 and 30 μSv for concrete and wooden houses, respectively, if the conversion coefficient from absorbed dose in air to effective dose equivalent were taken to be 0.7.

195. There are conflicting views on the impact of the use of fly ash on the dose from inhalation of radon decay products. Siotis and Wrixon [S5] measured the radon exhalation rates of concrete blocks made by a cement manufacturer under controlled conditions using cement containing varying proportions of fly ash of high ^{226}Ra activity mass concentration (330 and 800 Bq kg^{-1}). The concrete blocks containing fly ash were found to present radon exhalation rates higher than those of concrete blocks without fly ash. Siotis and Wrixon [S5] estimated, on the basis of these measurements, that the additional radon concentration in a model dwelling would be 1.5 Bq m^{-3}, resulting in an effective dose equivalent due to indoor exposure to radon decay products of about 40 μSv per year. Radon concentrations in Polish dwellings built with concrete blocks made almost entirely of fly ash were found to be higher than those in dwellings built with concrete blocks containing no fly ash [B14]. Stranden [S28], however, observed lower radon exhalation rates from concrete blocks containing 5% fly ash than from ordinary concrete blocks; the two types of blocks had been especially constructed for the experiment and differed only in the inclusion of fly ash. The resulting reductions in the radon concentrations in model dwellings have been calculated by Stranden [S28] to be 3 Bq m^{-3} in a wooden dwelling and 10 Bq m^{-3} in a concrete dwelling. The corresponding reduction in effective dose equivalent from indoor exposure to radon decay products would be 90 μSv per year in a wooden building and 300 μSv per year in a concrete dwelling. In other controlled studies [U6, V3], the substitution of varying amounts of ordinary cement with fly ash cement yielded no significant change in the radon exhalation rate from the concrete obtained, in comparison with ordinary concrete.

196. The conflicting evidence may be attributed to differences in the properties of the components used in concrete, their relative activity mass concentrations of ^{226}Ra or the manufacturing process. It seems that for a given activity mass concentration of ^{226}Ra, the radon exhalation rate of fly-ash is usually lower than that of concrete, so that enhanced indoor exposures to radon decay products may arise only when fly ash with a high activity mass concentration of ^{226}Ra is mixed with concrete.

197. In this Annex, the use of fly ash in building materials is assumed to result only in additional exposures from external irradiation. Annual additional effective dose equivalents of 30 μSv in wooden dwellings and 70 μSv in concrete dwellings are adopted as representative values on a world-wide basis. Taking the amount of fly ash concrete to be 1.3 tonnes in a wooden house and 4 tonnes in a concrete building (as in [S28]), the additional effective dose equivalents normalized per unit mass of fly ash concrete are found to be 23 and 18 μSv a^{-1} per tonne for wooden and concrete dwellings, respectively. Using an intermediate value of 20 μSv a^{-1} per tonne and assuming that an average of four persons live in each house and that the lifetime of the house is 50 years, the collective effective dose equivalent commitment arising from external irradiation attributable to the annual use of fly ash for constructing the dwellings is estimated to be about $5\ 10^{-4}$.

198. The use of fly ash as a component of cement and concrete for constructing dwellings represents only a small fraction of the total use of coal ash. The Committee has not found any published information on doses arising from other uses of fly ash (as a road stabilizer, in asphalt mix, as fertilizer, etc.).

199. The large fraction of coal ash that does not find a commercial application is usually dumped in the vicinity of the power plant. Two disposal methods are used. Ash mixed with water in a ratio of 2 to 3 is piped as slurry to lagoons created by adding walls to or otherwise modifying existing topographic features. When a lagoon is filled, it is allowed to drain and, if necessary, another lagoon is created on top. For dry ash dumps, the ash is conditioned with water to reduce dust and transported to the dumping site. The choice of disposal method depends on economic and environmental factors [U16]. When the dumping is finished, most dry ash dumps are covered by topsoil and converted into areas for agricultural or recreational use.

200. The main potential radiation hazards from ash disposal arise from resuspension and from contamination of surface and underground water either as a result of leaching, i.e., as certain compounds are washed out of the ash by percolating rain water, or as a consequence of the physical erosion of the ash surface during run-off. Dose assessments taking into account these processes are lacking.

4. Summary

201. Radiation exposures resulting from the various steps in the coal fuel cycle are summarized in Table 30. The exposures are expressed in terms of collective effective dose equivalent commitments per year of practice around the world. The highest exposures seem to stem from the use of coal in homes and the use of fly ash in concrete. It should be borne in mind that these estimates are fraught with large uncertainties that are difficult to quantify.

B. OTHER ENERGY PRODUCTION

202. The other types of energy production considered in this section are geothermal energy and combustion of oil and natural gas.

1. Geothermal energy

203. Geothermal energy is produced in Iceland, Italy, Japan, New Zealand, the USSR and the United States. In 1980, it amounted to only 1.5 GW a of electrical energy production, but its relative importance may grow in the future. Geothermal energy makes use of hot steam or water derived from high-temperature rocks deep inside the earth. Most of the activity found in geothermal fluids is due to the uranium decay chain. Isotopes of solid elements may cause problems regarding water pollution or land disposal but only radon, which is released into the atmosphere when the water or steam contacts the air, is considered here. From measurements of radon in geothermal fluids in Italy [G18, M15], the annual releases of radon have been estimated to be 110 TBq from the 400 MW Larderello plant, 7.0 TBq from the 15 MW plant at Piancastagnaio and 1.5 TBq from the 3 MW plant at Bagnore. The annual release of radon from the 11 units of the Geysers Power Plant in the United States, which has a net capacity of 502 MW of electrical energy, was assessed to be 21 TBq [A16]. These figures point to an average atmospheric discharge per unit energy generated of about 150 TBq per GW a. The corresponding collective effective dose equivalent commitment per unit energy generated is estimated to be about 2 man Sv per GW year if it is assumed that the equilibrium factor between radon and its short-lived decay products is 0.8, the population density around the plant is 100 km^{-2}, the effective dose equivalent per unit activity inhaled is 1.1 10^{-8} Sv Bq^{-1} and that indoor and outdoor concentrations are the same. The annual world-wide production of geothermal energy would yield a collective effective dose equivalent commitment of approximately 3 man Sv.

204. Annual individual effective dose equivalents resulting from inhalation of short-lived decay products of radon is estimated to be of the order of 10 μSv at 1 km from the stack of a 100 MW geothermal plant. The average additional radon concentration would be about 0.1 Bq m^{-3}, compared with a background concentration of 1-10 Bq m^{-3}. Measurements conducted near the Larderello plant in Italy and near other geothermal power plants in New Zealand and the United States failed to distinguish between the contribution of the radon activity from the plant and that from background [A16, G18, S59, W20].

2. Oil-fired power plants

205. As shown in Table 31, oil has a large number of applications, the most important being road transport, generation of electric power and domestic heating. Approximately 3 10^{12} kg of crude petroleum was produced in the world in 1980. In power plants, about 2 10^{9} kg of oil is needed to produce 1 GW a of electrical energy. As the ash content of oil is very low, oil-fired power plants are usually not equipped with efficient ash removal systems. From measurements in two oil-fired power plants in France, normalized atmospheric discharges of about 200, 300, 150 and 1,000 MBq per GW a for ^{238}U, ^{226}Ra, ^{232}Th and ^{40}K, respectively, have been estimated [A8]. These are very similar to the normalized discharges of coal-fired power plants fitted with efficient aerosol control devices. Measurements of activity mass concentrations of ^{232}Th, ^{226}Ra, ^{210}Pb and ^{40}K in fuel ash from oil-burning power stations of the USSR led to the same conclusion [B13]. The collective effective dose equivalent commitment per unit electrical energy generated has been found to be about 0.5 man Sv per GW a. Assuming that 15% of the world-wide production of crude petroleum is burned in electric power plants, the resulting annual collective effective dose equivalent commitment would be about 100 man Sv. The annual individual effective dose equivalent to a member of the critical group, calculated in the same way as for a coal-fired power plant, would be about 1 μSv.

206. Information on radiation exposures of workers in the oil industry is scarce. Kolb and Wojcik [K22, K23] measured a median ^{226}Ra concentration of 4,500 Bq m^{-3} in thermal brine, which is raised to the surface together with oil. This brine can be used for the extraction of oil from its deposits without any radiation problem as long as the radium activity remains in solution. Salt from oversaturated brine is often precipitated after expansion, however, and scale is formed on the inner walls of tubing, pumps and separation and storage tanks [K23, S60]. Such deposits can be detected with common radiation protection instruments; activity mass concentrations of up to 10^{6} Bq kg^{-1} of ^{226}Ra and ^{228}Ra have been detected in some samples. Under normal conditions of operation, the annual effective dose equivalent for maintenance personnel resulting from external radiation from such sources is estimated to be less than 5 mSv [K23].

207. Oil shale is currently being considered for the production of synthetic oil that could be burned in

power plants. Oil shale is a sedimentary rock containing kerogen, an organic substance originating from micro-organisms that accumulated in what were once large shallow lakes. When the rock is heated to a temperature of at least 400° C to bring about pyrolysis of the kerogen, synthetic shale oil is produced. The production of shale oil involves the processing of vast amounts of material. Retorting 1 tonne of shale can yield 110 litres of retort water, 0.8 tonne of spent shale and 1-40 m^3 of retort gases [F6].

208. In the rich shale deposits of Colorado, Wyoming and Utah, known as the Green River formation, as well as in Estonian shale, measured radionuclide concentrations of raw oil shale (about 60 Bq kg^{-1} of ^{238}U, 25 Bq kg^{-1} of ^{232}Th, 500 Bq kg^{-1} of ^{40}K) correspond fairly closely to those observed in typical soils [G19]; Antrim shales from the eastern United States and Moroccan shales show higher uranium concentrations (about 350 Bq kg^{-1}) [G19]. After retorting, nearly all of the activity content is found in the spent shale, with a marked increase with decreasing particle size for most radionuclides. Average concentrations in the respirable fraction of spent shale from the Green River formation are about 150 Bq kg^{-1} of ^{238}U, 130 Bq kg^{-1} of ^{226}Ra, 220 Bq kg^{-1} of ^{210}Pb, 40 Bq kg^{-1} of ^{232}Th and 740 Bq kg^{-1} of ^{40}K [G19]. Atmospheric discharges from an oil shale plant producing enough oil to generate 1 GW a of electrical energy have been estimated to be about 290 MBq of ^{238}U, 250 MBq of ^{226}Ra, 430 MBq of ^{210}Pb, 90 MBq of ^{232}Th and 1,500 MBq of ^{40}K [G19]. This is again very similar to the normalized discharges from a coal-fired power plant equipped with an efficient ash removal system. The collective dose equivalent commitment per unit electrical energy generated would be about 0.5 man Sv per GW a.

3. Natural gas

209. In 1980, the world-wide production of natural gas amounted to about 10^{12} m^3. Like oil, natural gas has many fields of application. Its uses in OECD countries are summarized in Table 32, the major uses being domestic heating, generation of electric power and as a source of heat in various industries. As discussed previously in this Annex, radon concentrations in natural gas may vary widely around a typical value of 1,000 Bq m^{-3}. Since about $2\ 10^9$ m^3 of natural gas must be burned in order to produce 1 GW a of electrical energy, the corresponding radon emission is approximately 2 TBq.

210. The corresponding collective effective dose equivalent commitment per unit energy generated is estimated to be about 0.03 man Sv per GW a. The assumptions for this estimate are that the equilibrium factor between radon and its short-lived decay products is 0.8, the population density around the plant is 100 km^{-2}, the effective dose equivalent per unit activity is $1.1\ 10^{-8}$ Sv Bq^{-1} and indoor and outdoor concentrations are the same. Assuming that 15% of the world production of natural gas is burned in electric power plants, the resulting collective effective dose equivalent commitment per year of practice would be about 3 man Sv.

211. The radon concentrations around a gas-fired power plant are expected to be, in most cases, undistinguishable from background; the individual doses are expected to be trivial.

4. Peat

212. Peat is burned for energy production in several countries, notably in Finland and Sweden [E9, E10, M31]. Natural radionuclides are carried into the peat bogs by flowing surface and ground water and then adsorbed to peat matter. In Sweden, the normal ^{238}U activity mass concentration in dried peat is about 40 Bq kg^{-1}, but some samples have shown concentrations up to 500 Bq kg^{-1}; in one extreme case, the ^{238}U concentration was more than 10,000 Bq kg^{-1} [E9]. In Finland, samples of peat used as fuel in a power plant showed concentrations of 16 Bq kg^{-1} of ^{238}U, 30 Bq kg^{-1} of ^{210}Pb, 5.3 Bq kg^{-1} of ^{228}Ra and 28 Bq kg^{-1} of ^{40}K [M31]. Since the ash content of peat normally lies between 3% and 8% [M31], the concentrations of natural radionuclides in peat ash are about 20 times those in peat.

213. There is little information on the environmental discharges of natural radionuclides from peat power plants. Assuming that $5\ 10^9$ kg of peat is needed to produce 1 GW a of electrical energy and using an average ^{238}U concentration in peat of 40 Bq kg^{-1} and a dust control efficiency of 99%, as well as the general assumptions used for coal-fired power plants, the normalized collective effective dose equivalent commitment is tentatively estimated to be 2 man Sv per GW a. In the long term, storage and disposal of uranium-rich peat ash may have the greatest impact [E9].

5. Summary

214. Radiation exposures attributable to natural radionuclide discharges from various systems of electricity production are summarized in Table 33. The exposures are expressed in terms of collective effective dose equivalent commitments for one year of practice throughout the world and are normalized to the generation of 1 GW a of electrical energy. The highest radiation exposures result from the use of coal.

C. USE OF PHOSPHATE ROCK

215. Phosphate rock is the starting material for the production of all phosphate products; it is the main source of phosphorus for fertilizers. The world production of phosphate rock was about 130 million tonnes in 1982 [F9], the main producers being China, Morocco, the USSR and the United States. There are three types of phosphate rock: (a) phosphate rock of sedimentary origin: this type, which covers 85% of current needs for phosphorus, comes from deposits spread almost throughout the world, but especially in Morocco and Florida (United States) [I11]; (b) phosphate rock of volcanic origin: the principal deposit of this type is found in the Kola peninsula (USSR); (c) phosphate rock of biological origin: the accumulated

droppings of marine birds have given rise to deposits of guano, from which the leached phosphoric acid has recombined with calcium from the underlying rock to give tricalcium phosphate.

216. Activity mass concentrations of natural radionuclides in phosphate rock were reviewed by UNSCEAR in its 1977 and 1982 Reports [U2, U1]. Activity mass concentrations of ^{232}Th and ^{40}K in phosphate rocks of all types are similar to those observed normally in soil, whereas concentrations of ^{238}U and its decay products tend to be elevated in phosphate deposits of sedimentary origin. A typical concentration of ^{238}U in sedimentary phosphate deposits is 1,500 Bq kg^{-1}. North American phosphate presents the highest concentrations of ^{238}U, followed by north African phosphate. Uranium-238 and its decay products are generally found in close radioactive equilibrium in phosphate ore.

217. Exposures of members of the public result from the following: (a) effluent discharges of radioactive materials of the ^{238}U decay series into the environment from phosphate rock mining and processing; (b) use of phosphate fertilizers; and (c) use of by-products and wastes.

218. Occupational exposures mainly occur during mining, processing and transportation of phosphate rock, as well as during transportation and utilization of phosphate fertilizers.

1. Effluent discharges during phosphate industrial operations

219. Phosphate industrial operations can be divided into mining and milling of phosphate ore, phosphate product manufacture by the wet process (in wet-process phosphoric acid plants) and phosphate product manufacture by the thermal process (in elemental phosphorus plants).

220. The technique used to mine phosphate ores varies from area to area depending on the type of deposit. In Florida phosphate ore is mined using the strip-mining technique, which involves stripping the overburden and mining the matrix with draglines creating mine cuts from 15 to 20 m in depth [R5]. Overburden is stored near mine cuts and used as backfill as subsequent cuts are made. Structures later built on reclaimed land may exhibit elevated radon concentrations, as discussed later in this Annex.

221. After mining, beneficiation, which is a physical separation process using screen flotation to remove non-phosphate components, is applied to phosphate ores not rich enough in P_2O_5. Exposures resulting from mining and beneficiation have not been assessed.

222. After phosphate rock has been mined and beneficiated, it is usually dried and ground to a uniform particle size to facilitate processing. Wet process plants produce phosphoric acid, the starting material for ammonium phosphate and triple superphosphate fertilizers; in that process, phosphogypsum is produced as waste or by-product. Thermal process plants produce elemental phosphorus, which is in turn used primarily for the production of high-grade phosphoric acid, phosphate-based detergents, and organic chemicals. Waste and by-products of the thermal process are slag and ferrophosphorus.

223. The drying and grinding operations that follow phosphate rock mining and beneficiation produce significant quantities of particulate material [U9]. Very limited data are available on actual field measurements of radioactive materials from drying and grinding operations. Measurements made by the United States Environmental Protection Agency (EPA) [P16] were summarized in the UNSCEAR 1982 Report [U1]. EPA has since established the parameters of a reference phosphate rock drying and grinding plant [U9], which it used to calculate the emissions of radioactive materials into the atmosphere (see Table 34); they correspond to a particulate emission rate of about 0.1 kg per tonne of phosphate rock throughput and to radioactive equilibrium of ^{238}U with its solid decay products. The atmospheric release of ^{222}Rn has not been estimated by EPA; assuming an activity mass concentration of ^{226}Ra in phosphate ore of 1,500 Bq kg^{-1} and, conservatively, a 100% release of ^{222}Rn, would yield 1.5 MBq per tonne of phosphate rock.

224. Some phosphate rock must be calcined before it can be processed. The need for calcining is determined primarily by the amount of organic material in the beneficiated rock. In the calcining operation, the rock is heated to about 800° C to remove unwanted hydrocarbons. Because calciners operate at a higher temperature than driers, they have the potential for volatilizing ^{210}Pb and ^{210}Po. Atmospheric releases from elemental phosphorus plants, which are mainly attributable to the operation of calciners, have been measured in the Netherlands and the United States [B31, U9, U10, U11, U12]. Estimated annual values are presented in Table 34. Discharges of ^{210}Pb and ^{210}Po are about two orders of magnitude higher than those of other solid radionuclides of the ^{238}U decay series. Analyses of particles collected from the calciner off-gas streams at two plants in the United States showed that most of the ^{210}Po activity was associated with submicron particles and that the dissolution rate of both ^{210}Pb and ^{210}Po in simulated lung fluid was very small [U10, U11].

225. Results of measurements at calciners at wet-process phosphoric acid plants are not yet available [U9]. Atmospheric discharges of natural radionuclides at two wet-process phosphoric acid plants not equipped with calciners, however, have been published by EPA [P5] and summarized in the UNSCEAR 1982 Report [U1]. EPA has since defined the parameters of a reference wet-process phosphate fertilizer plant that are used to estimate the annual discharges of natural radionuclides into the atmosphere (Table 34). Those discharges result from the production processes of phosphate fertilizers and apparently do not include the discharges due to ore drying and grinding. Releases of ^{226}Ra are estimated to be lower than those of ^{238}U and ^{230}Th because radium is chemically separated from uranium and thorium in the digestion

of phosphate rock and goes predominantly to phosphogypsum.

226. Annual airborne discharges, normalized to the production or processing of one tonne of phosphate rock, are presented in Table 35. The normalized discharges apply mainly to the operation of phosphate industrial plants in the United States and may not be representative of the world-wide situation. Estimates of collective dose commitments corresponding to the normalized discharges have been calculated using the methods described in the section on coal-fired power plants and are also presented in Table 35.

227. Rough estimates of the collective dose commitments resulting from the world-wide operation of phosphate industrial facilities, using the experience of the United States as a guide, are presented in Table 36. The corresponding collective effective dose equivalent commitment is about 60 man Sv per year of practice.

228. Environmental concentrations of natural radionuclides and assessments of maximum individual doses have been published for a few plants. At a distance of about 2 km from an elemental phosphorus plant in the Netherlands, the activity concentrations of ^{210}Pb and ^{210}Po in surface air have been found to be about 0.1 and 0.4 mBq m^{-3} above background, respectively [D7]. For the same plant, the estimated maximum annual individual effective dose equivalents due to atmospheric discharges are about 40 μSv [B31, S50]. In the United States, the atmospheric releases from the six elemental phosphorus plants in operation in 1983 were estimated to result in annual lung dose equivalents for nearby individuals ranging from 0.05 to 6 mSv [U9].

229. Discharges of radioactive materials into surface water have also been occasionally reported. In the Netherlands, about 50 GBq of ^{210}Pb and 30 GBq of ^{210}Po were discharged from an elemental phosphorus plant into the Scheldt estuary [B31]. In that country, all phosphogypsum produced by fertilizer plants (2 million tonnes per year) is also disposed of into the Rhine 15 km from the North Sea [K19]; these annual discharges, which contain about 0.4 TBq of ^{238}U, 2 TBq of ^{226}Ra, 0.7 TBq of ^{210}Pb and 2 TBq of ^{210}Po, are estimated, in a preliminary assessment, to result in maximum individual effective dose equivalents of 150 μSv per year and annual collective effective dose equivalents to the Dutch population of 170 man Sv from ingestion of seafood; the main contributor to the dose is ^{210}Po [K19]. In France, over 3 million tonnes of phosphogypsum was dumped into the Seine estuary [P18] but the corresponding radiation exposures have not been estimated.

230. In other countries, such as India and the United States, phosphogypsum is retained in sludge ponds or stockpiles near phosphate fertilizer plants; occasional drainage or seepage of radioactive materials into nearby surface water has been reported [A14, P17].

231. Limited information is available on occupational exposures attributable to processing and transport of rock phosphates. In Dutch wet-process phosphoric acid plants, higher than average external radiation doses in air, ranging from 2 to 100 μGy h^{-1}, have been detected in the vicinity of some piping and vessels near gypsum filters [L15]. Analysis of scaling samples revealed activity mass concentrations of ^{226}Ra of up to 0.4 MBq kg^{-1}; the potential radiation doses, attributable to external exposure and to inhalation of ^{226}Ra during periodic cleaning, have been estimated in eight phosphoric acid plants to be less than 2.2 mSv per year [L15]. In the Federal Republic of Germany, Pfister and Pauly [P15] estimated individual and collective external exposures for the small group of persons who handle rock phosphates as part of their job; their results correspond, in terms of effective dose equivalent, to individual exposures of 200 μSv a^{-1} and collective exposures of 0.5 man Sv a^{-1}. Extrapolating to the world's population by scaling over the phosphate fertilizer consumption results in an annual collective effective dose equivalent of approximately 20 man Sv. Exposures attributable to inhalation of phosphate rock, however, have not been taken into account.

2. Use of phosphate fertilizers

232. The world consumption of phosphate fertilizers was about 30 million tonnes of P_2O_5 in 1982 [F9]. The application rate of fertilizers depends on, among other things, the type of soil and the type of crop. The average consumption of phosphate fertilizer per unit area of agricultural land varied in 1982 from 3.6 kg P_2O_5 per hectare in the developing countries to 10.9 kg P_2O_5 per hectare in the developed countries, the world average being 6.7 kg P_2O_5 per hectare [F9].

233. Activity mass concentrations of natural radionuclides in phosphate fertilizers have been reviewed in the UNSCEAR 1982 Report [U1]. For a given radionuclide and type of fertilizer, the activity mass concentrations vary markedly from one country to another, depending on the origin of the components. General features are that the activity mass concentrations of ^{40}K and of ^{232}Th and its decay products are always low and that the activity mass concentrations of the radionuclides of the ^{238}U decay series are 5-50 times higher than in normal soil. The degree of radioactive equilibrium between ^{238}U and its decay products in a given type of fertilizer depends essentially on the relative contribution of phosphoric acid, since phosphoric acid usually has a very low ^{226}Ra concentration. For the purposes of this Annex, it is assumed that ^{230}Th and ^{234}U are in radioactive equilibrium with ^{238}U and that ^{210}Pb and ^{210}Po are in radioactive equilibrium with ^{226}Ra. Table 37 presents average activity mass concentrations of ^{238}U and ^{226}Ra in phosphate fertilizer from four countries [D8, M21, P15, U13]; the levels range from 1,700 to 9,200 Bq per kg P_2O_5 for ^{238}U and from 480 to 1,700 Bq per kg P_2O_5 for ^{226}Ra. Typical values are 4,000 and 1,000 Bq per kg P_2O_5 for ^{238}U and ^{226}Ra, respectively.

234. Since the activity mass concentrations of ^{238}U and ^{226}Ra in phosphate fertilizers are several times higher than in normal soil, they constitute an additional

source of radiation exposure for workers and members of the public.

235. The annual application of phosphate fertilizers can be calculated to represent less than 1% of the normal soil content of ^{238}U [M21, P15]. Assuming an accumulation of ^{226}Ra in the soil during the past 80 years, Pfister and Pauly [P15] have estimated that the mean additional absorbed dose in air above fertilized fields is about 0.8 nGy h^{-1}, a small fraction of the normal natural background from terrestrial sources of 50 nGy h^{-1}. Small additional doses also occur from ingestion of foodstuffs grown on fertilized agricultural areas; Drichko et al. [D8] have shown that although the transfer coefficient of ^{226}Ra from soil to plants is practically the same whether ^{226}Ra is included in fertilizer or soil structures, the activity mass concentrations of ^{210}Pb and ^{210}Po in the aerial parts of plants seem to be independent of the corresponding activity mass concentrations in soil; this implies the predominance of the air-to-vegetation transfer for such radionuclides.

236. The collective dose commitments incurred after application of phosphate fertilizers have been roughly evaluated by comparison with the activity mass concentrations of natural radionuclides in soil and the corresponding dose rates. Table 38 shows the results obtained under the following assumptions: the availability to plants of natural radionuclides is the same whether in fertilizer or in the normal constituents of the soil; the ploughed layer of soil is 0.3 m deep; the deposited activity becomes unavailable to the vegetation with a mean life of 100 years for the long-lived natural radionuclides; the fraction of time spent by the populations exposed on or near fertilized fields is 1%; and one tonne of phosphate rock produces, on average, 0.3 tonne of P_2O_5 in phosphate fertilizer. It can be inferred from Table 38 that the collective effective dose equivalent commitment per unit of phosphate rock is approximately 8 10^{-5} man Sv t^{-1}. Taking the world-wide production of phosphate rock to be 120 million tonnes, the collective effective dose equivalent commitment resulting from the world-wide use of phosphate fertilizers during one year is roughly estimated to be 10,000 man Sv.

237. Occupational exposures arising from external irradiation during transport, storage and application of phosphate fertilizer were assessed by Pfister and Pauly [P15] and reviewed in the UNSCEAR 1982 Report [U1]. Mean additional absorbed dose rates in air ranging from 20 to 200 nGy h^{-1} were measured for various transport and loading operations, with peak values of 800 nGy h^{-1} [P15]. From the number of workers involved or the time spent in the various operations considered, a total annual collective effective dose equivalent of 1.3 man Sv can be derived [P15]. A rough estimate of about 50 man Sv for the collective effective dose arising from occupational exposure to external irradiation resulting from world-wide handling of phosphate fertilizers can be inferred from the results of Pfister and Pauly, using the consumption of phosphate fertilizers as a reference scale.

3. Use of by-products and wastes

238. The main by-products resulting from phosphate industrial activities are phosphogypsum in wet-process fertilizer plants and calcium silicate slag in thermal process plants. Significant radiation exposures may occur if such by-products are used in the building industry. Another source of exposure stems from the reclamation of land on which phosphate mining has been completed and houses have been built. Since new information on radiation exposures has not been made available to the Committee in the past few years, the following is, to a large extent, a summary of the corresponding section in the UNSCEAR 1982 Report [U1].

239. Large quantities of phosphogypsum (about 100 million tonnes per year) are produced in wet-process phosphoric acid plants. The activity mass concentrations of ^{226}Ra in phosphogypsum, which depends on the origin of the phosphate ore processed, is typically about 900 Bq kg^{-1} [U1, U2]. Most of the phosphogypsum is considered waste and is either stored in ponds or stacks or discharged into the aquatic environment.

240. In the United States, a small amount of phosphogypsum (about 0.6 million tonnes per year) is used in agriculture to improve water movement in saline-alkaline soil and as a substitute for lime or limestone in alkaline soil [L16, P18].

241. Phosphogypsum is also used as a substitute for natural gypsum in the manufacture of cement, wallboard and plaster. Japan phased out its production of natural gypsum in 1976 and is currently recycling about 3 million tonnes of phosphogypsum per year [P18]. In Japan, approximately one half of the by-product gypsum is used as cement set-retardant; the other half is used to make wallboard and plaster. In Western Europe, about 10% of the phosphogypsum production, that is 2.5 million tonnes, was used in 1981 as by-product [P18]. Phosphogypsum is commonly used in construction blocks for floors and walls and in partition blocks for interiors in north Africa, the Mediterranean countries and the Middle East. It will be assumed in this Annex that 5 million tonnes of phosphogypsum, representing 5% of the world-wide annual production, is used annually as building material in dwellings.

242. O'Riordan et al. [O8] estimated the additional doses that would be received by the occupants of a residential building in which 4.2 tonnes of by-product gypsum would have replaced the established materials; the additional absorbed dose rate in air was calculated to be 0.07 μGy h^{-1}; the additional radon concentration, using an air exchange rate of 1 h^{-1}, was estimated to be about 10 Bq m^{-3}, resulting in an annual effective dose equivalent of 0.6 mSv. If it is assumed that 5% of the by-product gypsum is used as building material in dwellings, on average four persons live in the dwellings and the mean life of the dwellings is 50 years, the collective effective dose equivalent commitments resulting from one year of world-wide use of phosphogypsum in the building industry are

estimated to be 10^5 man Sv from external irradiation and $2\ 10^5$ man Sv from internal irradiation. These estimates are highly uncertain and need to be confirmed by measurements in dwellings that have been constructed using known amounts of phosphogypsum.

243. Calcium silicate slag may be used as a component for concrete. Measured activity concentrations in slag samples range from 1,300 to 2,200 Bq kg^{-1} of ^{226}Ra [B11, M16]. Results from an indoor survey indicate that the gamma absorbed dose rate in air can be as high as 0.3 μGy h^{-1} above background in dwellings constructed with 43% by weight slag in concrete slabs [B11]. In a similar survey carried out in Canada, absorbed dose rates of up to 0.2 μGy h^{-1} were obtained [M16].

244. Residents in structures built on reclaimed phosphate land in Florida, United States may also be exposed to significant radiation doses. About 600 km^2 of Florida land has been mined for phosphate rock in the past 80 years. About 200 km^2 of the land has been reclaimed to various degrees. The equilibrium equivalent radon concentrations in houses built on reclaimed land were found to range from 4 to 500 Bq m^{-3} with a weighted average of about 40 Bq m^{-3}, which is estimated to be 26 Bq m^{-3} above normal concentrations in houses in that area [G21]. This average additional concentration results in an annual effective dose equivalent of approximately 1.6 mSv. An experimental investigation and remedial action programme was carried out involving 10 houses and a mobile home on land that had phosphate mineralization or had been affected by phosphate mining activities and for which the annual average radon equilibrium equivalent concentration was believed to be in excess of 100 Bq m^{-3} [S51].

4. Summary

245. Table 39 summarizes the radiation exposures attributable to the use of phosphate rock, expressed in terms of collective effective dose equivalent commitment resulting from one year of practice. The total collective effective dose equivalent commitment is estimated to be 300,000 man Sv, essentially from the use of by-product gypsum in dwellings.

D. NON-URANIUM ORE MINING AND PROCESSING

246. Because of the presence of radon in confined spaces, metal and non-metal mining results in occupational exposures. Ventilation of mine air, in turn, results in environmental discharges of radon and exposures of members of the public. Ore processing gives rise to additional environmental discharges of natural radionuclides. Occupational exposures and exposures of members of the public are discussed below.

1. Occupational exposures from non-uranium mining

247. Investigations of radon and radon decay product concentrations in non-uranium mines have been conducted since the 1960s. This section is devoted to the discussion of occupational exposures in non-uranium mines other than coal mines, which are considered in another part of this Annex.

248. Measured concentrations of radon and radon decay products are presented in Table 40. The potential alpha energy concentrations of radon decay products range from 0.02 to more than 300 μJ m^{-3}, and there is no simple relationship between radon decay product concentration and type of mine. Mines extracting from sedimentary deposits usually have low radon decay product concentrations but this is not always true. Mines associated with igneous intrusions imply high uranium content and therefore high radon decay product concentrations, although ventilation can obscure this relationship. Geological and hydrogeological characteristics may also be of importance. Convective movements of air and water resulting from pressure differences between the mine and its environment explain large radon entry rates into the mine [Z2]. Detailed studies of the characteristics of non-uranium mines in countries of the European Economic Community show that the primary indicator of high radiation exposures is a radon concentration in exhaust air in excess of 400 Bq m^{-3} [Z2].

249. Occupational exposures attributable to radiation in non-uranium mines have decreased with time [D6, S34, S35], as monitoring has led to action being taken to relieve high exposure situations. Personal dosimetry is now being used in Polish non-uranium mines [C15] and is envisaged in Italy [S37] and the United Kingdom [M18].

250. The degree of equilibrium between radon decay products and radon in non-uranium mines has been investigated in several countries. Measurements in an Italian lead mine at a location representative of the radiological characteristics of the mine show an average value of 0.70 with a range of 0.50-0.85 [S37]. In 15 mines in the Federal Republic of Germany, the equilibrium factor was found to vary from 0.28 to 0.82 with a rather flat distribution [K15]. In Norway, the mean value of 210 measurements of the equilibrium factor in different mines was 0.46, with a minimum of 0.08 and a maximum of 0.93 [S38]. A typical value of the equilibrium factor in non-uranium mines seems to be 0.6 with large variations around this figure.

251. Stranden and Berteig [B20, S38] have established empirical relationships between the equilibrium factor F and the activity concentrations χ of the individual radon decay products ^{218}Po, ^{214}Pb and ^{214}Bi normalized to radon that seem to apply to most mining conditions when the value of F is in the range of 0.1-0.7:

$$\chi(^{218}Po) / \chi(^{222}Rn) = 1.1\ F^{0.55}$$

$$\chi(^{214}Pb) / \chi(^{222}Rn) = 1.0\ F^{0.94}$$

$$\chi(^{214}Bi) / \chi(^{222}Rn) = 0.77\ F^{1.12}$$

252. The unattached fraction f_p of the potential alpha energy of radon decay products in non-uranium mines was also studied by Berteig and Stranden [B20, S38]; in most working conditions, the value of f_p is usually found to be smaller than 0.05. The main factor

influencing the unattached fraction seems to be the aerosol particle concentration; high values of the unattached fraction are observed with low aerosol concentrations [G23, S38]. Suggested empirical relationships between the value of f_p and that of the unattached fraction of individual radon decay products are as follows [S38]:

$$f(^{218}Po) = 2.6\, f_p^{1.1}$$

$$f(^{214}Pb) = 0.77\, f_p^{0.94}$$

$$f(^{214}Bi) = 0.27\, f_p^{0.85}$$

253. Radiation measurements other than those of radon and their decay products are limited. A study of the complete spectrum of radiation exposures was carried out in eight British non-uranium mines [D5]. Table 41 presents the reported concentrations and an estimate of the resulting effective dose equivalents. In mines associated with igneous intrusions, the calculated doses are almost entirely due to the inhalation of radon decay products. In sedimentary mines, the contribution to the normal dose from the inhalation of radon decay products is always greater than 50%, even for low concentrations of radon daughters. Similar results have been observed for mines in the Federal Republic of Germany [K15]. The effective dose equivalent from inhaled thoron decay products, however, may be higher than that from inhaled radon decay products if the ratio of the activity mass concentration of ^{232}Th to that of ^{238}U in the mined rock is substantially greater than one, which is sometimes the case [S39, S40].

254. On the basis of the results presented in Table 40, annual effective dose equivalents received by non-uranium miners in the course of their work are estimated to range from 0.1 mSv to about 0.2 Sv, most of the values being between 1 and 50 mSv. If the arithmetic mean of the annual effective dose equivalent is tentatively assumed to be 10 mSv and the fraction of non-uranium miners in the world population is taken to be $5\ 10^{-4}$ [U14], a very rough estimate of the annual collective effective dose equivalent would be $2\ 10^4$ man Sv.

2. Occupational exposures from factory processes

255. Industrial processes are carried out for many purposes that use the materials extracted by non-uranium mining. In these factory processes, there are exposures to natural radioactivity but in most cases data on measured activities or estimates of doses have not been reported in the literature. Examples given in the following sections are the industrial use of zircon sand and the processing of ilmenite; another example not discussed separately in this Annex is the use of thorium in coating optical lenses. Information in this field is extremely limited and no reliable assessment of the collective exposures can be made.

(a) Processing of raw ilmenite

256. The activity concentrations of natural radionuclides in samples collected at an Australian titanium dioxide plant have been reported [C13]. In that plant, the white pigment titanium dioxide is produced from raw ilmenite (a dark-coloured, fine-grained beach sand) containing monazite as an impurity (0.14% by mass). The activity concentrations in ilmenite feed are about 400 Bq kg^{-1} of ^{228}Ra and ^{228}Th and 75 Bq kg^{-1} of ^{226}Ra. These radionuclides have been followed throughout the pigment production process. It was established that in the presence of high sulphate ion concentrations in both the plant feed liquor and the effluent, thorium remains in solution whereas radium, which forms an insoluble sulphate compound, tends to co-precipitate and is found in solid residues throughout the process. Very high activity concentrations of $4\ 10^5$ Bq kg^{-1} of ^{226}Ra and $1.5\ 10^6$ Bq kg^{-1} of ^{228}Th were measured in a sample of scale scraped from a heat exchanger unit. The mass balance study of the activity entering and leaving the plant shows, however, that essentially all of the activity entering the plant (14 MBq d^{-1} of ^{226}Ra and 67 MBq d^{-1} of ^{228}Ra and of ^{228}Th) is found in the acidic liquid effluent stream, which is pumped to disposal lagoons and is allowed to neutralize and drain away into an estuary. Effluent concentrations are about 6 Bq l^{-1} of ^{228}Ra and ^{228}Th and 1.4 Bq l^{-1} of ^{226}Ra, whereas in the estuary they are approximately 0.01 Bq l^{-1} for ^{226}Ra, 0.02 Bq l^{-1} for ^{228}Th and 0.10 Bq l^{-1} for ^{228}Ra. In contrast to the situation found in the plant and the effluent stream, radium is present in the estuary in a soluble form and thorium is not, leading to a possible accumulation of ^{232}Th and of its decay products in the estuary. The radiation doses attributable to the operation of the plant have not been assessed.

(b) Industrial use of zircon sand

257. Zircon sand occurs in river and beach placers, usually with monazite. About 700,000 tonnes of zirconium minerals are produced each year, mainly in Australia and South Africa [B37]. The activity mass concentrations of ^{238}U and ^{232}Th in zircon sand are in general well above 500 Bq kg^{-1} [B36, D13, N18]. Zircon sand is used in the zirconium extraction industry, foundry sands, refractories and ceramics. Use of the sand by foundries and the zirconium industry has resulted in the accumulation of large volumes of low-level radioactive waste.

258. Little information is available on the radiation protection aspects of the use of zircon sand [B36, N18]. It seems clear, however, that inhalation of radon decay products is not a problem because the exhalation rate of radon from the zircon sand is low. Doses from external irradiation may be significant only in storage areas. Absorbed dose rates in air of about 700 nGy h^{-1} have been measured at a distance of about 1 m from a stack of bags of zircon sand [B36]. In an Italian factory producing electro-fused refractory blocks, the absorbed dose rates in air were observed to be near background in all representative work places but reached about 500 nGy h^{-1} on top of the sand storage tanks [N18].

259. The inhalation pathway, also assessed in the Italian factory, seems to be more important. The airborne concentrations of natural radionuclides near areas of high temperature processes were estimated

to be about 200 mBq m^{-3} of ^{210}Po (typical background: 0.04 mBq m^{-3}) and 20 mBq m^{-3} for very long-lived natural radionuclides (typical background: 0.001 mBq m^{-3}). The particle size distribution was bimodal with a mode at approximately 0.3 μm and another between 5 and 10 μm. The resulting annual effective dose equivalent, assuming constant occupancy in the most highly contaminated areas, would be of the order of 5 mSv [N18].

260. No information on environmental discharges was made available to the Committee. Aquatic discharges would in most cases lead only to insignificant doses, as zircon compounds are extremely insoluble in water.

3. Exposures of members of the public

261. All industrial operations involving ore mining and processing release natural radionuclides into the environment. The results of radiation surveys conducted in several industrial facilities are given below.

(a) Zinc mining and processing

262. Zinc is usually found in nature as a sulphide ore called sphalerite. The ore, which usually contains impurities of lead, cadmium and traces of other elements, are processed at the mill to form concentrates typically containing 62% zinc and 32% sulphur. These concentrates are processed at the smelter to recover zinc metal [U8].

263. Atmospheric emissions of naturally occurring radionuclides were measured from an underground mine and mill [A7] and estimated from a reference zinc smelter [U8]; the results are shown in Table 42. The releases from the smelter cannot be compared to those of the mine and mill, as they are different facilities and the capacity of the mine and mill was not indicated. It is clear, however, that the only significant emission is that of radon from the mine (8 TBq per year). A radiation survey was conducted in that particular zinc mine because a relatively high radon concentration of 2,600 Bq m^{-3} had been measured at the mine portal exhaust. Concentrations of ^{238}U and ^{232}Th, in the ore as well as in the concentrate, were found to be significantly below the corresponding average concentrations in soil; the relatively high radon concentrations observed in the mine were attributed to the large quantity of water that flows into the mine. The collective effective dose equivalent commitment resulting from inhalation of radon decay products released in one year was estimated to be about 0.1 man Sv. The very small releases of other natural radionuclides due to low activity mass concentrations and to efficient particle control do not result in significant collective effective dose equivalent commitments.

(b) Fireclay mine and refractory plant

264. Clay mining and manufacturing is a large industry. Clay may be used to manufacture bricks and refractory materials. A clay mine and refractory plant was surveyed in the United States to estimate the quantities of naturally occurring radionuclides emitted into the atmosphere during mining and manufacturing processes [A6]. Ore samples collected from the mine contained average ^{238}U and ^{232}Th concentrations of about 50 and 70 Bq kg^{-1}, respectively. Equilibrium equivalent concentrations of radon of approximately 1 kBq m^{-3} were measured in the clay mine at the working face; the corresponding annual emission of radon is about 1 TBq. Particulate releases consist mainly of ^{210}Po, which is partially vaporized at the kiln's operating temperature of 1,100° C. Annual atmospheric emissions are summarized in Table 43. Estimated collective effective dose equivalent commitments resulting from one year of operation of the fireclay mine and refractory plant are 0.02 man Sv from inhalation of radon decay products and less than 0.01 man Sv from inhalation and ingestion of ^{210}Po.

(c) Aluminium ore processing

265. Bauxite is the principal aluminium ore found in nature. The ore is processed at mines to produce alumina (Al_2O_3), the basic feed in the aluminium reduction process. Aluminium metal is produced by the reduction of alumina in a molten bath of cryolite [U8].

266. Radionuclide measurements at both an alumina plant and an aluminium reduction plant have been carried out in the United States [A9, U8]. The ^{232}Th activities per unit mass measured in the process samples of the alumina plant are listed in Table 44. The levels in bauxite ore, red mud and brown mud are about 10 times higher than the average activities per unit mass in normal soil, whereas the radioactive content of alumina kiln feed and alumina product is less than that of normal soil.

267. Table 45 shows the estimated annual atmospheric emissions from the surveyed alumina plant and aluminium reduction plant. Again, radon emissions are much higher than those of other natural radionuclides. Among the particulates, ^{210}Pb and ^{210}Po are the most important contributors to the environmental releases, as the high temperatures of the kilns and of the reduction cells (above 950° C) cause these radionuclides to be volatilized [A9, U8].

268. The corresponding collective effective dose equivalent commitments are estimated to be about 0.1 man Sv; they are almost entirely due to the releases of ^{210}Po and ^{210}Pb.

(d) Copper ore mining and processing

269. Copper ores are milled to produce a concentrate containing copper, sulphur, iron and some insoluble material (primarily silica and aluminium). This concentrate is the basic feed to the copper smelter that eventually produces the refined copper product. Prior to smelting, part or all of the concentrates may receive a partial roast to eliminate some of the sulphur and other impurities [U8].

270. Radionuclide measurement studies at an underground copper mine and mill, an open pit mine and mill and two copper smelters have been carried out in

the United States [A9, U8]. The activity mass concentrations of ^{238}U and ^{232}Th in process samples from the mines and mills (Table 46) were found to be at or slightly above the corresponding levels in normal soil. Most of the ^{232}Th content, however, seems to be removed from the ore in the milling process.

271. Estimated annual atmospheric releases are presented in Table 47. Emissions from the copper smelter cannot be directly compared with those from the mine and from the mill, the capacity of which were not indicated. Calculated collective effective dose equivalent commitments from one year of releases are about 1 man Sv, with nearly equal contributions from thorium isotopes and ^{210}Pb-^{210}Po.

(e) Lead ore processing

272. Galena (PbS), frequently containing cerussite ($PbCO_3$) and anglesite ($PbSO_4$), is the principal lead-bearing ore found in nature. Galena contains small amounts of copper, iron, zinc and other trace elements. Lead smelting involves three distinct processes: sintering, to convert the ore from a sulphide to an oxide or sulphate form and prepare the feed materials for furnacing; furnacing, to reduce the oxide feed to lead metal; and drossing, to reduce the copper content of the lead bullion from the furnace. Off-gases from the sintering machine and the blast furnace are the most significant sources of particulate emissions from the lead smelting process [U8].

273. As the activities of ^{238}U and ^{232}Th per unit mass of lead ore are similar to the corresponding values in normal soil, the atmospheric emissions of natural radionuclides estimated for a reference plant processing annually 0.22 million tonnes of lead [U9] are fairly small (about 1 GBq of ^{210}Pb and of ^{210}Po). The resulting collective effective dose equivalent commitments per year of release from the reference plant are less than 0.1 man Sv.

(f) Other metal and non-metal mining

274. Annual radon releases into the atmosphere have been reported for a few other United States mines: 30 GBq for a limestone mine, 70 GBq for a fluorspar mine and 2,600 GBq for an iron mine [S32]. The resulting collective effective dose equivalent commitments from inhalation of radon decay products are less than 0.05 man Sv.

IV. SUMMARY

275. Estimates of per caput annual effective dose equivalents from external irradiation due to cosmic rays and terrestrial sources and from internal irradiation due to the most important naturally occurring radionuclides are presented in Table 48, together with the subjectively estimated typical ranges of variation for all the sources taken into consideration. By far the most important source of exposure is indoor radon, which leads to a per caput effective dose equivalent of 1,100 μSv and to a wide range of variation. The overall per caput annual effective dose equivalent is estimated to be about 2,400 μSv, corresponding to an annual collective effective dose equivalent of approximately 10^7 man Sv. The current estimate of the overall per caput annual effective dose equivalent is 400 μSv higher than the estimate provided in the UNSCEAR 1982 Report. This is mainly the result of a re-evaluation of the mean indoor radon concentration. The wide distribution of individual effective dose equivalents is dominated by the variability of the indoor radon concentrations, which span several orders of magnitude.

276. Individual exposures resulting from industrial activities are generally small in comparison to the overall exposure from natural sources of radiation. Their importance can best be characterized in terms of collective effective dose equivalent commitments. Table 49 provides estimates of collective effective dose equivalent commitments arising from one year of practice for most of the activities considered. Because of the scarcity, or lack, of data for the industrial activities listed in the table, most of the estimates are very tentative. The total collective effective dose equivalent commitment arising from one year of practice is less than 5 10^5 man Sv, representing a per caput effective dose equivalent commitment of about 100 μSv.

Table 1

Estimated per caput annual effective dose equivalent from natural sources in areas of normal background

(Estimates from the UNSCEAR 1982 Report are given in parentheses.)

Source of irradiation	Annual effective dose equivalent (μSv)		
	External irradiation	Internal irradiation	Total
Cosmic rays			
Ionizing component	300 (280)		300 (280)
Neutron component	55 (21)		55 (21)
Cosmogenic radionuclides		15 (15)	15 (15)
Primordial radionuclides			
K-40	150 (120)	180 (180)	330 (300)
Rb-87		6 (6)	6 (6)
U-238 series:			
U-238 → U-234		5 (10)	
Th-230		7 (7)	
Ra-226	100 (90)	7 (7)	1300 (1040)
Rn-222 → Po-214		1100 (800)	
Pb-210 → Po-210		120 (130)	
Th-232 series:			
Th-232		3 (3)	
Ra-228 → Ra-224	160 (140)	13 (13)	340 (330)
Rn-220 → Tl-208		160 (170)	
Total (rounded)	800 (650)	1600 (1340)	2400 (2000)

Table 2

Absorbed dose rates in air derived from exposure rate measurements in the centre of a 12-storey building [M9]

Level	Dose rate (nGy h^{-1})	Transmission factor
Roof	31.4	1
12	20.2	0.64
10	20.0	0.64
8	18.1	0.58
5	17.4	0.55
4	13.7	0.44
2	11.5	0.37
Basement	8.6	0.27

Table 3

Average activity mass concentrations
of potassium-40, uranium-238 and thorium-232
in soil and absorbed dose rate in air 1 m above the ground surface
[832]

Radionuclide or decay series	Dose rate per unit activity mass concentration in soil (nGy h^{-1} per Bq kg^{-1}) (wet weight)	Average activity mass concentration in soil a/ (Bq kg^{-1}) (wet weight)	Absorbed dose rate in air a/ (nGy h^{-1})
K-40	0.043	370 (100-700)	16 (4-30)
U-238	0.427	25 (10- 50)	11 (4-21)
Th-232	0.662	25 (7- 50)	17 (5-33)

a/ The typical range is given in parentheses.

Radionuclide or decay series	Dose rate per unit specific concentration in soil (nGy h^{-1} per Bq kg^{-1}) (wet weight)	Average specific concentration in soil a/ (Bq kg^{-1}) (wet weight)	Absorbed dose rate in air a/ (nGy h^{-1})
K-40	0.043	370 (100-700)	16 (4-30)
U-238	0.427	25 (10- 50)	11 (4-21)
Th-232	0.662	25 (7- 50)	17 (5-33)

a/ The typical range is given in parentheses.

Table 4

Estimates of outdoor absorbed dose rate in air from terrestrial radiation sources 1 m above ground level obtained in large-scale surveys

Country or area	Year reported	Number of measurements	Absorbed dose rate in air (nGy h^{-1})		Type of survey and instrumentation used	Ref.
			Average	Range		
Austria	1980	> 1000	43	20-150	Ground survey in populated areas with Geiger counter	[T9]
Belgium	1987	272	43	13-58	Ground survey with thermoluminescent dosimeters, gamma spectrometer and ionization chamber	[D15]
Canada	1984	33 areas	24	18-44	Aerial survey with a scintillation detector	[G28]
China	1986	38661	80	60-120 a/	Ground survey with ionization chambers and scintillation detectors	[W21]
China (Taiwan Province)	1972	26	69	-	Analysis of soil samples using gamma spectrometry	[W18]
Denmark	1980	14 sites	38	17- 52	Ground survey with ionization chamber and gamma spectrometer	[N16]
Finland	1980	-	65	-	-	[L17, R7]
France	1985	5142	68	10-250	Ground survey with thermoluminescent dosimeters (preliminary results)	[M25, R6]
German Democratic Republic	1969	1005	85 b/	24-270	Ground survey with ionization chamber	[O13]
Germany, Federal Republic of	1978	24739	53	4-350	Ground survey with scintillation detectors	[D9]
Hungary	1987	123 sites	55	20-130	Ground survey with thermoluminescent dosimeters	[N21]
Iceland	1982	-	28	11- 83	-	[E8,R7]
India	1986	2800	55	20-1100 c/	Ground survey with thermoluminescent dosimeters	[N19]
Ireland	1980	264	42	0-180	Ground survey with ionization chamber	[M26]
Italy	1972	1365	57	7-500	Ground survey with ionization chamber	[C17]
Japan	1980	1127	49	5-100	Ground survey with ionization chamber and scintillation detectors	[A15]
Netherlands	1985	1049	32	10- 60	Ground survey with ionization chamber	[V2]
Norway	1977	234	73	20-1200	Ground survey with ionization chamber placed in a car	[S53]
Poland	1980	352 sites	37	15- 90	Ground survey with thermoluminescent dosimeters	[N17]
Romania	1979	2372	81	32-210	Analysis of soil samples using gamma spectrometry	[T10]
Sweden	1979	-	80	18-4000	-	[J18, R7]
Switzerland	1964	-	60 d/	-	Ground survey with ionization chamber	[H19]
United Kingdom	1984	1400	40	0-100	Ground survey with energy-compensated Geiger counters (preliminary results)	[G30]
United States	1972	25 areas e/	46	13-100	Aerial survey with scintillation detectors	[O12]

a/ Range of the means.
b/ A contribution of 1.0 μR per hour from nuclear weapons fallout was subtracted from the total exposure rate.
c/ Range of the arithmetic means for each province.
d/ A contribution of 3.1 μR per hour from nuclear weapons fallout was subtracted from the total exposure rate.
e/ Inhabited by approximately 30% of the country's population.

Table 5

Population-weighted means of absorbed dose rate in air 1 m above ground level obtained in a large-scale survey in China

($nGy\ h^{-1}$)

[W21]

City, province or autonomous region	Number of measurements		Indoors		Outdoors	
	Indoors	Outdoors	Gamma rays	Cosmic rays (ionizing component)	Gamma rays	Cosmic rays (ionizing component)
Beijing	623	576	87.0	28.1	59.7	31.2
Shanghai	442	227	118.7	25.2	69.8	29.7
Tianjin	308	236	117.1	27.0	73.8	30.0
Hebei	292	316	97.0	30.6	66.0	34.0
Shanxi	3454	1842	105.9	30.9	69.7	37.3
Inner Mongolia	1620	1620	106.5	34.9	70.2	38.5
Heilongjiang	2073	2650	109.1	29.6	72.8	32.9
Jilin	1273	1107	109.9	29.0	77.3	32.7
Liaoning	472	481	96.0	29.1	67.0	71.3
Shandong	2149	2149	115.7	29.3	76.1	30.8
Jiangsu	2331	808	107.0	26.7	68.9	29.7
Anhui	1549	1549	104.1	28.0	69.5	30.0
Zhejiang	1312	1312	151.4	27.3	92.3	30.0
Jiangxi	2093	896	144.3	27.0	99.0	29.4
Fujian	6694	1276	161.6	27.6	119.2	29.7
Henan	4077	2236	118.1	27.5	77.2	30.2
Hubei	1462	1439	112.7	27.6	73.0	30.4
Hunan	1677	1534	130.4	28.5	89.1	30.2
Guangdong	1712	1712	158.2	24.7	104.2	29.1
Guangxi	1555	1555	133.6	27.8	93.0	29.8
Shaanxi	894	901	108.3	31.4	67.0	34.7
Ningxia	992	230	121.6	39.2	76.3	45.6
Gansu	850	853	118.6	51.0	81.1	55.9
Qinghai	230	257	113.9	67.7	78.2	76.9
Xinjiang	3250	2510	124.1	36.5	81.9	41.0
Sichuan	6846	4707	116.5	29.7	79.3	32.9
Guizhou	698	682	112.5	36.7	91.9	39.5
Yunnan	2198	2126	120.0	44.5	87.3	46.8
Tibet	803	759	126.4	106.1	94.1	119.0
Total for China	53952	38611	119.5	30.0	80.3	33.0

Table 6

Results of large-scale surveys of indoor absorbed dose rate in air due to gamma terrestrial radiation

Country	Year reported	Number of dwellings	Type of building	Absorbed dose rate in air (nGy h^{-1})		Comments	Ref.
				Average	Population-weighted a/		
Austria	1980	1900	Brick	110			[T9]
			Concrete	81	71		
			Wood	75			
			Natural stone	110			
China	1986	53952	Various types		120	Range of the means: 90-160 nGy h^{-1}	[W21]
Denmark	1985	82	Brick	60 b/		Pilot study	[S52]
			Concrete	50	60		
			Wood	30			
Finland	1983	-	-	80	(80)	Estimated	[R7]
France	1980	946	Various types	88 b/	(88)		[M22]
	1985	5798	Various types	75	(75)	The study covered 43% of the country and 50% of the population	[R6]
German Democratic Republic	1969	667	Various types		74		[O13]
Germany, Federal Republic of	1978	29996	Solid	70			[O9]
			Frame	71	70		
			Prefabricated	40			
			Wood	45			
Hungary	1987	123	Various types	84	(84)	Range 10-200 nGy h^{-1}	[N21]
Iceland	1982	-	Concrete	23	(23)	Range 14- 23 nGy h^{-1}	[E7,R7]
Ireland	1985	223	Various types	62 b/	(62)	Range 10-140 nGy h^{-1}	[M23]
Japan	1984	135	Various types		50	Calculated from outdoor value of 49 nGy h^{-1} [A15] and indoor/outdoor ratio of 1.02 [A18]	[A15, A18]
Italy	1984	600	Various types	~60 b/	~60	Range 20-110 nGy h^{-1} Pilot study	[B33]
Netherlands	1955	399	Various types	64	(64)	Range 30-100 nGy h^{-1}	[J17]
Norway	1965	2026	Brick	120			[S53]
	1977		Concrete	105	95		[S54]
Poland	1984	1351	Coal by-product prefabricate	77-120		The study covered about 10% of the country. Outdoor doses from terrestrial radiation are higher than the country average	[K20]
			Red brick	57-100			
			Gravel-sand prefabricate	54- 68			
			Wood	42- 51			
Sweden	1980	1282	Brick	92			[R7]
	1983		Concrete	120	110		
			Aerated concrete	170			
			Wood	53			
United Kingdom	1985	2300	Various types	60	60	Living areas and bedrooms of dwellings. Standard deviation 24 nGy h^{-1}.	[W22]

a/ The numbers in parentheses are the reported averages, assumed here to be equal to the population-weighted average.

b/ The cosmic-ray contribution has been subtracted from the published value.

Table 7

Intakes of uranium-238, thorium-232, and their decay products in normal areas

Source	Annual intake (Bq)	
	Inhalation	Ingestion
U-238 series:		
U-238	0.01	5
Th-230	0.01	2
Ra-226	0.01	15
Rn-222	200000	300
Pb-210	4	40
Po-210	0.3	40
Th-232 series:		
Th-232	0.01	2
Ra-228	0.01	15
Rn-220	100000	-

Table 8

Average activity mass concentrations in organs and tissues of uranium-238, thorium-232 and their decay products [mBq kg^{-1} (fresh weight)]

Source	Gonads	Breast	Lungs	Cortical bone a/	Trabecular bone a/	Red bone marrow	Thyroid	Kidney	Liver	Other tissues
U-238 series:										
U-238	7	2	15	50	50	2	2	5	3	2
Th-230	0.3	0.3	20	20	70	0.3	0.3	10	7	0.3
Ra-226	2.7	2.7	2.7	170	170	2.7	2.7	2.7	2.7	2.7
Rn-222	-	-	100 b/	-	-	-	-	-	-	-
Pb-210	200	200	200	3000	3000	140	200	200	200	200
Po-210	200	200	100	2400	2400	110	200	200	200	200
Th-232 series:										
Th-232	0.15	0.15	20	6	24	0.15	0.15	3	2	0.15
Ra-228	0.5	0.5	15	50	50	0.5	0.5	10	5	0.5
(Th-228)										
Rn-220	-	-	40 c/	-	-	-	-	-	-	-

a/ Dry bone (5 kg dry bone yielding 2.7 kg ash per skeleton).
b/ Radon gas. The radon concentrations in tissues other than that of the lungs are negligible.
c/ Thoron gas. The thoron concentrations in tissues other than that of the lungs are negligible.

Table 9

Annual absorbed doses resulting from internal irradiation by alpha emitters from the uranium-238 and thorium-232 decay series
(μGy)

Source	Gonads	Breast	Lungs	Red bone marrow	Bone lining cells	Thyroid	Kidney	Liver	Other tissues	Effective dose equivalent (μSv)
U-238 series:										
U-238→U-234	0.32	0.09	0.69	0.17	1.2	0.09	0.23	0.14	0.09	5
Th-230	0.007	0.007	0.47	0.56	7.4	0.007	0.24	0.02	0.007	7
Ra-226 a/	0.17	0.17	0.17	0.48	5.4	0.17	0.17	0.17	0.17	7
Rn-222→Po-214			630 b/ 80 c/							850
Pb-210→Po-210	5.4	5.4	2.7	5.1	36	5.4	5.4	5.4	5.4	120
Th-232 series:										
Th-232	0.003	0.003	0.4	0.17	2.0	0.003	0.06	0.04	0.003	3
Ra-228→Ra-224	0.08	0.08	2.4	0.35	4.4	0.08	1.6	0.80	0.09	13
Rn-220→Pb-208	0.3	0.3	44	2.5	28	0.3	19	3.2	0.3	160
Total d/	31	18	970	22	51	4	32	12	22	1160

a/ Including doses resulting from the formation of Rn-222 and its short-lived decay products in the body by decay of Ra-226 (retention factor of one third).
b/ Tracheo-bronchial tree.
c/ Lungs.
d/ Contributions to the annual effective dose equivalent (μSv).

Table 10

Activity concentration of polonium-210 in ground-level air
[W13]

Country and place	Period of measurements	Number of samples	Polonium-210 concentration (μBq m^{-3})		Ref.
			Mean	Range	
Finland					
Nurmijarvi	1969	32, daily	30	10- 60	[M11]
Helsinki	1969	32, daily	50	30- 80	[M11]
France					
Toulouse	1965-1968	37, monthly	50	20-100	[M12]
Germany, Federal Rep. of					
Munich-Neuherberg	1976-1979	207, weekly	30	<7- 80	[W13]
United Kingdom					
Harwell	1955-1957	4, daily	20	4- 30	[B6]
Chilton	1962-1965	41, monthly	40	15-140	[P7]
Sutton	1962	5, monthly	30	15- 45	[O5]
United States					
Boulder	1967-1972	15, daily	35	20-120	[P8]
USSR					
Vilnius	1966-1968	?	70(?)	20-120	Quoted in [P9]
Rostov-na-Donu	1971	3, monthly	130	55-220	[L9]

Table 11

Potential alpha energy ratio of thoron and radon decay products in dwellings

Country	Number of dwellings	Potential alpha energy ratio (thoron/radon)		Ref.
		Average (mean or median)	Range	
Austria	12	0.71	0.2-2.7	[S23]
Canada	95	0.3		[G15]
Hungary	22	0.48	0.1-1	[T2]
Germany, Federal	27	0.62	0.1-2.5	[W6]
Republic of	95	0.8	0 -1.9	[U3]
	150	0.51		[K13]
Norway	22	0.48	0.1-0.7	[S24]
United Kingdom	8	0.14	0.1-1.5	[C8]
United States	68	0.62		[S67]

Table 12

Dose factors for indoor and outdoor inhalation of thoron and thoron decay products by members of the public

Organ or tissue	Thoron gas	Thoron decay products		
	Dose rate per unit concentration	Dose per unit intake	Dose rate per unit concentration	
			Indoors	Outdoors
	(μGy a^{-1} per Bq m^{-3}) (α)	(nGy Bq^{-1}) (α)	(μGy a^{-1} per Bq m^{-3}) (α)	(μGy a^{-1} per Bq m^{-3}) (α)
Lungs	0.25	14	78	25
Red bone marrow	0.02	0.8	4.5	1.4
Bone lining cells	0.18	9	50	16
Liver	0.02	1	5.6	1.8
Kidneys	0.10	6	34	11
Spleen	0.004	0.2	1.1	0.4
Other soft tissues	0.002	0.1	0.6	0.04
Effective	0.96 (μSv a^{-1} per Bq m^{-3})	50 (nSv Bq^{-1})	280 (μSv a^{-1} per Bq m^{-3})	88 (μSv a^{-1} per Bq m^{-3})

Table 13

Range and representative values of parameters for estimating the area exhalation rate of radon-222 from soils
[G2, O1, S1, W1]

Parameter	Symbol	Unit	Reported range	Representative value	Ref.
Emanating power	F_r	-	0.01-0.8	0.2	[O1,S1]
Effective bulk diffusion coefficient	Δ_{eff}	$m^2\ s^{-1}$	10^{-11}-3 10^{-6}	5 10^{-7}	[O1,S1]
Porosity	$P_{soil,ps}$	-	0.01-0.5	0.25	[O1,S1]
Exhalation rate	R	Bq m^{-2} s^{-1}	0.0002-0.07	0.016	[G2,W1]

Table 14

Dimensions and relevant parameters of the reference house

Parameter	Symbol	Value
Volume	(V)	250 m^3
Surface area of floor	(S_F)	100 m^2
Surface area of external walls and ceiling	(S_w)	300 m^2
Total surface area, including internal walls, furniture, etc.	(S)	450 m^2
Air exchange rate	(λ_v)	1 h^{-1}
Thickness of floor and ceiling	(L_F)	0.2 m (concrete)
Thickness of external walls	(L_w)	0.2 m (bricks)

Table 15

Normal content of radium-226 and radon-222 in Swedish soil, measured at 1 m depth
[A10]

Soil type	Radium-226 activity mass concentration (Bq kg^{-1})	Radon-222 concentration (kBq m^{-3})
Till, normal	15- 62	5- 30
Till, with granitic material	30- 125	10- 60
Till, with uranium-rich granite material	125- 360	10- 200
Esker gravel	30- 75	10- 150
Sand, silt	6- 70	10- 80
Soil containing alum shale	175-2500	50->1000

Table 16

Radon-222 source characteristics for building materials

Material	Country	Number of samples	Radium-226 concentration (Bq kg^{-1})	Radon-222 mass exhalation rate (μBq kg^{-1} s^{-1})	Emanating power	Diffusion length (m)	Ref.
Concrete							
Heavy concrete	USSR	18	66	3.2	0.035	0.13	[K2]
Lightweight concrete	USSR	19	141	4.1	0.021	0.26	[K2]
Ordinary concrete	Sweden	3	40-60	11-31			[P27]
Aerated concrete based on alum shale	Sweden	1	1500	580			[P27]
Alum-shale concrete	Denmark	1	-	440		0.074	[J2]
Fly-ash concrete (4%)	USA	8	19	10	0.26		[I1]
Fly-ash concrete	Greece	4	11-17	6.4-20	0.03-0.09 a/		[S5]
Concrete	Hungary	~100	13	7.8	0.28		[T1]
Concrete	Denmark	4		4.7		0.04	[J2]
Concrete	Norway	137	28		0.01-0.20	0.13	[S6]
Concrete	Greece	-	11-12	2.9-5	0.02-0.03 a/		[S5]
Concrete	USA	50	9-32	2.5-20	0.13-0.25		[I1]
Concrete	USA	1				0.13	[Z1]
Concrete	USA	1				0.17	[Z1]
(Adopted reference value)			(25)	(10)	(0.20)	(0.15)	
Brick							
Red brick	USSR	12	50	1.6	0.017	0.15	[K2]
Red brick	Hungary	~200	55	3.9	0.036		[T1]
Red brick	Poland	3	18		0.02-0.05		[P1]
Red brick	USA	6	45	1.0	0.01		[I1]
Brick	Denmark	2		0.17			[J2]
Brick	Norway	18	63		0.01	0.15	[S6]
Brick	Greece	5	45	0.3-7.5	0.02		[S5]
Silicon brick	Poland	3	7-15		0.008-0.16		[P1]
Adobe brick	USA	2	30	3.5	0.06		
(Adopted reference value)			(50)	(2)	(0.02)	(0.15)	
Gypsum							
Gypsum	USA	12	12	6.3	0.28		[I1]
Gypsum board	Denmark	1		0.23			[J2]
By-product gypsum (apatite)	Poland	1	26		0.035		[P1]
By-product gypsum (phosphorite)	Poland	3	580-740		0.13-0.20		[P1]
Lightweight expanded clay aggregate	Norway	12	52		0.01-0.20	0.20	[S6]
Storage rock	USA	9	55	5	0.04		[I1]
Wood	USA	2		0.2			[I1]
Fly ash	Poland	33	100		0.005		[P1]
Slag	Poland	11	70		0.007		[P1]
Cement	Poland	4	> 9-25		0.008-0.08		[P1]
	USA	4	50	1.0	0.01		[I1]
Sand	USA	2	34	12	0.16		[I1]
	USA	2	10	3	0.12		[I1]
Gravel	USA	4	14	2.2	0.07		[I1]

a/ Calculated from data given in the reference, assuming a diffusion length of 0.2 m.

Table 17

Sources of radon entry rates in the reference house

Source	Radon entry rate (Bq m^{-3} h^{-1})	
	Estimated arithmetic mean	Typical range
Underlying soil		
Diffusion	1.7	] 0.1-200
Pressure-driven flow	40	]
Building materials	6.4	1- 20 a/
Outdoor air	5	1- 10
Water	0.1	0.001-100
Natural gas	0.3	0- 1
Total (rounded)	50	2-200

a/ Excluding alum shale; a radon entry rate of 80 Bq m^{-3} h^{-1} is estimated if aerated concrete based on alum shale is used.

Table 18

Equilibrium factor F for radon daughters

Country	Number of dwellings	Equilibrium factor F		Ref.
		Average	Range	
Canada				
March Township	-	0.38		[L4]
13 cities	9118	0.52 a/	0.19-0.67 b/	[M6]
	56	0.41 c/		[S44]
Finland	35	0.47	0.30-0.63	[M5]
Germany, Federal Republic of	38	0.37	0.25-0.65	[W5]
	200	0.30		[K6]
Italy	500	0.8 d/		[S14]
	50	0.7		[S14]
Netherlands	80	0.3		[P13]
Norway	25	0.5	0.3 -0.8	[S13]
Sweden	225	0.44	0.1 -0.8	[S12]
United Kingdom	130	0.30 e/		[W22]
United States				
New Jersey	21	0.52 f/	0.26-0.76	[G11]
		0.63 g/	0.33-0.82	[G11]

a/ Arithmetic mean of the ratio of the geometric means of the equilibrium equivalent concentration and of the radon concentration in 13 cities.
b/ Range for the 13 cities.
c/ Arithmetic mean; Gaussian distribution with $\sigma = 0.22$.
d/ Ratio of the medians of equilibrium equivalent concentration and of radon concentration.
e/ At mean ventilation rate of 2 air changes h^{-1}. Calculated to be 0.40 at 1 air change h^{-1}.
f/ Cellars.
g/ First and second floors.

Table 19

Radon and equilibrium equivalent concentrations obtained in recent large-scale indoor surveys

Country or area	Number of dwellings	Type of dwelling	Type of sampling	Purpose of the survey	Date of completion	Average value (Bq m^{-3})		Distribution or range	Ref.
						Radon	Radon EEC		
Argentina	112	Homes	Radon, passive	Population exposure	1985	37 (median)		Log-normal σ_g = 2.0	[P21]
Austria Salzburg	729	Homes	Radon and radon daughters, grab sampling, usually 4 times a year	Population exposure	1980?	15 (median)	9 (median)	Log-normal ? range <15-190 Bq m^{-3} (Rn)	[S16] [S17]
Belgium	79	73 houses and 6 apartments	Radon, passive, one-year exposure	Nation-wide population exposure (preliminary survey)	1984	41 (median)		Log-normal σ_g = 1.6	[P19]
Canada	13413	Houses	Radon and radon daughters, grab samplings, preferably in basements	Nation-wide variability of exposures	1980	33 (mean)	7 (median)	Log-normal	[L5] [G7] [U1]
Port Hope	2960	Homes	Radon, grab samples in basements	Remedial of industrial contamination	1976	36 (median)	11 (median)	Log-normal	[K8] [L5]
Uranium City	632	Homes in uranium mining communities	Radon and radon daughters, grab sampling, basements and living areas	Causes of increased radon levels in homes	1976		48 (median)	Log-normal σ_g = 4.1	[L5]
Elliot Lake	1921				1976		30 (median)	Log-normal σ_g = 2.9	[L5]
Bancroft	1162		Radon daughters, grab sampling, basements and living areas		1977		26 (median)	Log-normal σ_g = 3.1	[L5]
Saskatchewan	155 74 438 175 770 961	Homes	Radon daughters, grab sampling	Index map of radon problem areas	1980		11 (median) 28 (median) 65 (median) 13 (median) 16 (median)	Log-normal 47 (median)	[L5]
China	896		-	Population exposure	1983	120 (mean)	80 (mean)	-	[H18]
Beijing	537	364 houses 173 apartments		Population exposure	1987	30 (mean)		Range 0.6-259 Bq m^{-3} (Rn)	[P22]
Denmark	400	Homes	Radon, passive 3-month exposure in summer and in winter	Nation-wide population exposure (pilot survey)	1985	50 (median)		Range 50-700 Bq m^{-3}	[S62]
Germany, Federal Republic of	5970	Homes	Radon, passive, 3-month exposure, 1 in bedroom, 1 in living room	Nation-wide population exposure	1984	40 (median) 49 (mean)	13 (median)	Log-normal σ_g = 1.8	[W7, W15, S61]
Finland	8150	Homes	Radon, passive, 1-month exposure in living room	Nation-wide population exposure	1982	64 (median) 90 (mean)		Log-normal σ_g = 3.1	[C5] [C21] [C23]
France	765	Houses	Radon, passive and and RnDP, active, one-month exposure	Nation-wide population exposure (preliminary results)	1985	44 (median) 76 (mean)		Log-normal	[R6]
Hungary	833	Houses	Radon daughters, grab sampling, living areas, data reduced to 1 h^{-1} ventilation rate	Population exposure	1972		12	Log-normal	[T6]
Ireland	736	Houses	Radon, passive 6-month exposure	Nation-wide population exposure (preliminary results)	1987	37 (median)		3-1700 Bq m^{-3}	[M23] [C22]
Italy	1000	Houses	Radon, passive, 3-12 month exposures,	Nation-wide population exposure (partial results)	1984	43 (median)		Log-normal	[S14,
Japan	250	Houses	Radon, electrostatic integrating, 2 months; total 1.5 year exposure	Nation-wide population exposure	1988	10 (mean)			[N23]
Netherlands	1000	Houses	Radon, passive, exposure for 4 months in the living room	Nation-wide population exposure	1985	24 (median) 29 (mean)	10 (median) 12 (mean)	Log-normal σ_g = 1.6	[H1, P13, C22, P20]
Norway	1500	Houses	Radon, passive, 1 week exposure	Assessment of geographical variation	1985	90 (mean)		30-5000 Bq m^{-3}	[S66]

Table 19, continued

Country or area	Number of dwellings	Type of dwelling	Type of sampling	Purpose of the survey	Date of completion	Average value (Bq m^{-3})		Distribution or range	Ref.
						Radon	Radon EEC		
Poland	201	Apartments	Radon and radon daughters, grab sampling in the worst ventilation conditions	Nation-wide population exposure	1978	9 (mean)	4 (mean)	Log-normal ? range 0.4-17 Bq m^{-3} (EEC)	[G13]
Sweden	315	Detached houses built before 1975				122 (mean) 65 (median)		Log-normal	[S8, S42]
	191	Apartments built before 1975	Radon, passive 2-week exposure, 1 in bedroom, 1 in living room	Nation-wide population exposure	1982	85 (mean) 46 (median)	105 (mean)	Log-normal	
	96	Detached houses built 1978-1980				59 (mean)		Log-normal	
	38260	Detached houses built before 1982	Various	Search for dwellings with high radon levels	1986		Median of about 100	143 houses exceeding 2000 Bq m^{-3}	[S70, S71, S72]
	8008	Apartments built before 1982							
Switzerland	123	Homes	Radon, passive, detectors, 1 in basement, 1 in living room, 1 in bedroom	Nation-wide population exposure	1982	60 (median) 150 (mean)		Log-normal ?	[B4]
	105	Homes	-	Comparison of weatherproofed and conventional houses	1983			22-5500 Bq m^{-3} (Rn)	[B29]
United Kingdom	2300	Homes	Radon, passive, 1-year exposure in living room and in main bedroom	Nation-wide population exposure	1985	17 (median) in living-rooms 12 (median) in bedrooms		Log-normal σ_g = 2.4 in living-rooms σ_g = 2.2 in bedrooms	[W22]
Cornwall and other uraniferous regions	700	Homes	Grab sampling of radon and radon daughters plus ventilation rate plus 1-year exposure with passive radon detectors	Search for problem areas	1985?	140 (median) in living-rooms	14 (median) in living-rooms	Log-normal σ_g = 3.2 (Rn) σ_g = 5.2 (EEC)	[W8, B27, O9]
United States	552	Single-family homes	Various	Nation-wide population exposure	1984	35 (median) 61 (mean)		Log-normal σ_g = 2.8	[N11]

Table 20

Variability of radon daughter bronchial dose range as a per cent change from nominal annual dose for males [N14]

Factor	Variability	Comments
Unattached polonium-218 from 4% to 20%	-10	4% unattached polonium-218
	+30	20% unattached polonium-218
Daughter product equilibrium (1/0.9/0.7/0.7 to 1/0.9/0.6/0.4)	-20	
Particle deposition models		A few percent
Particle size (change in median size	+100	For 0.05 μm particles)
from 0.125 μm or 0.17 μm)	-20	For 0.17 μm particles)
Calculated physical dose to a given site in epithelium		A few percent
Breathing pattern (entirely active	+20	Active pattern
pattern to entirely resting)	-25	Resting pattern
(Mouth versus nose breathing)	(+35)	
Bronchial morphometry (child versus adult)	+60	
Mucociliary clearance (normal to complete stasis)	+10	
Mucus thickness (none to twice	+20	No mucus
normal)	-30	Twice normal mucus thickness
Location of target cell (shallow -22 μm to average -45 μm)	-80	

Table 21

Dose and effective dose equivalent factors per unit concentration of radon (or its decay products) in air (or water)

(Applicable only to adults in the general population.)

Pathway	Dose factor (nGy h^{-1} per Bq m^{-3})			Effective dose equivalent factor (nSv h^{-1} per Bq m^{-3})	Main assumptions
	Stomach	Tracheo-bronchial tree	Lungs		
Inhalation					
Radon gas	0.005		0.04	0.2	Decay of radon and decay products in the same tissue; solubility factor of 0.4
Radon decay products (EEC)					D_{T-B}/I_{pot} = 1.5 Gy J^{-1}
Indoors		7	0.9	10	D_P/I_{pot} = 0.2 Gy J^{-1} I_{th} = 0.8 m^3 h^{-1}
Outdoors		7	0.9	10	Same dosimetric coefficients for indoor and outdoor conditions
Ingestion					
Radon gas	0.0001			0.0001	D_{St}/A = 5 nGy Bq^{-1} Water consumption rate = 0.5 l d^{-1}

Table 22

Production of coal and reported activity mass concentrations of natural radionuclides

	Production of hard coal 1985 [U15]		Activity mass concentration (Bq kg^{-1})			Ref.
	Coal equivalent (10^9 kg)	% of the world production	U-238	Th-232	K-40	
Australia	118	4	30- 48	30	40	[B10,F5]
Austria	-		11-363	7-11	7-59	[B9]
Brazil	8	0.3	100	67	370	[S25]
Canada	34	1	12	7	26	[T4,T7]
Czechoslovakia	26	0.8	4-13			[J12]
China	810	26	7	16	30	[F5,J12]
Germany, Fed.Rep.						
Bituminous coal	88	3	20	< 20		[J13]
Brown coal			10	< 7		[J13]
Greece (lignite)			120-1300	0.7-0.9		[P10,P11]
Hungary	3	0.1	1.5			[J12]
India						
Average	150	5	24	38	83	[L12]
Range			10-70	20-90	15-440	[L12]
Italy (lignite)						
Central Italy			15-25		120-200	[D1,G16]
Sardinia			250			[D1,G16]
Poland						
Average	192	6	38	30	290	[T3]
Range			2-140	7-110	40-800	[T3]
South Africa	173	6	30	20	110	[S25]
USSR						
Average	494	16	28	25	120	[L11]
United Kingdom						
Yorkshire area			20	20	240	[B7]
Country range			7-94	2.4-19		[C9]
Country average	94	3	15	13	150	[S29]
United States						
Average	741	24	18	21	52	[B8]
Range			1-540	2-320	1-710	[B8]
Venezuela	0.04	0.001	< 20	< 20	110	[S25]
Yugoslavia	0.4	0.01	12-530	21-50		[B9]
World	3100	100				

Table 23

Uses of hard coal in OECD countries, 1985 [U15]

	OECD		Japan	United Kingdom	United States
	(10^9 kg)	(%)	(%)	(%)	(%)
Domestic	20	1.6	0.4	8.1	0.4
Commercial/public service	7	0.6	-	1.6	0.6
Power stations	835	67.8	22.9	71.7	84.4
Coke ovens	211	17.1	63.3	12.3	5.4
Iron and steel industry	5	0.4	0.3	0.03	0.3
Chemical industries	22	1.8	1.1	0.6	2.3
Other industries	77	6.2	8.3	4.9	5.5
Rail transport	0.2	0.02	-	-	-
Other	55	4.5	3.7	0.8	1.1
Total	1232	100.0	100.0	100.0	100.0

Table 24

Radon and radon decay products in coal mines

(Mean values with range in parentheses.)

Country or area	Year	Radon concentration ($Bq\ m^{-3}$)	Potential alpha energy concentration of radon a/ ($\mu J\ m^{-3}$)	Annual potential alpha energy exposure (mJ)	Ref.
European Communities b/	1981		0.09	0.2	[B19]
	1981		0.2	0.5	[B19]
	1981		0.5	1.2	[B19]
Germany, Federal Republic of	1976	105 (up to 400)			[E6]
India	1981		0.02 (0.01-0.04)		[N9]
Poland	1981		<2 c/		[D4]
United Kingdom	1968	(20-500)	0.4		[D3]
	1976			(up to 2)	[D10]
	1981			0.5 d/	[D11]

a/ 1 $\mu J\ m^{-3}$ corresponds to an equilibrium equivalent concentration of radon of 180 $Bq\ m^{-3}$.
b/ Radon decay product concentrations and annual exposures derived from two-month measurements; log-normal distributions.
c/ Less than 2 $\mu J\ m^{-3}$ for 94% of the measurements.
d/ Mean annual exposure in nationalized mines, employing 185,200 miners; the mean annual exposure in private mines, employing 1,500 miners, was 1.0 mJ.

Table 25

Ash production in the United Kingdom
[C11]

Source	Coal consumption (10^9 kg)	Ash content (%)	Ash production (10^9 kg)	Ash production (% of total)
Domestic and manufactured fuels	15.0	4.7	0.7	4.5
Power stations	73.1	17.1	12.5	80.1
Carbonization (coke, ...)	17.5	6.0	1.1	7.1
Other industry	12.1	9.6	1.2	7.7
Other, including export	1.5	6.3	0.1	0.6
Total	119.2	Weighted average 13.2	15.6	100.0

Table 26

Total emissions of respirable particulates in the United Kingdom
[C11]

Origin	Total emissions (10^6 kg)	Total emissions (% of total)
Domestic	337	74
Commercial/public service	12	3
Power stations	28	6
Other industry	68	15
Rail transport	10	2
Total	455	100

Table 27

Coal-fired power plants: estimates of annual atmospheric discharges per unit energy generated
(MBq per GW a)

Site	Fly-ash removal efficiency (%)	K-40	U-238 decay series				Th-232 decay series		Reference
			U-238	Ra-226	Pb-210	Po-210	Th-232	Th-228	
Canada a/									
Nanticoke, 4000 MW	99.5	350	100	100	300	300	70		[T4]
France	?	3500	7000	7000			6000		[A5]
Germany, Federal Rep. of									
Brown coal			100	70	200	400	40	40	[J14,S26]
Bituminous coal			500	500	4000	8000	200	200	[J14]
Bituminous dry ash removal	99		300	200	200	300	100	100	[H12]
Bituminous liquid ash removal	99		500	300	2000	6000	200	200	[H12]
India	95	31000		6400			10000		[L12]
Italy									
Lignite, 250 MW	95	42000	5500	5500	3500		22000		[D1,G16]
Lignite, 70 MW	95	120000	23000	23000	34000		34000		[D1,G16]
Coal, 1280 MW	99.5	3300	2500	2500			3300		[D1,G16]
USSR									
Zaporozhje b/ 1200 MW (old)	90	200000		20000	81000	74000	20000		[I6]
1000 MW (modern)	97.5	50000		5000	20000	18000	5000		[I6]
United Kingdom, 2000 MW	99.5	1000	1000	1000	1000	1000	1000	1000	[C9]
	99.5	4000	800	800	8000	8000	400	400	[C14]
United States	99		300	300	300	300	200	200	[M14]
			4900	5600			6300	6300	[N7]
M-1, 510 MW c/	99.3		2700	590	3200	7600	90	80	[U5]
M-2, 450 MW c/	99.3		720	520	1900	1800	190	220	[U5]
M-3, 125 MW c/	99.4		350	100	620	510	90	140	[U5]
M-4, 12.5 MW c/	99.7		450	100	830	720	220	230	[U5]
Milliken, 270 MW	99.7	550	70	70	100		60		[K14]
Model plant (modern)	99		1600	1200	3900	3900	670	670	[U5]
Model plant (1970)	80		16000	16000	32000	32000	14000	14000	[U5]
Model plant (modern)	98-99	1100	1000	780	2600	2600	410	410	[B8]
Model plant (1972)	90	10000	4700	4700	9300	9300	3800	3800	[B8]
UNSCEAR 1982 Report	97.5	4000	1500	1500	5000	5000	1500	1500	[U1]
This Annex									
modern	99.5	600	250	250	750	750	250	250	
old	90	12000	5000	5000	15000	15000	5000	5000	

a/ Assuming a coal consumption of 3 Tg per GW a.
b/ Ash content of the coal of 30-39% (Donety, Kuznets coal field); annual coal consumption of 5 Tg.
c/ Derived from the ratio of emission rate to activity feed rate for U-238.

Table 28

Estimates of collective effective dose equivalent commitments per unit energy generated resulting from atmospheric releases from a typical "old" coal-fired power plant a/
(10^{-2} man Sv per GW a)

Radionuclide	Inhalation during the cloud passage	Internal irradiation due to deposited activity	External irradiation due to deposited activity	Total
U-238	15	2		
U-234	17	2		
Th-230	60	3		
Ra-226	1.3	3	12	215
Rn-222 + daughters	0.1	20		
Pb-210 → Po-210	14	66		
Th-232	290	1		
Ra-228 + daughters	60	6.7	18	405
Rn-220 + daughters	-	30		
Total (rounded)	455	135	30	620

a/ Releasing about 10% of the fly-ash produced.

Table 29

Estimates of collective effective dose equivalent commitments per unit energy generated resulting from atmospheric releases from a typical modern coal-fired power plant a/
(10^{-2} man Sv per GW a)

Radionuclide	Inhalation during the cloud passage	Internal irradiation due to deposited activity	External irradiation due to deposited activity	Total
U-238	0.75	0.1		
U-234	0.9	0.1		
Th-230	3	0.15		
Ra-226	0.07	0.15	0.6	30
Rn-222 + daughters	0.1	20		
Pb-210 → Po-210	0.7	3.3		
Th-232	14	0.05		
Ra-228 + daughters	3	0.3	0.9	20
Rn-220 + daughters	-	1.5		
Total (rounded)	23	26	1.5	50

a/ Using emission control devices that allow only about 0.5% of the fly-ash produced to escape.

Table 30

Estimates of collective effective dose equivalent commitments per year of practice in the coal fuel cycle

Source	Collective effective dose equivalent commitment per year of practice (man Sv)	
	Public	Workers
Coal mining	0.5-10	2000
Use of coal		
Electrical energy production	2000	60
Domestic use	2000-40000	-
Coke ovens	Not estimated	Not estimated
Use of fuel ash	50000	Not estimated

Table 31

Use of oil in the OECD countries, 1978
[06]

Use	Type of fuel	Mass (10^9 kg)
Domestic	Gas/diesel oil	200
	Liquefied gases	33
Commercial/public service	Gas/diesel oil	26
Power stations	Residual fuel oil	200
Iron and steel industry	Residual fuel oil	22
Chemicals industry	Residual fuel oil	30
Other industries	Residual fuel oil	120
Air transport	Jet fuel	76
Road transport	Motor gasoline	490
Railways	Gas/diesel oil	20

Table 32

Uses of natural gas in OECD countries, 1985
[U15]

Use	OECD (total)		Japan	United Kingdom	United States
	Energy (PJ)	Share (%)	(%)	(%)	(%)
Domestic	7980	24.4	-	47.1	24.9
Commercial/public service	4310	13.2	-	5.8	13.7
Power stations	5850	17.9	75.1	1.1	17.1
Natural gas extraction	2340	7.2	1.5	5.6	8.6
Petroleum refineries	670	2.0	-	-	2.8
Iron and steel industry	}	}	2.0	2.2	}
Chemicals industry	} 10000	} 30.6	1.7	10.7	} 30.5
Other industries	}	}	0.2	15.2	}
Other	1550	4.7	19.5	12.3	2.4
Total	32700	100	100	100	100

Table 33

Collective effective dose equivalent commitment to members of the public attributable to various systems of electricity production

Type of plant	Collective effective dose equivalent commitment per year of practice (man Sv)	Normalized collective effective dose equivalent commitment (man Sv per GW a)
Coal-fired	2000	4
Oil-fired	100	0.5
Natural gas	3	0.03
Geothermal	3	2
Peat	-	2

Table 34

Estimated atmospheric discharges from phosphate industrial plants

Type of plant and location	Annual input of phosphate rock (10^6 t)	Radionuclide discharges (GBq a^{-1})						Ref.
		U-238	Th-230	Ra-226	Rn-222 a/	Pb-210	Po-210	
Ore drying and grinding								
United States, reference plant	2.7	0.6	0.6	0.6	4000	0.6	0.6	[U9]
Elemental phosphorus plant								
Vlissingen, Netherlands	0.75					150	350	[B31]
United States								
Pocatello, Idaho]		0.15	0.15	0.15	1500	4	330	[U9]
Soda Springs, Idaho]		0.2	0.2	0.2	1100	210	780	[U9]
Silver Bow, Montana]	~4	0.02	0.02	0.02	2000	4	26	[U9]
Mt. Pleasant, Tenn.]		0.007	0.007	0.007	40	2	4	[U9]
Columbia, Tenn.]		0.07	0.07	0.07	300	15	22	[U9]
Columbia, Tenn.]		0.007	0.007	0.007	40	2	4	[U9]
Wet-process fertilizer plant								
United States, reference plant	1	0.25	0.25	0.036		0.13	0.13	[U9]

a/ Atmospheric discharges of Rn-222 estimated by UNSCEAR.

Table 35

Estimates of collective dose commitments per unit mass of phosphate ore resulting from atmospheric releases from phosphate industrial plants

Radionuclide	Airborne discharge (Bq t^{-1})	Collective dose commitment (10^{-9} man Gy t^{-1})							
		Lungs	Bone surfaces	Red bone marrow	Liver	Kidneys	Spleen	Gastro-intestinal tract	Other soft tissues
Ore drying and grinding									
U-238	200	2.6	0.4	0.05	0.03	0.2	0.03	0.03	0.03
U-234	200	3.1	0.3	0.04	0.01	0.2	0.01	0.01	0.01
Th-230	200	2.9	23	1.7	0.04	0.04	0.006	0.006	0.006
Ra-226	200	0.2	1	0.1	0.03	0.03	0.03	0.03	0.03
Rn-222	$1.5\ 10^6$	17 a/ 2.2 b/							
Pb-210	200	0.3	3.1	0.5	0.5	0.5	0.4	0.4	0.4
Po-210	200	0.2	0.004	0.004	0.004	0.1	0.2	0.003	0.004
External irradiation		4.7	4.7	4.7	4.7	4.7	4.7	4.7	4.7
Total		22 a/ 16 b/	33	7.1	5.3	6.0	5.4	5.2	5.2
Elemental phosphorus plant									
U-238	100	1.3	0.2	0.03	0.01	0.1	0.01	0.01	0.01
U-234	100	1.5	0.2	0.02	0.007	0.1	0.007	0.007	0.007
Th-230	100	1.4	11	0.8	0.02	0.02	0.003	0.003	0.003
Ra-226	100	0.1	0.5	0.05	0.01	0.01	0.01	0.001	0.001
Rn-222	$1.2\ 10^6$	14 a/ 1.8 b/							
Pb-210	$5.9\ 10^4$	8.8	920	140	160	140	120	120	120
Po-210	$2.9\ 10^5$	340	5.8	5.8	5.8	170	300	4.1	5.8
External irradiation		2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3
Total		16 a/ 360 b/	940	150	170	310	420	130	130
Wet process fertilizer plant									
U-238	250	3.2	0.5	0.07	0.03	0.3	0.01	0.01	0.01
U-234	250	3.8	0.4	0.05	0.02	0.3	0.02	0.02	0.02
Th-230	250	3.6	28	2.1	0.05	0.06	0.007	0.007	0.007
Ra-226	40	0.04	0.2	0.02	0.005	0.005	0.005	0.005	0.005
Pb-210	100	0.1	1.6	0.2	0.3	0.2	0.2	0.2	0.2
Po-210	100	0.1	1.002	0.002	0.002	0.06	0.1	0.001	0.002
External irradiation		0.9	0.9	0.9	0.9	0.9	0.9	0.9	0.9
Total		12	32	3.3	1.3	1.8	1.3	1.2	1.2

a/ Tracheo-bronchial basal cell layer.
b/ Pulmonary epithelium.

Table 36

Estimates of collective dose commitments resulting from world-wide annual atmospheric releases from phosphate industrial plants

Radionuclide	Airborne discharge ($Bq\ t^{-1}$)	Collective dose commitment (10^{-3} man Gy)							
		Lungs	Bone surfaces	Red bone marrow	Liver	Kidneys	Spleen	Gastro-intestinal tract	Other soft tissues
Ore drying and grinding									
U-238	20	260	37	5.5	2.6	23	2.6	2.6	2.6
U-234	20	310	35	4.1	1.4	20	1.4	1.4	1.4
Th-230	20	21	9.9	10	2.6	2.6	2.6	2.6	2.6
Ra-226	200	0.2	1	0.1	0.03	0.03	0.03	0.03	0.03
Rn-222	130000	1700 a/ 200 b/							
Pb-210	20	30	310	48	55	47	40	39	39
Po-210	20	23	0.4	0.4	0.4	12	21	0.3	0.4
External irradiation		470	470	470	470	470	470	470	470
Total		2000 a/ 600 b/	3200	710	540	580	540	520	520
Elemental phosphorus plant									
U-238	1.5	19	2.8	0.4	0.2	1.7	0.2	0.2	0.2
U-234	1.5	23	2.6	0.3	0.1	1.5	0.1	0.1	0.1
Th-230	1.5	22	170	12.7	0.3	0.3	0.05	0.05	0.05
Ra-226	1.5	1.6	7.4	0.8	0.2	0.2	0.2	0.2	0.2
Rn-222	16000	180 a/ 24 b/							
Pb-210	800	1200	12000	1900	2200	1900	1600	1600	1600
Po-210	4000	4600	80	80	80	2300	4200	56	80
External irradiation		35	35	35	35	35	35	35	35
Total		220 a/ 5900 b/	12000	2000	2300	4200	5800	1700	1700
Wet process fertilizer plant									
U-238	10	130	19	2.7	11	1.3	0.01	1.3	1.3
U-234	10	150	17	2.1	0.7	10	0.7	0.7	0.7
Th-230	10	140	1100	85	2	2.2	0.3	0.3	0.3
Ra-226	2	2.1	1	1	0.3	0.3	0.3	0.3	0.3
Pb-210	6	9	94	15	16	14	12	12	12
Po-210	6	7	0.1	0.1	0.1	3.5	6.2	0.008	0.1
External irradiation		47	47	47	47	47	47	47	47
Total		490	1300	150	67	88	68	62	62

a/ Tracheo-bronchial basal cell layer.
b/ Pulmonary epithelium.

Table 37

Average activity mass concentrations of uranium-238 and radium-226 per unit mass of P_2O_5 in phosphate fertilizer

Country	Bq/kg P_2O_5		Ref.
	U-238	Ra-226	
Finland	1700	480	[M21]
Germany, Fed.Rep.of	2600	1700	[P15]
United States	9200	1300	[U13]
Union of Soviet Socialist Republics		930	[D8]

Table 38

Estimated collective dose commitment per mass of phosphate rock resulting from the use of phosphate fertilizers

Organ or tissue	Collective dose commitment (10^{-7} man Gy t^{-1})						
	U-238	U-234	Th-230	Ra-226	Rn-222	Pb-210	Total
Internal irradiation							
Lungs	1	1	4	0.4	300 a/ 40 b/		300 a/
Bone surfaces	20	20	60	10		100	300
Red bone marrow	3	2	4	1		20	30
Liver	1	1	0.07	0.4		10	10
Kidneys	10	10	2	0.4		10	30
Other tissues	1	1	0.07	0.4		10	10
External irradiation							
All tissues			2				2

a/ Tracheo-bronchial basal cell layer.
b/ Pulmonary epithelium.

Table 39

Estimates of collective effective dose equivalent commitment to the world population arising from one year of exploitation of phosphate rock

Source of exposure	Collective effective dose equivalent commitment (man Sv)	
	Members of the public	Workers
Phosphate industrial operations	60 a/	20 b/
Use of phosphate fertilizers	10000	50 b/
Use of by-products and waste	300000	Not estimated

a/ An additional 170 man Sv to the Dutch population is estimated to result from dumping phosphogypsum into the Rhine.
b/ From external irradiation only.

Table 40

Radon and radon decay products in non-uranium mines

Country	Year	Radon concentration (Bq m^{-3})	Potential alpha energy concentration of radon (μJ m^{-3})	Annual potential alpha energy exposure (mJ)	Type of mine	Ref.
China	1975-1978	9000-20000 a/	40-330 a/		Tin, copper, and tungsten	[S33]
Germany,	1984		1	3	Lead and zinc	[K15]
Federal	1984	280	1	3	Iron	[K15]
Rep. of	1984	20	0.2	0.4	Pyrite, zinc, barite	[K15]
	1984	110	0.4	1	Barite	[K15]
	1984		0.4; 6	0.9; 14	Fluorspan, barite	[K15]
	1984	4100	10	25	Fluorspan	[K15]
	1984	6600	20	60	Graphite	[K15]
		1200 b/	2 b/	5 b/		
	1984	120	0.2	0.4	Clay	[K15]
	1984	29000	80	200	Feldspar	[K15]
	1984	730	1	3	Slate	[K15]
	1984	460	0.8	2	Oil shale	[K15]
	1984	170	0.4	0.8	Rock salt	[K15]
	1984	8	0.02	0.04	Salt saline	[K15]
India	1981		0.04-0.3		Gold	[N9]
	1981		1.4 -3.5		Copper	[N9]
	1987	1200	4.8		Copper	[M29]
		19	0.07		Gold	[M29]
		190	0.7		Lead/Zinc	[M29]
		39	0.15		Manganese	[M29]
		79	0.3		Mica	[M29]
Italy	1981		0.04		Amianthinite	[S36]
	1981		1.2		Barite	[S36]
	1981		2.5		Bauxite	[S36]
	1981		0.2		Chalcopyrite	[S36]
	1981		0.04; 0.2		Feldspar	[S36]
	1981		1.5; 5.6		Fluorspar	[S36]
	1981		0.08-11.4 a/		Fluorspar	[S36]
	1981		4.8		Magnetite	[S36]
	1981		0.04- 5.4 a/		Siderite	[S36]
	1981		0.03- 0.7 a/		Steatite	[S36]
	1981		0.5; 3.4		Sulphur	[S36]
	1984	100-2800	0.4 - 10 c/		Not indicated	[S37]
Poland	1966-1970		1.9		Iron	[D4]
	1966-1970		3.1		Pyrite	[D4]
	1966-1970		0.6		Barite	[D4]
	1966-1970		2.2		Zinc-lead, copper	[D4]
	1972-1976		2.5 - 3.1 a/		Zinc-lead, copper	[D4]
	1977-1978		2.5		Zinc-lead, copper	[C15]
Sweden	1970-1980			3-20 d/	50 mines	[S35]
	1982			5	Iron, zinc, copper	[S58]
United	1973-1975		1.5 - 90	4-240 a/	Tin	[S34]
Kingdom	1973-1975		0.4 - 20 a/	0.6-30 a/	Haematite	[S34]
	1973		2	4	Iron	[S34]
	1973-1975		0.08-160 a/	0.4-120	Fluorspar	[S34]
	1973-1975		0.2 - 14 a/	0.7- 30	Limestone	[S34]
	1973		0.2; 1	0.4; 2	Calcspar	[S34]
	1973		0.6; 6	0.4; 15	Homestone	[S34]
	1973-1975		0.2 - 10	0.4- 20	Fireclay	[S34]
	1973		0.1; 1	0.4 - 5	Ballclay	[S34]
	1973-1975		6; 30	10; 55	Lead	[S34]
	1973		1.5	3	Slate	[S34]
	1973		15	40	Barite	[S34]
	1973-1975		0.06; 0.4	0.4; 1	Salt	[S34]
	1975		2	4	Silica sand	[S34]
	1981			11	Other than coal	[011]
United	1978	2600	5 - 8		Zinc	[S32]
States	1978	280	3		Iron	[S32]
	1978	1000	5		Fireclay	[S32]
	1978	50	0.8		Fluorspar	[S32]
	1978	160			Copper	[S32]
	1978	20	0.04-0.1		Limestone	[S32]

a/ Range of means.
b/ Winter measurements.
c/ Assuming an average equilibrium factor of 0.7.
d/ Average exposures decreasing with time from 20 mJ in 1970 to 3 mJ in 1980.

Table 41

Environmental radiation fields and occupational exposures in non-uranium mines of the United Kingdom
(Based on [D5])

RADIATION CHARACTERISTICS

Mine	^{238}U activity per unit mass ($Bq\ kg^{-1}$)	^{232}Th activity per unit mass ($Bq\ kg^{-1}$)	Potential alpha energy concentration of radon daughters ($\mu J\ m^{-3}$)	Potential alpha energy concentration of thoron daughters ($\mu J\ m^{-3}$)
1: sedimentary	66	53	0.06	0.04
2: sedimentary	65	56	0.15	0.11
3: sedimentary	<10	<10	0.94	0.96
4: igneous	60	2	29.5	0.1
5: sedimentary	18	6	1.9	0.51
6: igneous	180	90	9.4	0.64
7: sedimentary	35	44	0.89	0.41
8: igneous	270	65	29	1.4

OCCUPATIONAL DOSES

Mine	Average annual effective dose equivalent (mSv)	Contribution (per cent of the total)			
		External irradiation a/	Inhalation of radon daughters	Inhalation of thoron daughters	Inhalation of ore dust b/
1	0.7	25	51	10	14
2	1.4	13	66	13	7
3	7	<1	78	22	<1
4	180	<1	100	<1	<1
5	12	<1	93	7	<1
6	58	<1	97	2	<1
7	6	2	86	11	1
8	180	<1	98	1	<1

a/ Assuming a 4π geometry.
b/ Assuming an ore dust concentration of 3.8 mg m^{-3}.

Table 42

Estimated atmospheric releases of natural radionuclides from zinc mining and processing facilities
[A7, U8]

Radionuclide	Annual atmospheric releases (MBq)		
	Mine	Mill	Smelter
U-238	0.004	0.06	10
U-234	0.004	0.06	10
Th-230	0.004	0.05	5
Ra-226	0.003	0.03	7
Rn-222	8500000	40000	
Pb-210	0.01	0.05	20
Po-210	0.006	0.07	2
Th-232	0.002	0.02	3
Ra-228			3

Table 43

Atmospheric releases of natural radionuclides from a fireclay mine and refractory plant
[A6]

Radionuclide	Annual atmospheric emissions (MBq)	
	Mines	Kilns
U-238	< 0.1	5
U-234	< 0.1	5
Th-230	< 0.1	< 6
Ra-226	< 2	< 8
Rn-222	1200000	
Pb-210	<10	< 200
Po-210	< 7	130
Th-232	< 0.1	< 1

Table 44

Radionuclide activities per unit mass in surveyed alumina plant process samples
[A9, U8]

Sample	Activity per unit mass ($Bq\ kg^{-1}$)	
	^{238}U	^{232}Th
Bauxite ore	250	200
Alumina kiln feed	2	2
Alumina product	10	7
Red mud	280	180
Brown mud	200	460

Table 45

Estimated atmospheric releases of natural radionuclides from the surveyed alumina plant and aluminium reduction plant
[A9, U8]

Radionuclide	Annual atmospheric releases (MBq)		
	Alumina kilns	Red mud kilns	Aluminium reduction plant
U-238	2.5		
U-234	2.5		
Ra-226	2		
Rn-222		70000	
Pb-210		300	1200
Po-210		340	1000

Table 46

Radionuclide activities per unit mass
in surveyed copper mine and mill process samples
[A9, U8]

Sample	Underground mine and mill		Open pit mine and mill	
	^{238}U (Bq kg^{-1})	^{232}Th (Bq kg^{-1})	^{238}U (Bq kg^{-1})	^{232}Th (Bq kg^{-1})
Ore	30	23	80	110
Concentrate	24	3	50	40

Table 47

Estimated atmospheric releases of natural radionuclides
from copper ore mining and processing facilities
[A7, U8]

Radio-nuclide	Annual atmospheric releases (MBq)		
	Mine	Mill	Smelter
U-238		10	400
U-234		14	400
Th-230			700
Ra-226		7	60
Rn-222	240000	70000	
Pb-210		30	7000
Po-210		0.07	7000
Th-232			500
Ra-228			500
Th-228			500

Table 48

Estimates of per caput annual effective dose equivalents
and of ranges, excluding extreme values,
for the most important natural sources of radiation

Source of irradiation	Annual effective dose equivalent (μSv)	
	Mean	Typical range
External		
Cosmic rays	360	300-2000
Terrestrial sources	410	200-1000
Internal		
K-40	180	100- 200
U-238 → Ra-226	20	10- 50
Rn-222 → Po-214	1100	300-5000
Pb-210 → Po-210	120	50- 200
Th-232 → Ra-224	20	10- 50
Rn-220 → Tl-208	160	50- 500
Total (rounded)	2400	1500-6000 a/

a/ The 90th, 95th and 99th percentiles are estimated to be 2300, 3300 and 6100 μSv, respectively, assuming a log-normal distribution with a geometric standard deviation of 2.5.

Table 49

Estimates of collective effective dose equivalent commitments per year of practice for various industrial activities

Source	Collective effective dose equivalent commitment (man Sv)	
	Public	Workers
Coal combustion		
Power plants	2000	60
Domestic homes	2000-40000	
Coal mining	0.5-10	2000
Use of coal ash in the building industry	50000	
Geothermal energy production	3	
Oil combustion in power plants	100	
Natural gas combustion in power plants	3	
Phosphate industrial operations	60	20
Use of phosphate fertilizers	10000	50
Use of phosphogypsum in houses	300000	
Non-uranium mining	small	20000

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back to first page

ANNEX B

Exposures from nuclear power production

CONTENTS

Introduction

1. The generation of electric energy by nuclear reactors has increased since the Committee's assessment of doses from radioactive materials released during nuclear fuel cycle operations, as reported in Annex F of the UNSCEAR 1982 Report [U1]. The total world installed nuclear electricity generating capacity at the end of 1987 was 298 GW from 417 units in 26 countries [I1]. This represents an approximate doubling of nuclear capacity since the UNSCEAR 1982 Report, as may be seen from Figure I. Nuclear power was responsible for some 16% of the world's electricity generated in 1987, and currently some 120 reactors are under construction with an electrical capacity of 101 GW [I1]. Projections for world nuclear generating capacity for the year 2000 are still somewhat speculative, but the figure seems likely to be in the range of 400-500 GW [I2], somewhat less than earlier expectations but still representing a further expansion of 30-60% from currently installed capacity.

2. The number of power reactors operating at the end of 1987, their type and generating capacities for each country of the world is shown in Table 1. The reactor types include the pressurized water moderated and cooled reactor (PWR), the boiling water moderated and cooled reactor (BWR), the gas cooled reactors (GCR) of the Magnox and advanced gas cooled (AGR), graphite moderated type, the light water cooled graphite moderated reactor (LWGR), the heavy water moderated and cooled reactor (HWR), and the fast breeder reactor (FBR). The installed capacity per caput is also given in Table 1; it is highest in Sweden at 1.14 kW per caput and ranges from about 0.1 to over 0.8 kW per caput in other developed countries. The average installed capacity per person at about 0.14 kW represents an increase of 100% over the equivalent figure (0.07 kW) reported in the UNSCEAR 1982 Report. Table 2 shows the amounts and percentage of electricity generated in countries by nuclear power in 1987 [I2]. The highest use of nuclear reactors for electricity generation was in France (70%) and Belgium (66%).

3. The nuclear fuel cycle includes the mining and milling of uranium ores, conversion to nuclear fuel material, which usually includes the enrichment of the isotopic content of ^{235}U and fabrication of fuel elements; the production of energy in the nuclear reactor; the storage of irradiated fuel, or its reprocessing with the recycling of the fissile and fertile materials recovered, and the storage and disposal of radioactive wastes. Almost all of the artificial radionuclides associated with the nuclear fuel cycle are present in the irradiated nuclear fuel, although some neutron activation of structural and cladding materials takes place. The majority of irradiated fuel elements are currently stored; when reprocessing takes place, the highly active liquid wastes containing fission products and transuranium elements are stored in tanks isolated from the environment until they can be solidified. Solid wastes, arising at each stage of the fuel cycle, are mainly stored, although some wastes are disposed of.

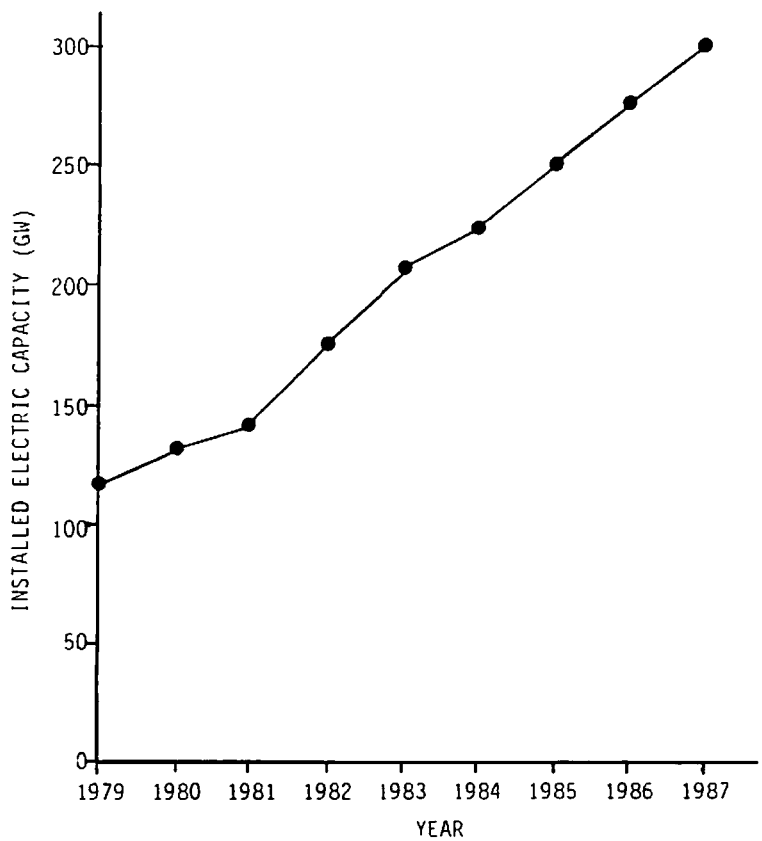

Figure I. The installed nuclear electric energy capacity on 31 December between 1979 and 1987.
[I1, I2, I3, I4, I5, I6, U1]

In routine operation of nuclear installations, small quantities of radioactive materials are released in effluents, which disperse in the environment and result in low-level exposures of the public.

4. The interest of the Committee is in assessing the radiation doses to individual members of the public from releases of radioactive materials and also the doses to workers from normal operation of the nuclear fuel cycle. Exposures of the public from high-level wastes, which arise in fuel reprocessing, have not been assessed by the Committee, as these wastes are still in storage. The majority of irradiated fuel is not being reprocessed. Preliminary estimates are made of the exposures in the future resulting from current disposals of radioactive solid wastes. The significant release of radioactive materials and the exposures to workers and the public that resulted from the accident at the Chernobyl nuclear power reactor are discussed in detail in Annex D, "Exposures from the Chernobyl accident", and Annex G "Early effects in man of high doses of radiation".

5. The quantities of radionuclides in effluents from nuclear facilities are usually reported and available to the Committee, reflecting the operational history of each plant, including periods of abnormal operation and maintenance shut-down. In this Annex the Committee reviews discharge data for the six-year period 1980-1985 and estimates average releases per unit of electric energy generated for each major power reactor type. Because the data for 1985 are incomplete, normalized releases are presented for the quinquennium 1980-1984. These normalized releases do not apply, of course, to any one plant but are deemed to be representative of current nuclear power generation. Future practices may lead to discharge levels considerably different from the normalized values presented here, which include new and old plants; therefore, any extrapolation to the future must be undertaken with caution.

6. Because of the system of controls applied to environmental releases from nuclear power installations, doses to individual members of the public correspond to low levels of individual risk. The doses to the most exposed individuals vary widely from installation to installation and from one location to another, and the level of individual dose generally decreases rapidly with distance from a given source. In this Annex an indication is given of the range of individual doses associated with each type of installation. To evaluate the total impact of radionuclides released at each stage of the fuel cycle, results are presented in terms of the collective effective dose equivalent commitment per unit quantity of electric energy produced, expressed as man Sv per GW a.

7. The collective dose commitment from nuclear power production is considered in four population groups: the occupationally exposed; the local population, being those within about 100 kilometres of the site; the regional population, those within about a 1,000 kilometres of the site; and the remaining world population. Each stage of the nuclear fuel cycle is treated separately, and the occupational, local and regional dose commitments are evaluated. The contributions from nuclides that, because of a combination of long radioactive half-lives and rapid dispersal in the environment, become globally dispersed and irradiate the world population are then discussed for the fuel cycle as a whole.

8. Collective dose commitments to local and regional populations must be estimated by environmental modelling, as the activity concentrations resulting from effluents from nuclear fuel cycle operations are very low both in environmental samples and in the general population. Monitoring of activity concentrations due to effluent releases has concentrated on areas immediately surrounding nuclear facilities to ensure compliance with relevant regulations. To estimate collective dose commitments it was decided in the UNSCEAR 1982 Report to establish a model facility at a representative site for each stage of the fuel cycle; mining and milling, fuel fabrication, reactor operation and reprocessing. The environment receiving the normalized releases from each model facility was chosen to represent broad averages containing typical features of existing sites and reflecting the most common environmental pathways. Such generalizations gave dose commitments indicative of the impact of the overall nuclear power programme though not applicable to any one site. In the UNSCEAR 1982 Report, the collective doses were evaluated for reported discharges at the three operating commercial reprocessing plants at Sellafield in the United Kingdom and Cap de la Hague and Marcoule in France.

9. The methods used by the Committee for estimating the dispersion of radionuclides released to the atmosphere or hydrosphere and the resulting doses to individuals were described in Annex A of the UNSCEAR 1982 Report. The Committee considers that, in general, these methods and the model facilities and representative sites used in the UNSCEAR 1982 Report are still valid for assessing the current impact of discharges from the fuel cycle. Therefore, in this Annex, the collective effective dose equivalent commitments are obtained by scaling the dosimetric results from the UNSCEAR 1982 Report, allowing for different releases of the various radionuclides involved. The Committee has decided to treat the reprocessing contribution differently in this Report. The hypothetical model facility is not used, but rather, in order to reflect the actual dose contributions made, the normalized dose commitments from the fraction of fuel reprocessed is added to the contributions from the rest of the fuel cycle.

10. Very long-lived nuclides pose a special problem. One example is ^{129}I (half-life: $1.6\ 10^7$ a), while another is radon gas, which emanates from mill tailings containing ^{230}Th (half-life: $8\ 10^4$ a) and ^{238}U (half-life: $4.5\ 10^9$ a). Assessments of human exposures over such periods of time are clearly hypothetical and the relevance of the results is doubtful. Dose commitments assessed for the purpose of calculating maximum dose rates in the future involve integration over the period of practice leading to the release of the radioactive material. This approach is taken in this Annex for effluents. For the solid waste disposal assessment, it is

in general only possible to assess the collective effective dose equivalent commitment.

11. There have been a number of attempts to generate rigorous definitions of the waste categories generally referred to as low-, intermediate- and high-level wastes [I13]. Although precise definitions have been agreed for particular purposes, the schemes proposed have not been universally satisfactory. None the less, the general characteristics of the three waste types are reasonably well established.

12. High-level wastes (HLW) are primarily the spent fuel elements or the solidified waste products from reprocessing. They have high activity concentrations of both actinides and fission products and are significantly heat-generating. As fuel elements are a significant potential source of fissile material, they will usually be stored in the short-to-medium term rather than disposed of. Occasionally, other waste streams with high activity concentrations are also regarded as HLW, but the quantities of activity in them are relatively small.

13. Intermediate-level wastes (ILW) are defined to some extent by exclusion from the other two categories; they contain either actinides or long-lived beta/gamma emitters in quantities that are not negligible or substantial activity concentrations of shorter-lived beta/gamma emitters and are not significantly heat-generating.

14. Low-level wastes (LLW) contain primarily reasonably short-lived beta/gamma emitters in low-to-moderate activity concentrations. They may contain actinides or long-lived beta/gamma emitters but only in very small quantities.

15. There will be other categories of materials that are uncontaminated, even though they were generated at a nuclear site or are of such a low level of activity concentration that they can be exempted from the requirements for storage and disposal as radioactive waste. The rationale for such exemption is that the radiological impact of uncontrolled disposal of these materials is insignificant [I14, N7]. These wastes are not considered part of this study, as their potential for radiological impact is by definition very low in comparison with that from the other waste categories.

16. In this preliminary assessment of doses from disposed wastes, only LLW and some categories of ILW are considered to be disposed of by shallow land burial. All other wastes are stored under conditions such that the doses to members of the public are essentially zero, and doses to occupational workers are included in those assessed for other operations at the same sites.

17. The Committee presented detailed comprehensive reviews of occupational exposures, including those from the nuclear fuel cycle, in both the UNSCEAR 1977 Report [U2] and the UNSCEAR 1982 [U1] Report. In this Annex the data on occupational exposures throughout the nuclear fuel cycle are brought up to date.

18. With regard to assessing occupational exposures, the relationship between measurements of external irradiation made in radiation fields by film, thermoluminescent or other personal dosimeters and the absorbed doses in the tissues and organs of the body was discussed in the UNSCEAR 1982 Report. The Committee adopted the convention that all numerical results reported by monitoring services represent the average absorbed dose in the whole body, recognizing that these are almost always readings from the dosimeters that are reported, without consideration of the relationships to the absorbed doses in organs and tissues of the body. In this Annex the Committee adopts a similar convention; but to simplify comparisons, and because most exposures are to penetrating gamma-radiation, the numerical result is taken to represent the effective dose equivalent. Exposures of uranium miners to radon and its daughters are also expressed in terms of effective dose equivalent.

19. The characteristics of occupational dose distributions identified by the Committee as of interest were: (a) the annual average effective dose equivalent H_{eff}, which is related to the average level of individual risk: this average has generally been calculated for all individuals monitored in a given occupational group; (b) the annual collective effective dose equivalent, S_{eff}, which is related to the impact of the practice; (c) the collective effective dose equivalent distribution ratio, defined as the ratio of the annual collective effective dose equivalent delivered at annual effective dose equivalents exceeding 15 mSv to the total collective effective dose equivalent. This is related to the proportion of workers exposed to higher levels of individual risk. These characteristics may be obtained for any form of the dose distribution, whether or not it exhibits a log-normal or other defined response over any part of the effective dose equivalent range. The collective effective dose equivalent is usually calculated from collated dosimetry results using the definition

$$S_{eff} = \sum_{0}^{\infty} N_i \overline{H}_{eff,i}$$

where N_i is the number of individuals in the effective dose equivalent range i for which $\overline{H}_{eff,i}$ is the mean annual effective dose equivalent. The annual average effective dose equivalent, $\overline{H}_{eff}$, is given by

$$\overline{H}_{eff} = S_{eff}/N$$

where N is the total number of workers monitored.

20. The normalized measure of the impact of the various components of the nuclear fuel cycle is the collective effective dose equivalent per unit electric energy generated. This is calculated as an average over a complete power programme or over several years to avoid anomalies such as those connected with the shut-down of reactors for maintenance. The results for doses from occupational exposures and to the local, regional, and global populations exposed as a result of effluent discharges to the environment may be taken to be a relative measure of the health impact of nuclear power production.

I. MINING AND MILLING

21. Uranium mining operations involve the removal from the ground of large quantities of ore containing uranium and its daughter products at concentrations between a tenth and a few per cent U_3O_8. These concentrations are several thousand times the concentration of these nuclides in the rest of the natural terrestrial environment. Uranium is mainly mined using underground or open-pit techniques, other methods such as heap leaching accounting for only a few per cent of the world production. The quantities produced during the period 1980-1984 are given in Table 3. Milling operations involve the processing of these large quantities of ore to extract the uranium in a partially refined form, often known as yellow-cake. This is further refined, converted and enriched, if necessary, before fabrication into fuel elements. Uranium mills tend to be located near mines to minimize transportation. The number of mills operating is related to uranium demand.

A. EFFLUENTS

22. The predominant gaseous effluent from active uranium mines is ^{222}Rn in the ventilation air from underground mines or released into the pit from surface mines. In a study covering 27 mines [J2] this accounted for 97% of the radon released. A recent study [N5] has also shown that for some surface mines, especially where a large volume of overburden has to be removed to expose the ore, waste rock piles formed a source of radon of a magnitude comparable to that of the pit. Release rates per unit mass of ore were estimated in the UNSCEAR 1982 Report at about 1 GBq t^{-1} from underground mines and about 0.1 GBq t^{-1} from surface mines. In general, however, the ore from underground mines was estimated to have about 10 times the uranium concentration of that from surface mines; the normalized radon emission was thus taken for both types to be 1 GBq t^{-1} of ore for 1% uranium oxide in the ore. Particulates in airborne dust contain ^{238}U and its daughters and sometimes ^{232}Th and its daughters.

23. The results of measurements or estimates of either total radon emission rates or normalized radon emission from a number of mines are given in Table 4. The data for underground mines relate to the ventilation air from the shaft, those for surface mines, to the mine pit. The results support retention of an overall normalized radon emission of 1 GBq t^{-1} of ore for 1% uranium oxide in the ore.

24. The uranium requirements per unit electric energy generated vary somewhat between current designs of thermal reactors; but the heavy metal requirements are generally in the range of 150-250 t $(GW\,a)^{-1}$. The grade of ore mined at present is usually between 0.1 and 1% U_3O_8. Taking a typical value for underground mines from the United States of 0.2% [E1], the normalized radon releases are about 20 TBq $(GW\,a)^{-1}$. This is the same value that was estimated in the UNSCEAR 1982 Report.

25. The processing of uranium at the mill was described in the UNSCEAR 1982 Report, as were the broad characteristics of the tailings piles, where most of the activity not extracted as usable uranium resides. This activity is predominantly ^{230}Th and its daughters. There are airborne emissions during operation of a mill, mainly of ^{222}Rn together with ^{238}U, ^{230}Th, ^{226}Ra and ^{210}Pb. The ranges of airborne release rates for a typical mill estimated in the UNSCEAR 1982 Report are shown in Table 5.

26. During operation of a mine, there are stockpiles of ore and piles of sub-ore, overburden and waste rock. After closure there will typically be a pile of overburden, possibly covered by sub-ore, in case processing of this becomes economically viable in the future. These also act as sources of airborne emissions, principally of ^{222}Rn. An estimate of the radon emanation rate from waste rock per 1% ore grade in the United States is 100 Bq m^{-2} s^{-1} [N4]. The number of inactive mines in the United States was estimated to be about 1,250 surface and 2,000 underground in 1980 [H9]. Some useful measurements have been made of radon emanation rates under dry conditions over a wide range of ore grades in the Northern Territory of Australia [L2, M5]. These suggest that a radon exhalation rate of 50 Bq m^{-2} s^{-1} per 1% ore grade is widely applicable; this figure is equivalent to 0.5 Bq m^{-2} s^{-1} per Bq g^{-1}.

27. Extraction of uranium during milling is clearly made as complete as possible but cannot reach 100%. Typically, the residual tailings from the mill will contain from 0.001 to 0.01% U_3O_8, depending on the grade of ore and the extraction process. Tailings are discharged from mills into impoundments, the characteristics of which depend on the local climate and geology [T1]. From the point of view of estimating effluents, the major differences are whether the tailings pile is wet or dry and whether it has been covered. All tailings piles act as sources of airborne releases, although if they are completely covered by water, the rates can be extremely low. Estimates of radon emanation for a number of typical mill tailings areas and impoundments are shown in Table 6. Most of these are taken from an extensive study by the Nuclear Energy Agency (NEA) [N5]. The radon exhalation rate per unit area and specific activity of ^{226}Ra was estimated in the UNSCEAR 1982 Report at about 1 Bq m^{-2} s^{-1} per Bq g^{-1} of ^{226}Ra in the tailings, although it was noted that the rate could vary from effectively zero to an order of magnitude higher than the above figure. It has been suggested that a more realistic figure would be 0.2-0.5 Bq m^{-2} s^{-1} per Bq g^{-1} [S12]. For comparison, 0.01% U_3O_8 ore contains approximately 1 Bq g^{-1} of ^{226}Ra. Detailed measurements have been carried out on seven tailings dams in South Africa [A7], giving a mean radon exhalation rate of 0.4 Bq m^{-2} s^{-1} per Bq g^{-1} for a radium concentration ranging from 0.2 to 0.7 Bq g^{-1}. Measurements on tailings in the Elliot Lake area of Canada [B26] showed a range from 0.2 to 7.6 Bq m^{-2} s^{-1} per Bq g^{-1}. Experimental investigations on two types of bare dry tailings in Australia [S13] showed exhalation rates from 0.3 to 0.7 Bq m^{-2} s^{-1} per Bq g^{-1}; these were reduced by a factor of 3 for 1 m dry cover and by more than a factor of 10 for 1 m moist cover.

28. In considering the longer-term impact of effluents from tailings piles, it must be assumed that activity concentrations from uranium nuclides remain practically constant indefinitely, due to their long half-lives. The rest of the activity in the tailings is dominated by ^{230}Th, which has a half-life of 80,000 a. The radionuclides in the decay chain from ^{230}Th with the greatest radiological significance are ^{226}Ra, which can be leached out by water access, ^{210}Pb and ^{222}Rn, which can escape into the air.

29. At present, tailings have tended to be kept in open, uncontained piles or behind engineered dams or dikes with solid or water cover. It is likely, however, that some further engineering will be carried out to minimize the release of radionuclides from the abandoned piles. Such techniques were analysed in the NEA study [N5] for a number of sites. The radon flux density varied by factors of more than 10^6, dependent on the treatment assumed, showing that this is clearly a crucial parameter in the assessment of the impact of tailings piles. The options assumed for one typical site in an arid region and the relative radon flux densities assumed to result are shown in Table 7. Similar reductions in radon emission have been found using covers of various types [H10]. Assuming some reasonably impermeable cover is used, the radon exhalation rate from a typical tailings pile is taken to be 10^5 Bq m^{-2} a^{-1}. This is less than the figure assumed for emanation from the unstabilized material stockpiled around working mines and comparable with the value expected to be achieved in the United States [E4]. The cover is assumed to provide some protection against erosion, so that the radon exhalation rate remains essentially constant with time. Otherwise, an increase of up to double the initial rate of emanation from a bare pile could have been expected over a period of about 10^4 years [N5]. As can be seen from the results of the UNSCEAR 1977 and UNSCEAR 1982 Reports, these are critical assumptions in determining the overall impact of the fuel cycle.

30. Mine and mill sites in dry areas give rise to effectively no liquid effluents. For those in wet climates, however, run-off water will contain radionuclides and may need treatment before release into watercourses. The most important radionuclide in liquid effluents is ^{226}Ra, and typical releases at wet sites were estimated in the UNSCEAR 1982 Report to be 1 GBq (GW a)$^{-1}$. A review by Kaufmann [K5] suggests values of the order of 0.1 GBq (GW a)$^{-1}$, given normal procedures for water treatment.

B. LOCAL AND REGIONAL COLLECTIVE DOSE COMMITMENTS

31. In the dose estimation procedure used in the UNSCEAR 1982 Report, the typical characteristics of a mine and mill site in terms of population density, rainfall, farming, etc. were first established. The population densities used were 3 km^{-2} for 0-100 km and 25 km^{-2} for 100-2,000 km. A deposition velocity of 10^{-2} m s^{-1} was taken for particulate releases. The collective dose for radon release was then calculated using an atmospheric dispersion model with characteristics typical of a semi-arid area and an effective release height of 10 m. The atmospheric dispersion model was described in the UNSCEAR 1982 Report and in the original reference [C1]. The resultant collective effective dose equivalent commitments per unit activity released are shown in Table 8, with the exception of the figure for radon. This has been reduced for the reasons discussed in Annex A which have led to a reduction in the dosimetric coefficient for outdoor air from 17 to 9 nSv h^{-1} per Bq m^{-3}. These figures have been used in this Annex to estimate the normalized collective effective dose equivalent commitments from current atmospheric releases which is about 0.3 man Sv (GW a)$^{-1}$. The doses from liquid effluents are negligible by comparison.

32. Using the figure estimated for the initial rate of exhalation of radon from a typical tailing pile leads to an annual release of about 1 TBq ha^{-1}. The production of a mine generates about 1 ha (GW a)$^{-1}$ of tailings, so the releases during a period of five years, corresponding to the duration taken for the current discharge, would add a normalized collective effective dose equivalent commitment of 0.1 man Sv (GW a)$^{-1}$. The rate of release as a function of time is assumed to be constant, and given the very long duration of the source, the normalized collective effective dose equivalent commitment is proportional to the duration considered reasonable for assuming the release. Taking this period to be 10^4 years for the sake of illustration, the result is an estimated 150 man Sv (GW a)$^{-1}$. An alternative perspective on this component can be obtained by assessing the truncated collective effective dose equivalent commitments up to different times. Some examples of the results of such calculations for the various coverings described in Table 7 are shown in Table 9, taken from the same study [N5].

C. OCCUPATIONAL EXPOSURES

33. The main source of radiation exposure of underground uranium miners is radon and its daughters. The annual average exposure of underground miners was taken to be 1.5 WLM in the UNSCEAR 1982 Report; this was converted to an annual effective dose equivalent of about 13 mSv. Surface miners have a lower exposure to radon and daughters, with annual doses estimated to be about 3-4 mSv, but they and underground miners are exposed through inhalation of dust containing uranium and its daughters. Both underground and surface miners are also exposed to some external gamma radiation. The estimate of annual doses for underground miners was rather broad in the UNSCEAR 1982 Report, 1-10 mSv; that for surface miners was taken to be 1-2 mSv. Where the authors have not carried out their own conversions, use has been made of the conversion coefficients given by the International Commission on Radiological Protection [I12] between committed effective dose equivalent and time integrated equilibrium equivalent radon daughter concentration in air of 17 nSv h^{-1} per Bq m^{-3} or 10 mSv WLM^{-1}, where 1 WLM is one working month (170 ha) of exposure to a potential alpha-energy concentration of 2.08 10^{-5} J m^{-3} [I10].

34. Exposure of uranium miners to radon and daughters has been monitored by a combination of measurement of levels in air at a variety of places through the mine and estimates of the time spent by miners in those places. In recent years, however, there has been considerable development work on dosimeters suitable for monitoring of radon daughter exposures for individual underground uranium miners. Some recent results for underground uranium miners are shown in Table 10. The United States data for 1980, assumed to be primarily for underground miners, are from a very general summary prepared by the Environmental Protection Agency [E3]; those for 1981 and 1982 relate only to the mines in New Mexico [S8]. The data for Canada [A4] include exposures at the uranium mills associated with the mines. Data can be clearly separated into underground and a surface mine for the Canadian mines, and the results for the surface mine at Key Lake [A4] are shown in Table 11. A comparison between mine company records and exposures based on measurements by inspectors for 1979 and 1980 in the United States showed reasonable agreement [C7]. In this study the annual average effective dose equivalent to underground workers in 61 mines from exposure to radon and daughters was estimated to be in the range of 18-29 mSv, depending on the assumptions made in deducing the personnel exposures from the measurements in working areas. This is somewhat higher than the estimates given in Table 10.

35. Information on gamma exposures to workers in both underground and open pit mines in Canada [A4] shows annual average effective dose equivalents ranging from 0.1 to 3.4 mSv for the years 1981-1983. Some underground mines showed average gamma doses as low as for surface mines, but the major underground mines employing more than 80% of the work-force had an annual average effective dose equivalent of 3 mSv. An estimate of 3 mSv as the annual average effective dose equivalent from inhalation of dust has also been made for the Ranger surface mine in Australia [A8].

36. Taking all the above information into account, the average annual effective dose equivalent to underground uranium miners from both external exposure and radon daughter exposure is 10-12 mSv; that for surface miners is lower, possibly around 5 mSv. Given the predominance of underground miners, an overall annual average of 10 mSv seems a reasonable estimate for the early 1980s. Taking the productivity to be 3 t a^{-1} of natural uranium per miner and a natural uranium requirement of about 200 t $(GW\ a)^{-1}$, the normalized collective effective dose equivalent would be 0.7 man Sv $(GW\ a)^{-1}$. This is comparable to the estimate in the UNSCEAR 1982 Report of 0.9 man Sv $(GW\ a)^{-1}$, which was rounded up to 1 man Sv $(GW\ a)^{-1}$.

37. Recent data on doses received by 3,000 workers at uranium mills in the United States show an annual average effective dose equivalent of 2.7 mSv [E3]. The external average effective dose equivalent to 131 workers at the Nabarlek mill in Australia during the period 1981-1982 was 1.5 mSv [M9] and at the Ranger mill during the period 1985-1986 as low as 0.9 mSv [A8]. The contribution from workers at mills to the collective effective dose equivalent per unit electric energy generated is so small that in the UNSCEAR 1982 Report it was not included as a separate item. This situation does not appear to have changed.

II. URANIUM FUEL FABRICATION

38. The uranium ore concentrate produced at the mills is further processed and purified and converted to uranium tetrafluoride (UF_4), and then to uranium hexafluoride (UF_6), if it is to be enriched in the isotope ^{235}U, before being converted into uranium oxide or metal and fabricated into fuel elements. Natural uranium, containing 0.7% ^{235}U, is used in graphite or heavy water moderated reactors. Enrichments of 2-5% are required for light water reactors (LWRs) and advanced gas cooled reactors (AGRs).

39. To produce natural uranium metal fuel, the uranium tetrafluoride is compressed with shredded magnesium and heated, and the resulting reduced uranium is cast into rods that are machined and inserted into cans. Natural uranium oxide is sintered into pellets and clad in zirconium alloy for HWR fuel pins. For LWR and AGR fuel, the UF_4 is converted into the gaseous form UF_6. The first type of enrichment plant to be developed commercially employed the gaseous diffusion process. In this, the UF_6 diffuses through a porous membrane, the lighter compound containing ^{235}U and ^{234}U diffusing more rapidly than the heavier compound containing ^{238}U. Partial separation occurs, but in practice many stages of such membranes are required in series to provide a cascade.

40. The pumping power required to move the UF_6 through the cascade requires a large amount of electric energy. The alternative gas centrifuge process consumes only about 5% of the electric energy demanded by the diffusion process. The gas centrifuge process is based on the separation effect on a mixture of UF_6 isotopes in a strong centrifugal field in a rotating cylinder, suitably combined with the cascading effect of counter-current circulation. More separation is attained in one centrifuge stage than one diffusion stage but, as the mass flow is less, a series-parallel configuration is required.

41. To fabricate LWR fuel the enriched UF_6 is converted to the oxide (UO_2) powder, which is granulated, sintered and pressed into pellets. These are inserted into tubes (cladding) that are sealed after being filled with pellets. For LWR fuel cans zirconium alloy is used, while for AGRs stainless steel cans are adopted. After the enrichment process, large quantities of depleted uranium remain, containing about 0.3% or more ^{235}U. This uranium would become a source of public exposure were it to be disposed of, but currently it is stored for possible use in breeder reactors and for other purposes. The solid wastes arising during operation of the uranium fuel fabrication facilities will contain the same radionuclides as those at uranium mines and mills, but will be trivial in quantity by comparison. It does not, therefore, seem worth while to assess their impact separately.

A. EFFLUENTS

42. Emissions of radionuclides from the conversion, enrichment and fuel fabrication processes are generally small. Most of the uranium compounds are solid and are easily removed from airborne effluent streams, while settling tanks are used to reduce liquid effluent discharges. Few data published in the United States or Europe give discharge rates of radionuclides from these fuel cycle facilities. The Committee concluded in the UNSCEAR 1982 Report that discharges were small and estimated releases from model facilities producing LWR fuel. In the United Kingdom, reported discharges are given in terms of total alpha, total beta activity and masses of uranium (Table 12), and some isotopic breakdown can be obtained [G2] for centrifuge enrichment plant effluents. Most of the beta-discharges are from the short-lived ^{234m}Pa (half-life: 1.17 min) which is separated with ^{234}Th (half-life: 24.1 d). Canadian data are also available for effluents from a conversion plant [A4, L1] with their isotopic composition [M2]. There are small releases of ^{99}Tc reported from the British enrichment plant, indicating some recycling of reprocessed uranium, but these releases are atypical and no dose assessment has been made.

43. The data presented in Table 12 have been used to obtain the effluent releases which are applied to the same model facility sited on a river as was used in the UNSCEAR 1982 Report. The normalized releases are based on an LWR cycle uranium requirement of 150 t $(GW\,a)^{-1}$ and an HWR cycle requirement of 170 t $(GW\,a)^{-1}$. The results are given in Table 13 for atmospheric and aquatic effluents. The conversion plant figures are based on data from Canada [M2], as are those for fabrication, since these relate to fresh-water discharges in contrast to the British figures, which relate to marine discharges. The values quoted in Table 13 are typical figures taken from those calculated for the five Canadian fabrication plants, based on a fuel cycle requirement of 170 t $(GW\,a)^{-1}$. The discharges of ^{234}Th are obtained by assuming that this radionuclide is in equilibrium with ^{234}U.

44. The results in Table 13, which were derived from reported discharges, can be compared with the effluents from the model facilities quoted in the UNSCEAR 1982 Report, which were based mainly on the notional results produced by the Environmental Protection Agency [E2]. The results using present data suggest that for conversion, atmospheric releases are generally about twice those quoted previously for uranium and thorium isotopes, while aquatic releases as reported are about 10% of those assumed previously, and in the case of ^{226}Ra are only 1% of that in the Environmental Protection Agency model facility assumed previously. Atmospheric releases from enrichment are about one half of those quoted in the UNSCEAR 1982 Report; liquid effluents are only a few per cent of the Committee's previous estimates. For fuel fabrication, based on a weighted average of natural and enriched fuels, the atmospheric and aquatic releases are again about one half the previously assumed values.

B. LOCAL AND REGIONAL COLLECTIVE DOSE COMMITMENTS

45. The Committee concluded in the UNSCEAR 1982 Report that releases to the atmosphere provided the major exposure to the population (over 90%) from fuel conversion, enrichment and fabrication processes. To obtain an order of magnitude assessment of the collective dose commitments, the Committee specified a model facility with a constant population density of 25 km^{-2} out to 2,000 km. This was chosen to be representative of North America and Europe, and collective dose commitments were derived for inhalation from the plume, ingestion of foodstuffs contaminated by activity deposited from the plume and by external irradiation from the activity deposited on the ground. The same results have been used here, but the collective effective dose equivalent commitments have been scaled for the normalized releases derived in Table 13; the resultant doses are given in Table 14. The most significant pathway of exposure continues to be inhalation of particulate activity, with radon daughters contributing about 15% of the dose.

46. In summary, the normalized collective effective dose equivalent commitment due to uranium fuel fabrication is estimated to be 2.8 10^{-3} man Sv $(GW\,a)^{-1}$. The main contribution arises from inhalation of the isotopes of uranium. The figure is similar to that derived in the UNSCEAR 1982 Report [2.0 10^{-3} man Sv $(GW\,a)^{-1}$]. Individual doses in the vicinity of fuel fabrication facilities are estimated to be less than 50 mSv per year for members of the public [B1, B2, B3, B8, B16, B29].

C. OCCUPATIONAL EXPOSURES

47. The annual average effective dose equivalents to workers in fuel fabrication plants were found in the UNSCEAR 1982 Report to be generally low, ranging from 0.3 to 3 mSv. The annual collective effective dose equivalent distribution ratio (see paragraph 19) was also in general small, often approaching zero. Data on the number of workers employed and the corresponding annual average individual and collective effective dose equivalents are given for some countries in Table 15. These are not always complete for a country for any particular year and could include workers not strictly employed in fuel fabrication. For example, the data from the United States [N2] are quoted as corresponding to fabrication and reprocessing, but it has been assumed that the contribution from reprocessing in the years 1980 and 1981 was negligible; the data for the United Kingdom [H8, B12] include exposures during enrichment. Annual average doses to fuel fabrication workers have remained low, in the range of 1-2 mSv. The collective effective dose equivalent distribution ratio for United States workers, which was 0.12 in 1980, decreased to 0.09 in 1981 [N2]; that for British workers was 0 in 1982 and 0.02 in 1983 [B12]; that for Japanese workers was 0 in the period 1981-1984 [T12].

48. Some data on the external doses from the fabrication of plutonium fuel at the PNC works in

Japan have been published [A5]. These are shown in Table 16. During the period 1977-1982 the total amount of fuel fabricated was 37.6 t for an advanced HWR and 1.2 t for an FBR. From 1980 to 1982, it was necessary to process reactor grade plutonium recovered from high burn-up fuel, and this led to an increase in both average and collective doses to the work-force.

49. The estimates of normalized collective effective dose equivalent in the UNSCEAR 1982 Report were considerably reduced from previous estimates; the overall figure estimated to be 1 man Sv $(GW\ a)^{-1}$. More recent estimates are shown in Table 15. The normalized collective effective dose equivalents for Canada and the United Kingdom were obtained by directly relating the collective dose in a year to the electric energy generated in the year [13, 14, 15, 16], as seems appropriate for nuclear power programmes in an approximately equilibrium situation. For the United States the same assumption is made as in the UNSCEAR 1982 Report; 60% of the fuel fabricated is for United States nuclear power stations. For Japan, figures for 1981-1984 are used [J3], and these have been tentatively related to the total electric energy generated in the corresponding years by nuclear power. Giving appropriate weight to more recent data, an overall average of 0.5 man Sv $(GW\ a)^{-1}$ now seems more appropriate.

III. REACTOR OPERATION

50. Nearly all the electric energy generated by nuclear power is produced in thermal reactors in which the fast neutrons produced by the fission process are slowed down to thermal energies by use of a moderator. The most common materials still used for moderators are light water, heavy water and graphite. The choice of moderator and coolant, light or heavy water or carbon dioxide gas, greatly affects the design, size and heat removal system of the reactor.

51. The uranium fuel is contained in discrete pins, both to prevent leakage of the radioactive fission products into the coolant circuit and to improve neutron economy by reducing parasitic neutron captures in the resonance neutron energy region of ^{238}U. The heat generated in the fuel pins by the slowing down of the fission fragments is removed by forced convection, the most usual coolants being light or heavy water or carbon dioxide gas. In the case of fast reactors, the neutrons are not moderated and induce fissions with energies close to those at which they are produced. The usual heat removal system is liquid sodium, which is a good heat transfer medium and does not greatly moderate the neutrons.

A. EFFLUENTS

52. During the production of power by a nuclear reactor, radioactive fission products are formed within the fuel, and neutron activation produces radioactive components in structural and cladding materials. Radionuclides are formed in the coolant circuit because the coolant becomes activated, because of the diffusion of fission product elements with radioactive isotopes from the small fraction of the fuel with defective cladding, and because of the corrosion of structural and cladding materials anywhere in the coolant circuit which leads to particles being carried through the core and becoming activated. All reactors have treatment systems for the removal of radionuclides from gaseous and liquid wastes, which arise from leakage out of the core or from clean-up of the coolant.

53. The quantities of different radioactive materials discharged from reactors depend on the reactor type, its design and the specific waste treatment plant installed. Radionuclides discharged to atmosphere include fission noble gases (krypton and xenon), activation gases (^{14}C, ^{16}N, ^{35}S, ^{41}Ar), tritium, iodine and particulates. Radionuclides released into the aquatic environment in liquid effluents usually include tritium, fission products and activated corrosion products. The discharge data for the years 1980-1985 are presented in this section, and the annual normalized releases are evaluated for each reactor type and averaged over all reactors of each type as TBq $(GW\ a)^{-1}$. Normalized results are not presented for individual sites because releases in any one year may reflect a need for maintenance or irregular procedures which are the culmination of a number of years of previous operation. The total releases of radionuclides between 1980 and 1984 have been normalized by dividing by the total electric energy generated (GW a) over the same period. These normalized releases are used to assess collective dose commitments because the 1985 data were incomplete. Generally, the normalized releases for 1985 from the partial data lead to lower values than for the previous five years, although the 1980-1985 averages are mostly within 10% of the 1980-1984 averages.

1. Fission noble gases

54. At least nine identified radioactive isotopes of krypton and 11 of xenon are formed during fission. Most have half-lives of minutes or seconds and decay before they migrate significantly in the fuel. A fraction of the noble gas inventory of the fuel pins diffuses to the free space between the fuel and the cladding, leading to a build-up of gas pressure. The presence of noble gases in the coolant circuit is generally an indication of fuel cladding failure.

55. Table 17 lists the reported discharges of noble gases from PWRs. The releases span many orders of magnitude partly because of the design of newer plants and partly because of the need for irregular operations and maintenance. Thus, the normalized releases presented are averaged over all PWR electric energy production from 1980 to 1984. Short-lived noble gases only appear in PWR effluents because of leakages in the primary water pressure circuit. Gaseous wastes can also arise from the condenser exhaust on the steam circuit and from blow-downs or contain-

ment purges. These wastes are usually held under pressure in delay tanks to allow decay of short-lived isotopes before release. The isotopic composition of noble gases released from PWRs in the United States in 1982 is shown in Table 18. Comprehensive data are available for each year from United States reactors; and data available from other countries are similar to those from the United States. The data for the United States for 1982 are therefore assumed to be representative of the isotopic composition of releases between 1980 and 1984 and are used for dose estimation.

56. Data for 1985 are incomplete and the releases are not included in the normalized set. The normalized releases seem to have remained fairly steady over the five-year period 1980-1984, but the average of 218 ± 40 TBq $(GW\ a)^{-1}$ appears to be about half of the value reported previously by the Committee [430 TBq $(GW\ a)^{-1}$]. Xenon-133 accounted for 75% of the discharge and ^{135}Xe for 12%. In the UNSCEAR 1982 Report the comparable figures were 85% and 5%, respectively. Some of the reduction in discharges is thought to be due to better fuel can performance, which would account for the lower releases to cooling water. The other feature is the inclusion of newer reactors with lower levels of discharge.

57. Reported discharges of noble gases from BWRs are shown in Table 19. The releases vary by six orders of magnitude, although the average releases continue to have been reduced from previous years. The normalized releases are shown in Table 19 for all BWRs from 1980 to 1985. The main source of noble gas release from BWRs is gases in the steam circuit that are continuously removed by the main condenser air-ejector system. The isotopic composition depends on the hold-up time, which is usually less than for PWRs, thus allowing more short-lived isotopes to be released. Table 20 gives the radionuclide composition of noble gas releases from United States BWRs in 1982, which is similar to that of reactors in other countries. These figures again are taken to be representative of BWR releases in all countries during the period 1980-1984 and are used for dose assessment.

58. For BWRs the average discharge rate for noble gases during the five-year period 1980-1984 is $2,150 \pm 520$ TBq $(GW\ a)^{-1}$ compared with 8,800 TBq $(GW\ a)^{-1}$ reported in the UNSCEAR 1977 Report for 1975-1979. This reduction seems to have been achieved because of significant reductions in releases from those reactors that previously had the highest discharge rates. The normalized release for 1985 is significantly lower (460 TBq $(GW\ a)^{-1}$), partly because of the missing data but mainly because of very large reductions in discharges from the largest previous sources (Browns Ferry and Brunswick). The isotopic composition shown in Table 20 reveals that most of the activity consists of ^{88}Kr (half-life: 2.8 h), ^{133}Xe (half-life: 5.3 d), ^{135}Xe (half-life: 9.2 h) and ^{138}Xe (half-life: 17 min) in almost equal quantities.

59. In GCRs, noble gas releases are insignificant compared with activation gases. Magnox reactors, AGRs, LWGRs and HWRs utilize on-load refuelling and, in the event of fuel element failure, fuel rods can be replaced. Releases of noble gases from HWRs and LWGRs are given in Table 21. Normalized release for HWRs has been 212 ± 48 TBq $(GW\ a)^{-1}$, similar to that for PWRs. The highest figures are for LWGRs at $5,470 \pm 1,370$ TBq $(GW\ a)^{-1}$, about three times the figures for BWRs. The available discharge data indicate that FBRs have lower releases of noble gases; measurements at BN-350, an FBR in the USSR, indicate 65-130 TBq $(GW\ a)^{-1}$ [P6].

2. Activation gases

60. Although GCRs do not generally release fission noble gases, several gases are formed during gas cooled reactor operation. These are primarily ^{41}Ar, formed by activation of the stable argon in air, and ^{35}S produced from sulphur and chlorine impurities in the graphite core. The discharge data for ^{41}Ar are reported in Table 22. For ^{35}S, measurements were made in the United Kingdom at Hinkley B, Oldbury and Wylfa [H1, H2], and discharges have consistently averaged 0.2 TBq $(GW\ a)^{-1}$ with only about 20% variation around the mean.

61. The quantity of ^{41}Ar (half-life: 1.8 h) released depends upon the detailed design of the reactor. For early Magnox reactors having steel pressure vessels, the principal source of ^{41}Ar is the activation of stable argon in the air used as cooling air around the outside of the pressure vessel. For advanced reactors with prestressed concrete pressure vessels, the principal source of ^{41}Ar is leakage of the coolant CO_2, which contains small amounts of air, to the atmosphere. The normalized releases from AGRs are 5-15% of the values for Magnox reactors. The average normalized release from GCRs is $2,320 \pm 220$ TBq $(GW\ a)^{-1}$ compared to 3,240 TBq $(GW\ a)^{-1}$ in the UNSCEAR 1982 Report. The reduction is due to the proportion of power now generated by AGRs and addition of French data. For BN-350, the Soviet FBR, normalized ^{41}Ar releases average 470 TBq $(GW\ a)^{-1}$ [K4, P6].

62. Nitrogen-16 (half-life: 7 s) causes direct external radiation at nuclear power plants. The photons produced in its decay have energies of 6.1 and 7.1 MeV. In BWRs, the ^{16}N generated in the coolant water is transferred in the steam to the turbine buildings. Direct radiation from gas ducts in steel pressure vessel gas cooled reactors produces the major dose to individuals close to those sites.

3. Tritium

63. In LWRs tritium arises from ternary fission in the nuclear fuel and from the neutron activation of lithium and boron isotopes dissolved in, or in contact with, the primary coolant. The Committee assessed in the UNSCEAR 1982 Report the tritium production rate from ternary fission as 0.75 PBq $(GW\ a)^{-1}$. Tritium generation from activation reactions in PWRs seems to result mainly from boron, which is used for reactivity control, in the coolant, whereas in BWRs it results mainly from boron in control rods. In GCRs it is the result of lithium impurities in the graphite and

the presence of water vapour in the core. For HWRs it is principally the result of the activation of the deuterium moderator and coolant. The activation production rate only exceeds that from ternary fission in HWRs, where the activation rate was previously estimated by the Committee to be 30 times higher at about 25 PBq $(GW\ a)^{-1}$.

64. Table 23 presents the tritium releases to the atmosphere for 1980-1985 for PWRs, BWRs and HWRs. For PWRs the normalized release over the five-year period 1980-1984 is 5.9 ± 2.4 TBq $(GW\ a)^{-1}$ and no particular trend is apparent over this period. The corresponding figure was 7.8 TBq $(GW\ a)^{-1}$ for 1975-1979. The BWR releases normalized for the same period average 3.4 ± 1.6 TBq $(GW\ a)^{-1}$, compared with 3.4 TBq $(GW\ a)^{-1}$ for 1975-1979. The decrease in annual BWR normalized releases from 1980 to 1984 seems primarily attributable to reductions from the Dresden nuclear plant alone, while the higher figure for 1985 is due to Hatch 1. These figures indicate that about 1% of the tritium produced in the fuel of LWRs finds its way into the coolant and then enters airborne effluent streams. For HWRs the production of tritium in the moderator is the most probable source of tritium releases, which averaged 670 ± 190 TBq $(GW\ a)^{-1}$ for 1980-1984, as compared with 540 TBq $(GW\ a)^{-1}$ for 1975-1979. For some HWRs, however, the coolant may be the main source of tritium production. There is little release of tritium to the atmosphere from Magnox gas cooled reactors mainly because humidriers remove water vapour from the gas circuit. There is some release of tritium to the atmosphere from AGRs, and the normalized release is 5.4 ± 0.9 TBq $(GW\ a)^{-1}$, similar to LWR releases.

65. From Table 24 it can be seen that releases of tritium to the hydrosphere from PWRs have been fairly constant over the past five years, and the 1980-1984 normalized average is 27 ± 1.8 TBq $(GW\ a)^{-1}$, with the figure for 1985 similar. This compares to the 38 TBq $(GW\ a)^{-1}$ obtained for 1975-1979. The comparable figures for BWRs are 2.1 ± 0.5 TBq $(GW\ a)^{-1}$ for 1980-1984, which is 50% higher than the 1.4 TBq $(GW\ a)^{-1}$ for 1975-1979 and no trend is apparent. For GCRs the normalized release to surface waters is 96 ± 13 TBq $(GW\ a)^{-1}$, which contrasts with 25 TBq $(GW\ a)^{-1}$ for 1975-1979. There appears to have been a significant increase in tritium releases from GCRs over the past five years. HWR releases in liquid effluent streams averaged 290 ± 68 TBq $(GW\ a)^{-1}$ for 1980-1984, compared with 350 TBq $(GW\ a)^{-1}$ for 1975-1979. LWGRs have low liquid releases at 1.7 TBq $(GW\ a)^{-1}$.

66. Thus, about 0.3% of BWR tritium production appears in liquid effluents, with a similar amount going to the atmosphere. For PWRs about 3% of the tritium produced is in liquid effluents, about five times more than that going to the atmosphere. For HWRs liquid effluents are about one half those discharged to the atmosphere.

67. For PWRs and LWGRs in the USSR the atmospheric releases of tritium are reported to average 7.4 TBq $(GW\ a)^{-1}$ and 1.9 TBq $(GW\ a)^{-1}$, respectively, [B6, V4], similar to other PWRs and AGRs. The liquid discharges amount to about 5 TBq $(GW\ a)^{-1}$ and 1 TBq $(GW\ a)^{-1}$ for PWRs and LWGRs, respectively [B6, P6, V4]. Measurements indicated that on average 90% of the atmospheric releases of tritium was in oxide form [B6]. Practical experience at the Novovoronezh APS (PWR) showed that it is possible to reduce the tritium concentration in the coolant water by 50% [B7].

4. Carbon-14

68. Discharges of ^{14}C are of interest because of its long half-life (5,730 a) and contribution to collective dose commitments. Estimates of ^{14}C production in fuels depend on the nitrogen level in the fuel can, although some is produced from reactions on oxygen in oxide fuels. The Committee concluded in the UNSCEAR 1982 Report that the normalized production rate within fuel for PWRs, BWRs, GCRs and HWRs was close to 1 TBq $(GW\ a)^{-1}$. Little of this is released into the reactor coolant circuits, it appears to be released during reprocessing [B1, B2, B3, B8, B16, B29]. Carbon-14 is produced in the moderators of all reactors, production in HWRs being perhaps 100 times greater than in LWRs or GCRs, because of the ^{17}O (n, α) ^{14}C reaction in the greater mass of oxygen in the moderator, and there is a consequential release.

69. The National Council on Radiation Protection and Measurements (NCRP) [N1] has estimated the production rate of ^{14}C in PWRs to be between 2 and 3 TBq $(GW\ a)^{-1}$, and for BWRs 3-4 TBq $(GW\ a)^{-1}$, arising in both cases mainly in stainless steel and zirconium alloy. For the estimation of release rates to the environment, NCRP assumes that the ^{14}C formed in the hardware remains there, but that the fraction formed in dissolved nitrogen in the cooling water is totally released. The NCRP estimate for PWRs is 370 GBq $(GW\ a)^{-1}$ and for BWRs 220 GBq $(GW\ a)^{-1}$. The NCRP estimate of the release of ^{14}C to the environment for FBRs is essentially zero at the reactor.

70. Environmental discharges of ^{14}C are not routinely reported for all reactors. The data summarized in Table 25 are from a series of measurements made in Argentina, the Federal Republic of Germany, Finland, and USSR. For BWRs it appears that essentially all the ^{14}C appears as carbon dioxide, and the normalized release rate for 1980-1984 is 330 ± 110 GBq $(GW\ a)^{-1}$, significantly less than the Committee's estimate in the UNSCEAR 1982 Report of 520 GBq $(GW\ a)^{-1}$. For PWRs in the Federal Republic of Germany [W1], Finland [B17] and the USSR [R1], the data indicate a release rate of 345 ± 80 GBq $(GW\ a)^{-1}$, which is significantly higher than the figure of 220 GBq $(GW\ a)^{-1}$ given in the UNSCEAR 1982 Report. For PWRs only about 5-50% of the emission appears as CO_2. It now appears that normalized ^{14}C releases from PWRs and BWRs are similar.

71. In recent measurements at three LWRs in the United States [K2], two PWRs emitted an average of 390 GBq $(GW\ a)^{-1}$. The source of ^{14}C was different at

the two sites: the first had 42% arising from venting of gas decay tanks, 35% from auxiliary building ventilation and 32% from containment venting; the second had emissions resulting primarily from pressure relief venting and purging of the containment air, with only 7% from venting of gas decay tanks. For the BWR, the discharge rate was 460 GBq (GW a)$^{-1}$ with 97% of the release via the off-gas discharge, which was 95% $^{14}CO_2$. For the PWR 94% of the discharge was $^{14}CH_4$. The ^{14}C content of liquid and solid wastes was less than 5% of the aerial discharge for all reactors.

72. Measurements at LWGRs in the USSR gave average releases of 1.3 TBq (GW a)$^{-1}$ [R1]. In the United Kingdom, reported releases were 0.74 TBq (GW a)$^{-1}$ from Magnox reactors and 1.9 TBq (GW a)$^{-1}$ from AGRs. Weighted by energy production, the normalized ^{14}C release for GCRs is 1.1 TBq (GW a)$^{-1}$ [H8]. The main source of ^{14}C releases from GCRs is the leakage of the primary coolant, at a rate typically of a few per cent per day, which contains radionuclides released to the coolant by corrosion of the graphite moderator.

73. For HWRs it has been reported that a significant fraction of the inventory formed in the moderator can be released to atmosphere. Measurements at Atucha 1 [B18, O3], however, for 1983-1985 have indicated that releases are significantly lower than previously calculated for 1980-1982. The five-year normalized release is 6.3 ± 3.3 TBq (GW a)$^{-1}$, whereas the Committee had estimated 17 TBq (GW a)$^{-1}$ in the UNSCEAR 1982 Report. The form is again variable, between 40 and 80% being reported as CO_2. In Argentina, regular monitoring of discharges of ^{14}C has continued for several years so that more reliable estimates can be made.

5. Iodine

74. The volatile element iodine is produced by the fission process, the isotopes of radiological interest being ^{129}I (half-life: 1.6 10^7 a), ^{131}I (half-life: 8.04 d), ^{132}I (half-life: 2.3 h), ^{133}I (half-life: 21 h), ^{134}I (half-life: 53 m) and ^{135}I (half-life: 6.6 h). Because, apart from ^{129}I, the iodine isotopes have such short half-lives, equilibrium activity concentrations in the fuel are reached quickly and releases depend on the number of fuel cladding failures and the coolant leakage rate. Iodine has been studied for many years in view of its mobility in the environment and selective thyroid irradiation. Because of its long half-life, ^{129}I is of interest in evaluating collective dose commitments; however, its release from reactors is very small and often not reported. Most of ^{129}I in fuel is released during reprocessing, from which it makes a greater contribution than from reactor operation.

75. Table 26 gives the reported atmospheric discharges of ^{131}I from operating reactors in various countries for 1980-1985. There are considerable differences in the absolute quantities; these appear to be attributable to differences in the ages of the plants and in the waste treatment designs. There does not appear to be any trend in PWR releases, but BWR normalized data show a sharp downward trend.

76. The annual normalized discharges of ^{131}I from PWRs were 1.75 ± 0.33 GBq (GW a)$^{-1}$ for 1980-1984, not significantly changed when compared with 1.9 GBq (GW a)$^{-1}$ for 1975-1979. The ^{131}I releases from BWRs for 1980-1984 have averaged 9.3 ± 4.9 GBq (GW a)$^{-1}$ compared with ^{131}I releases of 40 GBq (GW a)$^{-1}$ for 1975-1979. This reduction was because the few reactors that had large releases are currently releasing far less. The results for HWRs indicate releases of 0.23 ± 0.08 GBq (GW a)$^{-1}$. From early GCRs, which utilize on-load refuelling, releases were negligible, and releases from AGRs were 1.4 ± 1.1 GBq (GW a)$^{-1}$. LWGRs released 80 ± 40 GBq (GW a)$^{-1}$ [A1], and measurements indicated that 60% of the iodine in the reactor off-gases was in organic form, 40% inorganic and about 1% particulate [B9, D1, S6].

77. The isotopic composition of iodine releases from LWRs in the United States in 1982 is shown in Table 27 [T5]. The isotopic composition was taken to be representative of reactor operations in all countries and was used as the basis for dose calculations. For PWRs about 25% of the discharge is accounted for by ^{131}I and 75% by ^{133}I, compared with the figures reported by the Committee in the UNSCEAR 1982 Report of 30% accounted for by ^{131}I. For BWRs, ^{131}I releases represented about 7% of the discharges, with ^{133}I and ^{135}I contributing 28% and 65%, respectively. This compares with less than 10% previously reported for ^{131}I and 30% and 60% for ^{133}I and ^{135}I, respectively. For LWGRs, 24% is accounted for by ^{131}I, 43% by ^{133}I and 33% by ^{135}I [B21]. It might be concluded that there was little change in the isotopic composition in the periods 1975-1979 and 1980-1984.

6. Particulates in airborne effluents

78. Radionuclides in particulate form can arise directly or as decay products of fission noble gases or may arise from corrosion of materials in the primary coolant circuit. Aerosols are generated because of primary circuit leaks or because of maintenance work on active components removed from the primary circuit. The air in all areas where aerosols might arise is continually purged and the plenum activity is filtered by high efficiency particulate (HEPA) filters. Results of recent measurements on particle size distributions indicate a mean aerodynamic diameter of 1 μm for fission products and 10μm for activation products [B4]. Measurements at LWGRs in the USSR have indicated mean aerodynamic diameters of 0.1-0.4 μm for particulates; for ^{131}I and ^{51}Cr, 30-40% were particulates with a mean aerodynamic diameter of less than 0.1 μm [B10, C2].

79. Releases of particulate activity to the atmosphere are summarized in Table 28 for reactors around the world. The quantities are extremely low, and the nuclide composition appears to be unique to each operating plant; it depends on the particular impurities in cladding and structural materials, coolant chemistry and fuel failure modes. The isotopic composition of the release from a plant can vary from year to year, because of different operational and maintenance needs. Consequently, the range of nuclides reported in

atmospheric discharges is extremely large, several tens of nuclides often being reported from one plant. No single nuclide can be identified as contributing the majority of the activity released for any one type of reactor. Radionuclides identified include ^{7}Be, ^{22}Na, ^{51}Cr, ^{54}Mn, ^{59}Fe, ^{57}Co, ^{58}Co, ^{60}Co, ^{63}Ni, ^{65}Zn, ^{76}As, ^{88}Rb, ^{89}Sr, ^{90}Sr, ^{91}Sr, ^{95}Zr, ^{97}Zr, ^{95}Nb, ^{99}Mo, ^{99m}Tc, ^{103}Ru, ^{105}Ru, ^{106}Ru, ^{108m}Ag, ^{110m}Ag, ^{113}Sn, ^{115}Cd, ^{122}Sb, ^{124}Sb, ^{125}Sb, ^{123m}Sn, ^{123m}Te, ^{134}Cs, ^{137}Cs, ^{139}Ce, ^{140}Ba, ^{140}La, ^{141}Ce, ^{144}Ce and ^{182}Ta.

80. For PWRs the normalized release was 4.5 ± 2.9 GBq $(GW\ a)^{-1}$ for 1980-1984, compared with 2.2 GBq $(GW\ a)^{-1}$ for 1974-1979. For BWRs, the average release was 43 ± 24 GBq $(GW\ a)^{-1}$, compared with 53 GBq $(GW\ a)^{-1}$ for 1974-1979. For HWRs the data yield 0.04 ± 0.016 GBq $(GW\ a)^{-1}$, similar to the 0.044 GBq $(GW\ a)^{-1}$ normalized release reported previously, while for LWGRs the average release appears to have been 15.7 ± 16.2 GBq $(GW\ a)^{-1}$. There were no figures previously for LWGRs, nor were there any for GCRs, which now average 1.4 ± 0.8 GBq $(GW\ a)^{-1}$.

7. Liquid effluents

81. The sources of radionuclides other than tritium in liquid effluents are essentially the same as those described for particulate releases to the atmosphere. The reported levels of discharge are equally variable, the magnitude and isotopic composition depending upon the design and operating practice of the reactor, impurity levels and trace quantities of material in structural and cladding components. Table 29 summarizes reported liquid effluent discharges from reactors around the world. In Table 30 the isotopic composition of liquid discharges from power reactors in the United States in 1982 is presented, and in Table 31, that for GCRs in the United Kingdom is given, also in 1982.

82. The normalized release levels based on the reported discharges for each reactor type using reported figures for electric energy generated between 1980 and 1984 can be summarized from Table 29 and contrasted with the figures given in the UNSCEAR 1982 Report.

PWR: 132 ± 49 GBq $(GW\ a)^{-1}$,
compared with 180 GBq $(GW\ a)^{-1}$
BWR: 115 ± 47 GBq $(GW\ a)^{-1}$,
compared with 290 GBq $(GW\ a)^{-1}$
GCR: $4{,}520 \pm 1{,}790$ GBq $(GW\ a)^{-1}$,
compared with 4.800 GBq $(GW\ a)^{-1}$
HWR: 25.7 ± 8.7 GBq $(GW\ a)^{-1}$,
compared with 470 GBq $(GW\ a)^{-1}$

The normalized releases for PWRs between 1980 and 1984 are similar to previous years although there has been an increasing trend, while BWR releases are less. Canadian HWRs were previously reported as giving discharges of about 50 GBq $(GW\ a)^{-1}$, while the higher figures for the GCRs reflect the fact that discharges are made, with the exception of Trawsfynydd, to the marine environment. It appears from the above results that aquatic discharges from BWRs have been reduced by a factor of 2.5. In the UNSCEAR 1982 Report, the Committee found that PWR releases had been reduced by a factor of about 2 and BWR releases by a factor of 10 from the figures given in its UNSCEAR 1977 Report. These reductions do not seem to have been attributable to the removal of any single nuclide but are applicable to all nuclides in the release.

83. The isotopic composition of liquid effluents from United States reactors in 1982 is shown in Table 30. About 20% of the normalized PWR discharge is due to ^{58}Co and almost 20% to ^{131}I, while ^{137}Cs accounts for about 11%. In the BWRs about 30% of the release is due to ^{60}Co and about 13% to ^{137}Cs, the other nuclides with significant contributions being ^{24}Na and ^{65}Zn; ^{131}I contributed about 3%. These figures represent small changes from those in the UNSCEAR 1982 Report, with some reduction in the percentages of caesium.

84. For GCRs, 40% of the discharges to the aquatic environment are due to ^{137}Cs and the ratio of ^{134}Cs to ^{137}Cs is 0.22, compared with 0.6 for PWRs and 0.5 for BWRs, which reflects differences in fuel burn-up. About 16% of GCR releases is due to ^{35}S, and ^{90}Sr accounts for about 6%.

85. There is a wide range of activation products and fission products reported in liquid effluent discharges, and the isotopic composition varies even between reactors of the same type. The normalized figures are used, however, to make an estimate of the collective doses due to liquid effluent discharges.

B. LOCAL AND REGIONAL COLLECTIVE DOSE COMMITMENTS

86. National authorities usually require environmental monitoring programmes in the vicinity of a nuclear power plant to be carried out by the operator, another competent agency or both. In general, activity concentrations of radioactive materials from effluent discharges are too low to be measurable except close to the immediate point of discharge. Dose estimates for the population, therefore, rely on modelling the environmental transfer and transport of radioactive materials.

87. In the UNSCEAR 1982 Report, the Committee established a model site that was most representative of areas of northern Europe and north-eastern United States, since those areas contain a large proportion of the power-producing reactors. Agricultural production patterns and population distributions typical of those areas were also established. The cumulative population within 2.000 km of the site is about $2.5\ 10^{8}$, giving an average population density of 20 km^{-2}. Within 50 km of the site, the population density was taken to be 400 km^{-2} in order to to reflect current siting practice. The objective of the Committee remains unchanged to give a representative value of the collective dose commitments per unit of electric energy generated by nuclear power stations and to reflect the levels of dose received by the most exposed individuals. The results do not apply to any one reactor or any one location, and the collective dose

commitments should not be applied to a given reactor with known discharge data to obtain estimates of health detriment.

1. Fission noble gases

88. Using the normalized releases for PWRs from Table 17 for noble gas atmospheric releases and the radionuclide composition from Table 18, the normalized collective effective dose equivalent commitments averaged between 1980 and 1984 were calculated for the model PWR facility and are shown in Table 32. The normalized release term from Table 17 is 218 TBq (GW a)$^{-1}$, and the radionuclides that contribute significantly to the collective effective dose are ^{133}Xe and ^{135}Xe. The in-growth of daughter products, e.g., ^{88}Rb from ^{88}Kr, has been included in the dose calculations, which are those presented in the UNSCEAR 1982 Report, but scaled for the different normalized release and isotopic composition.

89. The normalized collective effective dose equivalent commitment amounts to 2.6 10^{-2} man Sv (GW a)$^{-1}$ compared with the Committee's assessment of 4.2 10^{-2} man Sv (GW a)$^{-1}$, which was given in Annex F of the UNSCEAR 1982 Report. This reflects the reduction in discharges with little difference in the distribution of radionuclide composition. About 64% of the total collective dose is now due to ^{133}Xe (80% in 1982) and 28% to ^{135}Xe (11% in 1982). As in the UNSCEAR 1982 Report, some 90% of the collective dose commitment is accumulated within 500 km. There is little contribution from the inhalation of radioactive daughter products, and the dose estimates, as before, include an allowance for the shielding from buildings and the fraction of time spent indoors.

90. For the quinquennium 1980-1984, Table 19 shows the normalized releases from BWRs to be 2,150 TBq (GW a)$^{-1}$, compared with the value of 8,800 TBq (GW a)$^{-1}$ given in the UNSCEAR 1982 Report. Taking the relative isotopic composition from Table 20, the normalized collective effective dose equivalent commitment is given in Table 33 as 0.56 man Sv (GW a)$^{-1}$, compared with the Committee's estimate in the UNSCEAR 1982 Report of 1.9 man Sv (GW a)$^{-1}$. The main isotope contributing to the collective dose is ^{88}Kr (half-life: 2.8 h) accounting for about 57%, somewhat more than in 1982. Most of the remainder of the collective dose arises from ^{135}Xe (16%), ^{138}Xe (9%) and ^{133}Xe (8%), in somewhat smaller proportions than in the UNSCEAR 1982 Report.

91. The in-growth of ^{88}Rb (half-life: 15.4 min) from ^{88}Kr and ^{138}Cs (half-life: 32.2 min) from ^{138}Xe decays are included in the dose estimation, and the collective doses include a contribution from the inhalation of the ^{88}Rb and ^{138}Cs radioisotopes. The spatial distribution of the normalized collective effective dose equivalent commitment is biased towards the source, with more than 80% of the dose accumulated within 50 km and nearly 50% within 10 km. This behaviour is caused by the dominance of ^{88}Kr, which decays with a half-life corresponding to about 40 km distance travelled.

92. The normalized release of noble gases from HWRs is 212 TBq (GW a)$^{-1}$ (Table 21), and assuming the same relative isotopic composition as PWRs, the normalized collective dose commitment is 0.024 man Sv (GW a)$^{-1}$, while for LWGRs a normalized release of 5,470 TBq (GW a)$^{-1}$ (Table 21) and the assumption of an isotopic composition similar to that of BWRs yield a normalized collective effective dose equivalent commitment of 0.72 man Sv (GW a)$^{-1}$.

93. In summary, the normalized collective effective dose equivalent commitment from noble gas releases is 0.20 man Sv (GW a)$^{-1}$, based on the five-year (1980-1984) weighting of electricity generated by PWRs, BWRs, HWRs and LWGRs. The Committee gave a figure of 0.63 man Sv (GW a)$^{-1}$ in the UNSCEAR 1982 Report, so that an average reduction of dose from noble gas effluents of about a factor of 3 has been found owing to reductions in reported discharge levels, mainly from BWRs. The annual average effective dose equivalent to the most exposed individuals in hypothetical critical groups has been calculated at 10 μSv for the model BWR and more than 10 times lower for the PWR site, taking an average distance of about 1 km from the site. Many reactors give lower doses, although for some early BWRs, the doses could be about 10 times higher.

2. Activation gases

94. The primary activation product of interest for gaseous releases is ^{41}Ar. Because of its short half-life (1.83 h), it contributes most of its collective dose within a few tens of kilometres of the release point, although the exact result depends on the close-in population density. The normalized release of ^{41}Ar from GCRs (Table 22) between 1980 and 1984 is 2,320 TBq (GW a)$^{-1}$, and the associated collective effective dose equivalent commitment is 0.78 man Sv (GW a)$^{-1}$, compared with the estimate in the UNSCEAR 1982 Report of 0.95 man Sv (GW a)$^{-1}$. The reduction is due to the fact that new AGRs are producing electricity with much lower ^{41}Ar discharges than GCRs. The weighted collective effective dose equivalent commitment, allowing for the fraction of electricity generated by GCRs, is 0.039 man Sv (GW a)$^{-1}$, significantly lower than the value given in the UNSCEAR 1982 Report of 0.1 man Sv (GW a)$^{-1}$. Because of reporting procedures, ^{41}Ar releases for LWRs are included in noble gas data as shown in Tables 18 and 20.

95. The consequences of the release of ^{35}S from GCRs have been studied in detail. The isotope is released in the form of carbonyl sulphide (COS), which has a low deposition velocity and a slow reaction rate in air. The major route of human exposure is via milk, and the Committee estimated 2.2 10^{-4} man Sv GBq^{-1} in the UNSCEAR 1982 Report, so that, using the normalized release of 200 GBq (GW a)$^{-1}$, the normalized collective effective dose equivalent commitment is 0.044 man Sv (GW a)$^{-1}$ and the contribution weighted for GCR electricity production is 2.4 10^{-3} man Sv (GW a)$^{-1}$, compared with 3.8 10^{-3} man Sv (GW a)$^{-1}$ in the UNSCEAR 1982 Report.

3. Tritium

96. The collective effective dose equivalent commitment to the local and regional population was evaluated in the UNSCEAR 1982 Report on the basis of a specific activity model. For atmospheric releases the Committee obtained 1.5 10^{-3} man Sv TBq^{-1} by inhalation and 9 10^{-3} man Sv TBq^{-1} by ingestion to give a total of 0.011 man Sv TBq^{-1} released.

97. Normalized tritium atmospheric releases for the quinquennium 1980-1984 from PWRs are 5.9 TBq $(GW\,a)^{-1}$ (paragraph 64), giving 0.065 man Sv $(GW\,a)^{-1}$; BWR releases of 3.4 TBq $(GW\,a)^{-1}$ give 0.037 man Sv $(GW\,a)^{-1}$; HWR releases of 670 TBq $(GW\,a)^{-1}$ give 7.4 man Sv $(GW\,a)^{-1}$ for atmospheric releases. Releases from GCRs and LWGRs are comparable with PWRs and give similar dose contributions. In summary, weighted by the amount of electricity generated by reactor type, the normalized collective effective dose equivalent commitment for atmospheric releases of tritium is 0.53 man Sv $(GW\,a)^{-1}$, compared with the Committee's estimate given in the UNSCEAR 1982 Report of 0.46 man Sv $(GW\,a)^{-1}$. For the model site used by the Committee, the annual individual effective dose equivalent for critical groups would be less than 1 μSv from LWR atmospheric ^{3}H releases, while the HWR dose would be 50 μSv per year.

98. For tritium in liquid effluents, the river model used by the Committee gave a collective effective dose equivalent commitment of 8.1 10^{-4} man Sv TBq^{-1}, on the assumption that the river is used as a source of drinking water. Using the normalized discharges for 1980-1984 for PWRs, BWRs, HWRs, GCRs and LWGRs from paragraph 65, the normalized collective effective dose equivalent commitments were calculated and are shown in Table 34. The normalized dose weighted by the proportion of electricity generated by each reactor type is 0.033 man Sv $(GW\,a)^{-1}$, which compares with the estimate of 0.04 man Sv $(GW\,a)^{-1}$ given in the UNSCEAR 1982 Report. The doses from aquatic discharges are therefore about 16 times lower than for atmospheric effluents per unit electric energy generated, similar to the difference of a factor of 10 reported in the UNSCEAR 1982 Report.

4. Carbon-14

99. The local and regional collective doses attributable to ^{14}C releases from reactors represent only a small proportion of the total dose commitments. The main significance of ^{14}C stems from its entry into the carbon cycle and the resulting global dispersion, leading to long-term irradiation, which is considered in chapter V. The first pass local and regional collective dose commitment was previously assessed by the Committee using the specific activity approach which was also used for tritium. The Committee also assumed in the UNSCEAR 1982 Report that the form of release of ^{14}C was CO_2. The normalized local and regional collective effective dose equivalent commitment per unit release previously determined by the Committee was 1.8 man Sv TBq^{-1} for ingestion and 0.0003 man Sv TBq^{-1} for inhalation following release to the atmosphere. The normalized doses per unit electric energy generated are shown in Table 35 and are based on the normalized releases taken from Table 25.

100. The normalized collective doses ranged from 0.59 man Sv $(GW\,a)^{-1}$ for BWRs to over 11 man Sv $(GW\,a)^{-1}$ for HWRs. The weighted average, allowing for the proportion of electricity generated by each reactor type, was 1.6 man Sv $(GW\,a)^{-1}$ to the local and regional population. This is about one half of the estimate of 2.8 man Sv $(GW\,a)^{-1}$ given in the UNSCEAR 1982 Report, largely because of lower reported HWR releases. For the model site the annual effective dose equivalents to most exposed individuals was 3 μSv for PWRs and BWRs, 10 μSv for GCRs, about 70 μSv for HWRs and about 13 μSv for LWGRs.

5. Iodine

101. Releases of radioactive iodine from nuclear power plants are small, and there is little contribution to the local and regional collective effective dose equivalent commitment. Because of its long radioactive half-life, ^{129}I enters the global cycle for iodine and has the potential to irradiate the global population for tens of millions of years. The release of ^{131}I contributes only to the local and regional collective doses, but its assessment is complicated by the chemical form in which the iodine is released, i.e., elemental, organic or particulate. Elemental iodine readily deposits on vegetation and enters terrestrial food chains. The deposition of organic iodine is usually less than 1% of that for elemental iodine per unit time integrated air concentration. In this Annex, as in the UNSCEAR 1982 Report, the Committee assumes that 75% of the iodine released is in organic form and 25% in elemental form.

102. In the dose evaluation used in the UNSCEAR 1982 Report, the collective effective dose equivalent commitment per unit release of ^{131}I was 4.0 10^{-4} man Sv GBq^{-1}. Taking the releases of ^{131}I from Table 26, the normalized collective doses for ^{131}I per unit of electric energy generated were calculated and are shown in Table 36. Results for the other iodine isotopes are found by scaling from the results in the UNSCEAR 1982 Report, allowing for the change in isotopic composition. The PWR figures are about 10% lower than in the UNSCEAR 1982 Report, while the BWR results are about 25% of those found previously. For both BWR and LWGR reactors the short-lived ^{133}I and ^{135}I make significant additions to the dose. The weighted average, taking into account the proportion of electricity generated by each reactor type, is 3.3 10^{-3} man Sv $(GW\,a)^{-1}$. Representative effective doses to individuals about 1 km from the model site are about 0.5 μSv per year for PWR releases and 4 μSv per year for the BWR releases. As in the UNSCEAR 1982 Report, 90% of the collective dose contribution is estimated to come from the milk pathway.

6. Particulates in airborne effluents

103. As noted in paragraph 79, the quantities of radionuclides in particulate releases to the atmosphere may vary greatly, even if releases from reactors of the

same type or those from the same reactor from year to year are compared. Furthermore, there are several tens of radionuclides identified in the releases. The solution previously adopted by the Committee for estimating doses was to assume that the normalized releases are composed of equal amounts of activity concentration from a range of nuclides most frequently reported in atmospheric discharges.

104. Dosimetric calculations allowed for transfer through foodchains to man as well as external irradiation from deposited radionuclides and inhalation from the dispersing plume of activity. Allowance was made for uptake by roots of growing vegetation. The full environmental modelling and resultant doses were described in the UNSCEAR 1982 Report. The nuclides considered were ^{51}Cr, ^{54}Mn, ^{59}Fe, ^{58}Co, ^{60}Co, ^{65}Zn, ^{89}Sr, ^{90}Sr, ^{90}Y, ^{95}Zr, ^{95}Nb, ^{124}Sb, ^{134}Cs, $136^{136}Cs$, $137^{137}Cs$, ^{140}Ba, ^{140}La, ^{141}Ce and ^{144}Ce.

105. The collective effective dose equivalent commitments per GBq release of the isotopic mixture is taken from the UNSCEAR 1982 Report to be 5.4 10^{-3} man Sv $(GBq)^{-1}$, with nearly two thirds coming from external radiation from deposited activity and one third from ingestion. The collective doses per unit energy generated have been calculated using the normalized releases from Table 28 and are shown in Table 37, from which it can be seen that the most important pathway is the external dose received from activity deposited on the ground, followed by the dose from ingested foodstuffs. The normalized doses cover three orders of magnitude, with HWRs giving the lowest figure of 0.00022 man Sv $(GW\ a)^{-1}$ and BWRs the highest value of 0.23 man Sv $(GW\ a)^{-1}$. The doses can be compared with the previous figure of 0.012 man Sv $(GW\ a)^{-1}$ for PWRs, about one half of the current estimate. For BWRs the previous figure of 0.29 man Sv $(GW\ a)^{-1}$ was slightly higher than the present value. GCR figures are significantly lower than before (0.007 man Sv $(GW\ a)^{-1}$ compared with 0.012 man Sv $(GW\ a)^{-1}$).

106. Some 95% of the collective effective dose equivalent commitment from ground deposits is delivered within 50 years of the deposition and the major nuclides contributing are ^{137}Cs and ^{60}Co. For ingestion, ^{90}Sr, ^{134}Cs and ^{137}Cs all contribute equally by three pathways: grain, vegetables and meat. The normalized collective effective dose equivalent commitment, weighted for the proportion of electricity produced by each reactor type, is 0.08 man Sv $(GW\ a)^{-1}$, essentially the same as the estimate of 0.1 man Sv $(GW\ a)^{-1}$ in the UNSCEAR 1982 Report. Individual effective dose equivalents from the normalized releases at the end of a plant's operating lifetime range from about 0.01 μSv at 1 km from the model BWR to 1,000 times less for HWRs.

7. Liquid effluents

107. Aquatic releases are made into freshwater or marine environments. For releases of radionuclides into rivers or lakes, the pathways of exposure were previously taken by the Committee to be drinking water, irrigation leading to transfer to foodstuffs, and external radiation from sediments. For discharges to marine environments it is usually sufficient to consider the ingestion of ocean fish and crustacea. In the UNSCEAR 1982 Report pathways such as swimming in contaminated waters or consumption of unusual food items were considered to contribute little to the collective dose commitment.

108. The Committee has recognized the difficulty in assigning values to parameters in assessing the consequences of liquid effluents, in particular, water utilization and flow rates for rivers, fish production rates and sedimentation rates. The assessments based on the model used in the UNSCEAR 1982 Report, therefore, must be regarded as merely giving a representative value of nuclear power impact and should not be applied to discharges from a specific site to estimate collective doses from that site.

109. The normalized releases for PWRs, BWRs, GCRs, HWRs and LWGRs for 1980-1984 were summarized in paragraph 82, and the isotopic composition for these discharges were assumed to be those of United States reactors shown in Table 30 and those of United Kingdom reactors in Table 31. Collective effective dose equivalent commitments were evaluated assuming the discharges took place to freshwater and to marine environments. The results are shown in Tables 38 and 39.

110. The normalized collective effective dose equivalent commitment for releases from the PWR to fresh water was 1.6 10^{-3} man Sv $(GW\ a)^{-1}$, compared with the finding of 1 10^{-3} man Sv $(GW\ a)^{-1}$ in the UNSCEAR 1982 Report. Drinking water accounted for about 80% of the total and ^{131}I was the major contributing nuclide (70% of the dose). For BWRs the normalized collective effective dose equivalent is 6.6 10^{-4} man Sv $(GW\ a)^{-1}$, compared with the assessment of 2.8 10^{-3} man Sv $(GW\ a)^{-1}$ in the UNSCEAR 1982 Report. The reduction is the result of an overall decrease of discharge, as well as a greater reduction in the more significant radiological nuclides. About 75% of the dose was from drinking water, and ^{60}Co, ^{131}I, ^{134}Cs and ^{137}Cs contributed almost equally to this dose. No results were provided for GCRs since these were coastal-sited. The collective dose from these radionuclides normalized for the amount of electricity generated were 1.1 10^{-3} man Sv $(GW\ a)^{-1}$ for PWR and BWR releases to the model river.

111. For PWR releases to salt water the normalized results are shown in Table 39. The collective effective dose equivalent commitment was 3.6 10^{-3} man Sv $(GW\ a)^{-1}$, about one half the value found in the UNSCEAR 1982 Report. The fish and mollusc pathways were both equally important, although the most important nuclide was different for each pathway: ^{137}Cs for fish and ^{60}Co for molluscs.

112. For BWR releases to the marine environment, the normalized collective effective dose equivalent commitment was 3.8 10^{-2} man Sv $(GW\ a)^{-1}$, compared with the figure of 4.2 10^{-2} man Sv $(GW\ a)^{-1}$ given in the UNSCEAR 1982 Report. The major contribution, was, as before, from ^{65}Zn, which concentrates in

molluscs, and therefore the marine results differed markedly from those for fresh water. For GCR releases the normalized collective effective dose equivalent commitment was 0.19 man Sv (GW a)$^{-1}$, essentially the same value found in the UNSCEAR 1982 Report. The majority of the dose arose from discharges of ^{137}Cs. The weighted normalized collective effective dose equivalent commitment, allowing for the respective electricity generation was 0.025 man Sv (GW a)$^{-1}$.

113. Again, it should be emphasized that the figures given in Tables 38 and 39 are representative of the generation of unit quantity of electric energy and should not be applied to a specific site where particular releases and specific environmental pathways exist that have not been considered here and might lead to significant differences in collective dose contributions. The normalized collective effective dose equivalent commitment due to aquatic discharges has been estimated to be 0.013 man Sv (GW a)$^{-1}$, assuming that one half the discharges are made to fresh water and one half to the marine environment.

C. OCCUPATIONAL EXPOSURES

114. As was noted in the UNSCEAR 1982 Report, more data on occupational exposure to radiation are reported for reactor operation than for any other area. There are difficulties in normalizing data on occupational exposure to the electric energy generated, particularly for water reactors, as most of the doses are incurred during maintenance when no energy is produced. Normalized results are therefore only usefully derived over several years for a number of reactors. Average annual effective dose equivalents to reactor workers were estimated to be similar in the UNSCEAR 1977 and 1982 Reports and ranged from 3 to 8 mSv. During the same period, however, there was a large increase in the number of workers per reactor in the United States. The trend in the normalized collective effective dose equivalent was downwards, but overall the best estimate for LWRs was taken in the UNSCEAR 1982 Report to be 10 man Sv (GW a)$^{-1}$, the same as in the UNSCEAR 1977 Report.

115. As can be seen from Table 1, PWRs and BWRs have been installed in many countries, although installed capacity in 1987 was still dominated by the United States. Recent data on occupational exposure and normalized collective effective dose equivalents are given in Table 40 for PWRs and BWRs. For some countries, the data are comprehensive and published regularly by the appropriate authorities. For other countries, data are not available for all years or all the units installed. None the less, the data are sufficient to give a reasonably comprehensive indication of the situation world wide, as substantial numbers of LWRs enter the middle phase of their predicted operating lifetimes. In general, the data on electricity generated were taken from the summaries produced by the International Atomic Energy Agency (IAEA) [I3, I4, I5, I6], if not otherwise given in the references for a particular country.

116. A comprehensive survey of data on LWRs in Western Europe has been carried out [B33]. The data cannot be added to Tables 40 or 41, as the reactors were not identified specifically by country. The normalized collective effective dose equivalent for 23 PWRs dropped from about 6 man Sv (GW a)$^{-1}$ in 1980-1981 to 4 man Sv (GW a)$^{-1}$ in 1984. The comparable figures for 17 BWRs were more variable but were in the range of 3-6 man Sv (GW a)$^{-1}$. A particular study on PWRs has also been carried out by Lochord and Benedittini [L5]. A distinct difference is emerging between PWRs and BWRs in the annual collective dose per reactor and per unit electric energy generated for reactors of similar electrical capacity. This trend, which is illustrated in Table 41 for PWRs and BWRs from the United States and Japan, becomes more apparent as the reactors enter the second decade of their operating life and has been reported in the Federal Republic of Germany [E7], Japan [I9, T12], Sweden [P7] and the United States [N3]. The collective western European data, however, do not support the conclusion [B33]. The collective dose can be higher in BWRs than in PWRs by up to a factor of 2, possibly because more maintenance work has to be performed in radiation areas on BWRs, especially around the turbines.

117. It appears that the significant trend towards increasing numbers of workers per reactor, which was noted in the UNSCEAR 1982 Report, especially in the United States, levelled off in the early 1980s. This aspect has been studied in detail by the Nuclear Regulatory Commission [N3], who showed that although the number of workers per reactor doubled from 600 to 1,200 over the period 1975-1980, it remained constant at the higher figure during the period 1980-1983. The collective effective dose equivalent distribution ratio (see paragraph 19) was assessed separately for PWRs and BWRs [N3]. For both types of reactors the average values of the ratios for the years 1981-1983 were in the range of 0.4-0.6. Annual average doses have been quoted for the years 1980-1982 at three PWRs in the USSR [V2, V3]. The values range from 4-8 mSv and provide detailed support for the overall average figure of 5.6 mSv given by Varobyov [V1] and used in Table 40. For the Novovoronezh PWR [V5], however, the normalized collective effective dose equivalent in 1980 of 3.1 man Sv (GW a)$^{-1}$, based on an annual collective effective dose equivalent of 4.0 man Sv and an electric energy generated of 1.3 GW a [A1] was somewhat lower than the overall figure of 11 man Sv (GW a)$^{-1}$ used in Table 40 [V1]. Data on doses to personnel at LWGRs in the USSR have been reported at some reactors [P4, B15]. For the two reactor units at Kolskaya, the collective dose in 1980 of 2.3 man Sv was typical of earlier years; the normalized collective dose of 1.9 man Sv (GW a)$^{-1}$ for 1980 was somewhat lower than the average value of 2.5 man Sv (GW a)$^{-1}$ for 1977-1980. The annual average dose to personnel was about 5 mSv over that period. The data for Japan in Tables 40 and 41 were compiled mainly from detailed statistics supplied by Kumatori [K1] and Terasima [T12].

118. Recent data for HWRs in Canada are shown in Table 42 [A4]. The values include internal doses from

exposure to tritium. The electric energy generated was obtained from IAEA tabulations [I3, I4, I5, I6]. The results for Canada show considerably higher collective effective dose equivalent per unit energy generated than would be obtained by considering only the two largest power plants that produce the bulk of the energy. The lifetime collective effective dose equivalent generated by the Atucha 1 HWR in Argentina has been estimated at 27 man Sv $(GW\ a)^{-1}$ [P8].

119. Most of the GCRs in the world are in the United Kingdom. Comprehensive data on them [H1, H2, H3, H4, P5, W2] are shown in Table 42. The step change in the number of workers after 1980 is because the figures prior to 1981 did not include workers not directly employed by the Central Electricity Generating Board. The collective effective dose equivalent distribution ratio has been low: less than 0.01 in 1984 [P5]. The GCR in Japan is of a similar design to the early GCRs in the United Kingdom [K1]; that in the United States is a small high temperature gas cooled reactor [B27, N3], so the doses from it are not directly comparable to the others.

120. Collective doses to personnel at the Dounreay establishment in the United Kingdom concerned with operation of the prototype FBR were 0.15 man Sv in 1984 and 0.29 man Sv in 1985 [U3]. Most of this collective dose resulted from charge machine refurbishing and irradiated fuel cell entries. Although many aspects of the design and operation of FBRs were reviewed at a recent symposium [I11], no data on occupational exposures were reported. The Committee would welcome more information on this aspect, especially from prototype and nearly commercial-scale reactors.

121. Despite the lack of data from some countries and for some years, there is enough information from the countries for which each reactor type is installed in large numbers to make a reasonable estimate of the normalized occupation dose for the quinquennium 1980-1984 for the major reactors. These estimates, which are based on the data in Tables 40 and 42, are given in Table 43. Bearing in mind the world-wide predominance of LWRs, the overall estimate must be heavily weighted by the estimate for this reactor type, but a figure of 10 man Sv $(GW\ a)^{-1}$ does not appear unreasonable.

D. SOLID WASTE DISPOSAL

1. Solid waste production

122. During operation of a power station, solid wastes are generated in a number of ways. In LWRs the main cause is treatment of the circulating water, giving rise to spent ion exchange resins, filter sludges and evaporator concentrates. Although these are originally wet wastes and may be stored in this form at the site, they are generally solidified before disposal. A similar type of waste arises from purification of the water in spent fuel storage ponds at reactors. Even though fuel elements are eventually removed for long-term storage or reprocessing, provision is made for short-term storage at reactors for initial decay heat removal. In addition, waste may include some structural components from the core or fuel, such as the outer fuel element structures from GCR and AGR fuel elements.

123. The radionuclides present in the above wastes are fission products, activation products and actinides, the particular radionuclides, quantities and relative activities being dependent on the reactor type, the state of the fuel cladding, the levels of corrosion, etc. In most cases these wastes will be the bulk of what is normally classified as intermediate-level wastes (ILW), i.e., wastes containing substantial activity concentrations but not significantly heat generating.

124. The other main cause of solid wastes during operation is the protective material of various kinds used around the station. Much of this is burnable and considerable volume reduction can be achieved by incineration and compaction. The radionuclide composition is even more variable than for the wet wastes and the activity concentrations are small to zero. These wastes are generally classified as low-level wastes (LLW).

125. In order to characterize the wastes for analysis of the impact of disposal, it is necessary to determine the volumes and the activity concentrations with identification of the relative quantities of important radionuclides, especially long-lived radionuclides and any actinides.

126. There were a number of studies in the mid-1970s on the quantities of wastes produced at LWRs [B30, M7, M8]. The results, summarized in Table 44, have been extracted from a review carried out by the Environmental Protection Agency of the United States [E5]. More recent reviews have been carried out in the United States by the Nuclear Regulatory Commission [N8, N9] and by the United States Department of Energy [D4], as well as in other countries using LWRs or planning to do so [N10]. On the basis of these studies, the quantities of conditioned wastes arising from LWRs per unit energy generated are assumed to be as shown in Table 45. These are only approximate; variations of up to an order of magnitude are possible in particular circumstances, depending on the type of treatment or conditioning used.

127. The assumed radionuclide compositions for the wastes in Table 45 are shown in Table 46. These are based primarily on the analyses reported in the United States [N8, N9, E6] and the United Kingdom [P10].

128. The quantities and activity concentrations of operating wastes of HWRs derived from data given for Canadian CANDU reactors [B31] are assumed to be as shown in Table 47. The quantities of wastes from operational GCRs have recently been reviewed [F3], and the results are also summarized in Table 47. The main differences in radionuclide composition from the LWR wastes are the higher alpha activity of the Magnox reactor sludges and the graphite debris containing ^{14}C. Although there are significant differences between reported inventories for Magnox

reactors and AGRs [P12] and LWRs, the composition in Table 46 is taken for this preliminary study, given the predominance of LWRs.

2. Solid waste disposal facilities

129. A large proportion of the LLW produced at all facilities during operation can be disposed of by burial at a shallow depth. Burial facilities range from simple trenches or pits containing untreated wastes and capped with soil, to concrete structures containing conditioned wastes and capped with weather-resistant materials. These will be referred to as trenches and engineered disposal facilities.

130. Considerable quantities of LLW have been disposed of in such facilities throughout the past few decades. Many of the earlier disposal sites were not used for disposal of wastes from the generation of nuclear power, except perhaps for some research and development aspects. For example, there are 14 sites in the United States operated by the United States Department of Energy for the disposal of wastes generated from certain defence research activities. Some major closed and currently operating LLW burial sites are shown in Table 48 [C8, C9, N9]. These have accepted wastes from a range of operations [H15, M10].

131. Typical simple trenchs are about 10 m deep and 25 m wide and could be from 100 to 200 m in length, depending on the site. They are covered by about 1 m of compacted soil. The waste is not conditioned except to render the material non-combustible where necessary. This is similar to the minimum engineered trench specified by Pinner [P11] and the base case of the Nuclear Regulatory Commission of the United States [N9]. Some major routes by which radionuclides will be released from such a trench will be into rainwater percolating through the trench and into ground water. There will be considerable differences between the behaviour of elements that form easily soluble compounds, such as iodine, and those that do not, such as uranium, as well as a marked dependence on the environmental and hydrological conditions of the site.

132. On the basis of knowledge of the chemical behaviour of the elements of which there are important radionuclides, the release behaviour of the wastes can be classified into three groups [P11]. It is also assumed that, since LLW is usually disposed of in trenches without packaging, radionuclides begin to be released into water as soon as the site is closed. The reference site is assumed to be above the water table in reasonably permeable, weathered material that has an underlying less permeable rock. In a site with these characteristics, water filtering through the waste will tend to move down through the unsaturated zone until it reaches the water table and the impermeable boundary where it moves downslope. It is assumed to reach a stream at a distance of 2,000 m from the site.

133. Some categories of ILW containing radionuclides with longer half-lives or at activity concentrations too great for disposal in simple trenches have been disposed of in engineered shallow disposal facilities. A typical facility is an excavation about 20 m deep and 25 m wide lined with 1 m of concrete. Such facilities are filled with concreted wastes to about one half their depth, the interstices being filled with concrete and finished with layers of concrete and clay to form an impervious cap. The canisters and concrete around the wastes will prevent rain or ground-water access for a considerable time, which is taken to be 100 years. After this time it is assumed that all radionuclides are released into percolating water at a constant fractional rate of 10^{-2} a^{-1}.

134. As a result of the greater depth of emplacement, it is likely that engineered facilities would be positioned below the water table. It is also sensible to locate the facilities in materials with good sorption properties, so the reference site is assumed to be in clay. Many clay outcrops are associated with harder, more permeable rocks leading to artesian conditions, i.e., rising ground water. The trench would interfere with this locally so that the eventual flow pattern assumed for the reference site is that water infiltrating the trench from above will tend to move downward, then upwards and outwards, eventually entering streams at a distance of about 1,000 m on either side of the site [P10].

135. An alternative method of disposal for packaged solid wastes is to dump them into a sea-bed at considerable depth. Although such disposals were carried out for many years, they ceased in 1982 under a temporary moratorium. The amounts of wastes disposed of to this date in the north-east Atlantic have been summarized by NEA [N6] and are given in Table 49. It is not possible to assign the wastes to a particular power programme, and it is known that some of the major radionuclides, such as ^{14}C, arose as wastes in the form for sea dumping largely from the preparation of radiopharmaceuticals.

3. Collective dose commitments

136. After closure of a burial facility, there will be a period during which control over the site is maintained. This does not necessarily preclude the transport of radionuclides released from the wastes into percolating ground water but could reasonably be relied upon to prevent major human intrusion into the site, such as for building purposes. Thus, during the controlled period, taken to be 100 years, only release by water contact is considered; other pathways are assessed after this period. The major pathways possibly leading to exposure are shown in Figure 11.

137. The actual transport of radionuclides with ground water, after release from the waste, the container and any surrounding engineered structures, will be very dependent on the hydrogeologic characteristics of the site. Considerable effort is being devoted to the development of calculational techniques capable of handling detailed knowledge of particular sites. For this study a more general approach is appropriate, such as the one adopted in other generic appraisals of shallow land burial [P10, N9, N10].

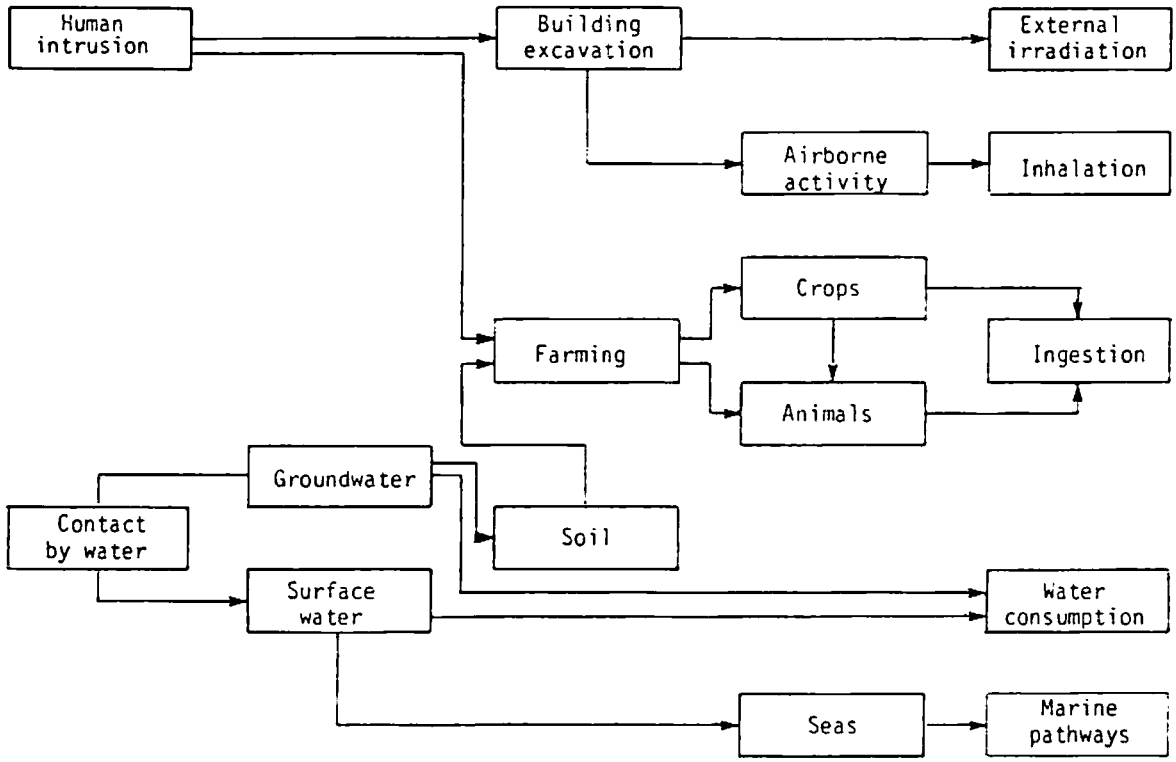

Figure II. General release and transport mechanism and pathways from shallow burial facilities.

138. The simplest representation of ground water flow velocity, caused only by the natural hydraulic gradient, is that given by Darcy's law:

$$V_w = K_p \, k_i / \varepsilon_k$$

where V_w is the ground water flow velocity, K_p is the hydraulic conductivity, k_i is the hydraulic gradient and ε_k is the kinetic porosity. This is the basis for several transport codes, such as FEFLOW, which is used in the Nordic study [N10], GEOS, which is used by the National Radiological Protection Board of the United Kingdom [H12], and that used by the Nuclear Regulatory Commission of the United States [N9]. It is adapted to radionuclide transport through a porous medium by including a retardation factor or distribution coefficient.

139. Generic assessments have also been carried out using a somewhat more realistic two-dimensional model, NAMMU [R4], to calculate the pressure head distributions and, hence, flowpaths and velocities in saturated porous media. This has been applied to migration through undisturbed clay and to movement in the surface soil layer [P10]. Whatever calculational method is used, the general result is that those nuclides with small retardation factors or distribution coefficients, such as tritium, ^{14}C, ^{99}Tc and ^{129}I, move at a velocity close to that of the ground water, whereas nuclides with large retardation factors or distribution coefficients, such as ^{235}U, ^{238}U and ^{239}Pu, move very slowly. The values for some important radionuclides are intermediate. Values adopted in three major studies [H12, N9, N10] are reasonably consistent.

140. The output from the radionuclide transport calculations is the rate of input of activity into either the nearest stream, as described for the generic site, or via ground water into soil that could be used for farming. Water could also be abstracted via a well. In calculating doses it is assumed that the water forms a source of drinking water for humans and animals. It is also assumed that fish from the stream are caught and eaten. The river model is a compartment type, with each compartment representing a homogeneous freshwater body and incorporating adsorption on to and resuspension of sediment particles [I15]. The flow rate of the river is taken to be 6 10^6 m^3 a^{-1}. The eventual transfer from rivers via estuaries to the sea is also included. In assessing doses from drinking water, it is assumed that suspended sediments are removed by filtration. Collective doses from streams and wells are assessed on the assumption that 0.2% of the flow rate and 1% of the abstraction rate are actually ingested.

141. If the land is used for farming, this will give rise to a large number of exposure pathways. The contamination can result from transfer directly upwards through the soil from ground water or via streams and rivers through irrigation. The calculation of collective doses requires an estimate of the total quantities of each foodstuff consumed, shown in Table 50, together with average values for activity concentrations obtained from the radionuclide transport models.

142. The collective dose equivalent rates per unit activity as a function of time after release from an engineered facility via all the pathways are shown for a number of important radionuclides in Tables 51, 52

and 53, for three major time periods of interest. In general, farming and water consumption pathways both contribute significantly to the collective dose.

143. The results of applying the specified models are shown in Tables 54 and 55 for the shallow earth trench and the engineered trench, respectively. The results are presented per unit activity in the trench and show the collective effective dose equivalent commitment and the maximum collective effective dose equivalent rate. Also shown is the time at which the specified percentage of the maximum collective effective dose equivalent rate is reached [S15].

144. Using the estimated volume for LLW from Table 45 of 200 m^3 $(GW\,a)^{-1}$ at an activity concentration of 1 GBq m^{-3} as appropriate for PWRs and assuming the radionuclide composition given in Table 46, it can be seen from Table 54 that the normalized collective effective dose equivalent commitment from burial of these relatively short-lived wastes is less than 10^{-10} man Sv $(GW\,a)^{-1}$. Only if long-lived radionuclides were present could there be a collective dose of any significance; if this proportion were taken to be one thousandth of the quantity present in ILW, as shown in Table 46, the normalized collective effective dose equivalent commitment would be about 10^{-5} man Sv $(GW\,a)^{-1}$.

145. Taking the estimated volume for ILW from Table 45 of 50 m^3 $(GW\,a)^{-1}$ at an activity concentration of 100 GBq m^{-3}, again, as appropriate for PWRs, and combining this with the data from Table 55 for the radionuclides specified to be present in Table 46, the normalized collective effective dose equivalent commitment from disposal of ILW in a model engineered trench is 0.5 man Sv $(GW\,a)^{-1}$. The main contribution is from the 0.1% by activity of ^{14}C assumed to be present in the waste. The contribution by radionuclide is shown in Table 56.

IV. FUEL REPROCESSING

146. At the fuel reprocessing stage of the nuclear fuel cycle, the elements uranium and plutonium in the irradiated nuclear fuel are recovered to be used again in fission reactors. Spent fuel elements are stored under water, which provides both biological shielding and cooling, while waiting to be reprocessed. Fuel elements are usually left until the short-lived ^{131}I has decayed to a low level, normally a minimum of four or five months. Since one reprocessing plant can serve large numbers of nuclear reactors, the quantities of nuclides passing through the plant that are significant from the point of view of health will be high in absolute terms. Careful design limits discharges, however, so that releases per unit of electricity generated by the fuel passing through the plant, i.e., $(GW\,a)^{-1}$, may be relatively small.

147. The only commercial operating reprocessing plants are at Sellafield (formerly Windscale) in the United Kingdom and Cap de la Hague and Marcoule in France. The capacity at Sellafield is 2,000 t a^{-1} (heavy metal) and that of Cap de la Hague is 900 t a^{-1} oxide fuel, while the plant at Marcoule processes up to 400 t a^{-1} of GCR metal fuel. The annual throughput of irradiated fuel from civilian power programmes in these three reprocessing plants is currently equivalent to about 8 GW a of electric energy, representing about 5% of the reported annual nuclear electric energy production (189 GW a, Table 2). Thus, the majority of irradiated fuel, which arises from LWRs, is not reprocessed but is stored pending future policy decisions as to whether to dispose or reprocess. A summary of the attitudes of countries with power reactors towards reprocessing is given in Table 57, while in Table 58 national programmes for commercial reprocessing are given [C3].

A. EFFLUENTS

148. The design and operation of reprocessing plants to avoid releases of large amounts of radioactive material is complex. The gaseous and volatile fission product elements (iodine, tritium, carbon, krypton, ruthenium, technetium, xenon and caesium) are largely separated from the fuel when it is dissolved in nitric acid. The dissolver off-gas is treated for nitric acid recovery and iodine removal before being mixed with the off-gases from other stages in the process. The vessel off-gases are treated by caustic scrubbing, drying and filtering through high efficiency filtration systems before being discharged to the atmosphere. The aqueous wastes containing almost all the fission products and transuranic elements are concentrated by evaporation and stored in double containment stainless steel tanks before they are treated further.

149. The radionuclides of principal concern in reprocessing effluents are the long-lived nuclides: ^{3}H, ^{14}C, ^{85}Kr, ^{129}I, ^{134}Cs, ^{137}Cs and isotopes of transuranium elements. Table 59 lists the reported discharges to the atmosphere, and Table 60 those in aquatic releases, from Sellafield, Cap de la Hague and Marcoule for 1980-1985. The amount of activity in the effluents depends upon the specific waste treatment and processing design of the plant, as well as the type of fuel processed, its irradiation history and storage time prior to reprocessing. Table 61 gives the isotopic composition of liquid effluent discharges from the Sellafield and Cap de la Hague plants in greater detail for the years 1980-1985. Atmospheric release data and liquid discharge data for Marcoule are not available beyond 1980.

150. The throughput of fuel at both Sellafield and Cap de la Hague has been calculated on the basis of ^{85}Kr reported discharges and on the assessment of the ^{85}Kr generation in different reactor fuel cycles made in the UNSCEAR 1982 Report. In that Report, the Committee has used production rates of 14 PBq $(GW\,a)^{-1}$ for GCRs and 11.5 PBq $(GW\,a)^{-1}$ for PWRs. These figures make assumptions about fuel burn-up and reactor thermal efficiency that are not likely to have changed significantly since the UNSCEAR 1982 Report. On this basis, the electric energy production of the annual throughput of fuel at Sellafield has varied between 1.7 GW a and 3.7 GW a, while for

Cap de la Hague the range has been 2.4 to 6.1 GW a. For Marcoule, there are little data on atmospheric discharges, although the electric energy of annual fuel throughput for 1980 has been estimated at 1.4 GW a.

151. For tritium, the Committee used in the UNSCEAR 1982 Report a production rate in LWR fuel of 0.75 PBq (GW a)$^{-1}$ and, assuming this applies to GCR fuel, the inventory passing through Sellafield has varied between 1.3 PBq (1985) and 2.8 PBq (1981). In 1981, atmospheric discharges of ^{3}H were 0.46 PBq and liquid discharges 2 PBq, giving 2.46 PBq, compared with the estimated throughput of 2.8 PBq. Thus, it appears that nearly all the tritium in irradiated fuel is released in reprocessing and about 20% is released to the atmosphere. This is identical to the percentage estimated in the UNSCEAR 1982 Report. The remaining tritium may be immobilized in cladding wastes. For Cap de la Hague the normalized releases of tritium have been 0.26 PBq (GW a)$^{-1}$ of which only about 1% is in reported atmospheric releases and thus it seems that only about one third of the throughput is released.

152. The results of routine measurements of ^{14}C atmospheric discharges from the Sellafield reprocessing plant are given in Table 59. The normalized production rate of ^{14}C in GCR fuel was estimated in the UNSCEAR 1982 Report at 3.2 TBq (GW a)$^{-1}$. Atmospheric discharges from Sellafield therefore seem to account for essentially the whole of the estimated throughput of ^{14}C between 1980 and 1985. For the French reprocessing plants, ^{14}C discharges are not reported. In the UNSCEAR 1982 Report, the Committee estimated the ^{14}C content of LWR fuel to be 0.66 TBq (GW a)$^{-1}$, of which about 75% was assumed emitted to the atmosphere, but in view of the Sellafield data, all ^{14}C can be considered released to the atmosphere for the dose assessment.

153. The ^{131}I content of irradiated nuclear fuel varies, depending upon the cooling time and the final power level of the fuel discharge. The ^{131}I normalized content of LWR fuel cooled for six months is estimated at 2.8 TBq (GW a)$^{-1}$, falling by a factor of 2,000 for a cooling period of nine months. Since irradiated fuel is generally cooled for at least a year prior to reprocessing, ^{131}I discharges are very small. For 1980-1985, Sellafield atmospheric releases of ^{131}I (Table 59) gave normalized values of 19 GBq (GW a)$^{-1}$; the corresponding figure for 1975-1979 was 1.7 GBq (GW a)$^{-1}$, the increase being due to a high release figure in 1981.

154. The quantity of ^{129}I in fuel depends upon burn-up and is assessed at 37-74 GBq (GW a)$^{-1}$. Atmospheric discharges of ^{129}I, as well as liquid effluent amounts, have been reported for Sellafield and Cap de la Hague but not for Marcoule. The normalized atmospheric release is 3.7 GBq (GW a)$^{-1}$ for Sellafield and 4.9 GBq (GW a)$^{-1}$ for Cap de la Hague for the period 1980-1985, which is about twice the value given in the UNSCEAR 1982 Report. Liquid effluents averaged about 30 to 60 GBq (GW a)$^{-1}$ at each plant over the same period, compared with 40 GBq (GW a)$^{-1}$ previously reported. It seems likely that all the ^{129}I in the fuel is released with a few per cent going to the atmosphere.

155. Atmospheric releases of aerosols are summarized in Table 59. The normalized alpha releases from Sellafield are 0.2 GBq (GW a)$^{-1}$, of which more than 75% is due to plutonium isotopes [B1, B2, B3, B8, B16], the remainder being accounted for by ^{241}Am and ^{242}Cm. This figure is one half that reported in the UNSCEAR 1982 Report. The alpha-aerosol results are available from France for Cap de la Hague and are some 30 times lower. For atmospheric beta releases, the largest component from Sellafield is ^{137}Cs, although since 1981 the levels have been reduced. The normalized release is 63 GBq (GW a)$^{-1}$, compared with 88 GBq (GW a)$^{-1}$ for 1975-1979. For Cap de la Hague the normalized release is 0.04 GBq (GW a)$^{-1}$ for beta-aerosols, and no isotopic breakdown is available. The reduction in aerosol releases in recent years from Sellafield is the result of improvements in the Magnox cladding silo stores, including the installation of inert gas blankets and filtration systems.

156. The liquid effluents discharged from Sellafield, Cap de la Hague and Marcoule are given in Table 60 for total alpha, total beta, ^{3}H, ^{90}Sr, ^{106}Ru and ^{137}Cs. There is a yearly isotopic breakdown for the French plant at Cap de la Hague but not for Marcoule. The isotopic compositions of Sellafield and Cap de la Hague discharges are given in Table 61 for 1980-1985.

157. The normalized alpha release from Sellafield to the sea is 8.0 ± 5.2 TBq (GW a)$^{-1}$, compared with an average of 25 TBq (GW a)$^{-1}$ between 1975 and 1979. For Marcoule and Cap de la Hague the figures are 0.063 and 0.16 TBq (GW a)$^{-1}$, while for the previous period they were 0.016 and 0.24 TBq (GW a)$^{-1}$. Most of the Sellafield alpha activity was $^{239/240}$Pu, and the level of alpha discharge has been reduced by a factor of 6 over the reporting period.

158. For liquid discharges of beta activity the normalized releases from Sellafield, Cap de la Hague and Marcoule are 0.97, 0.24 and 0.027 PBq (GW a)$^{-1}$, respectively, compared with 3.7, 0.52 and 0.04 PBq (GW a)$^{-1}$ for 1975-1979. The isotopic composition of the effluents varies between the sites: 55-70% of the Sellafield discharge is attributable to ^{137}Cs, whereas 40% of the Cap de la Hague discharge is attributable to ^{106}Ru. The ^{137}Cs levels from Sellafield were reduced by a factor of 9 over the review period, although the ^{106}Ru levels remained constant at about 400 TBq (GW a)$^{-1}$ until 1985. After ^{3}H, ^{106}Ru is the main isotope released from Cap de la Hague; the ^{106}Ru discharges are comparable to those of the Sellafield plant.

159. Monitoring of the marine environment is undertaken by regulatory authorities to ensure compliance with authorized discharges and to ensure that doses to exposed populations are at the levels predicted. The results of monitoring around the United Kingdom in the vicinity of all operating nuclear plants have been published by Hunt [H5, H6, H7]. The most significant

results arise from discharges of the Sellafield plant. Measurements of activity in fish and shellfish in 1983 are shown in Tables 62 and 63 for various locations around the United Kingdom. In order to interpret these results, consumption data are required to assess intakes of radionuclides.

160. Aarkrog [A2] has summarized bio-indicator studies in Nordic waters to identify levels of radioactive contamination. The marine bio-indicators are the blue mussel (Mytilus edulis) and bladder wrack (Fucus vesiculosus), which are sensitive to contamination from nuclear fallout and from Sellafield discharges and nuclear power plants in Sweden, Finland and the rest of coastal northern Europe. Discharges from Sellafield have been traced from the Irish Sea, along the western Norwegian coast, down along eastern Greenland and then western Greenland. The transit time from the Irish Sea is measured to be four years and the activity concentration is diluted by a factor of 100.

161. The measured concentrations of ^{137}Cs in sea water decrease from the highest levels of 1,000 Bq kg^{-1} near Sellafield to 8-10 Bq kg^{-1} in the Baltic Sea, 1-2 Bq kg^{-1} near Greenland and less than 1 Bq kg^{-1} near Iceland. Levels of ^{99}Tc from Sellafield discharges closely follow those of ^{137}Cs. Measurements of plutonium show enhanced levels primarily in British and Irish coastal waters, although very low levels have recently been detected in areas further from the coast.

B. LOCAL AND REGIONAL COLLECTIVE DOSE COMMITMENTS

162. The evaluation of the collective dose commitments from reprocessing nuclear fuel requires a study of the local and regional effects and of the global consequences of the releases. Estimates of the local and regional collective dose commitments are given in this section and the global contribution is provided in chapter V. The collective dose commitments are evaluated for the normalized discharges from Sellafield and Cap de la Hague by scaling the normalized results given in the UNSCEAR 1982 Report. As there are only three reprocessing plants operating with significant commercial throughput of fuel, the collective effective dose equivalent commitments per unit of electric energy generated are weighted by the fraction of fuel reprocessed to provide the current contribution from all operating reactors. In the UNSCEAR 1982 Report, the Committee gave typical discharge figures for notional new designs of reprocessing plant. This has not been repeated in this Report since all fuel may not be reprocessed. The weighted average, therefore, reflects actual exposures from the nuclear fuel cycle as currently operated.

1. Krypton-85

163. The averaged ^{85}Kr normalized discharge from Sellafield between 1980 and 1985 was 14 PBq $(GW\ a)^{-1}$ (Table 59), and the collective effective dose equivalent commitment obtained by the Committee in the UNSCEAR 1982 Report was 0.0074 man Sv $(PBq)^{-1}$. Thus, the normalized local and regional collective effective dose equivalent commitment is 0.1 man Sv $(GW\ a)^{-1}$. The normalized discharge from Cap de la Hague is 11 PBq $(GW\ a)^{-1}$, giving 0.08 man Sv $(GW\ a)^{-1}$. The average annual electric energy generated in recent years has been over 160 GW a, and an annual amount of fuel equivalent to 8 GW a was reprocessed. Thus, the normalized collective effective dose equivalent commitment from ^{85}Kr is 0.005 man Sv $(GW\ a)^{-1}$ electric energy generated.

2. Tritium and carbon-14

164. The Committee used in the UNSCEAR 1982 Report specific activity models to estimate collective doses from ^{3}H and ^{14}C discharges. The dose resulting from the release of tritium to the atmosphere was estimated in that Report at 0.0027 man Sv TBq^{-1} for the Sellafield site, some four times lower than the value for the reactor site, due to differences in site, population density and meteorological conditions. Releases from Sellafield to the atmosphere averaged 120 TBq $(GW\ a)^{-1}$ (Table 59), giving a collective effective dose equivalent commitment of 0.32 man Sv $(GW\ a)^{-1}$, 85% of which was from ingestion. For Cap de la Hague the normalized release of 3.5 TBq $(GW\ a)^{-1}$ gives a collective effective dose equivalent commitment of 0.01 man Sv. Releases to the regional marine environment were estimated in 1982 to lead to lower dose commitments. Using the value derived in the UNSCEAR 1982 Report of 1.8 10^{-3} man Sv PBq^{-1} released to oceans and the average release to the sea of 579 TBq $(GW\ a)^{-1}$ for 1980-1985 from Sellafield (Table 60) leads to a collective dose commitment of 1 10^{-3} man Sv $(Gw\ a)^{-1}$. The total normalized collective effective dose equivalent commitment weighted by the relative energy of fuel reprocessed at Sellafield and Cap de la Hague is 0.15 man Sv $(GW\ a)^{-1}$, essentially the same figure that was given in the UNSCEAR 1982 Report. Again, allowing for the fraction of fuel reprocessed, the weighted normalized collective effective dose equivalent commitment is 0.007 man Sv $(GW\ a)^{-1}$.

165. For ^{14}C releases to the atmosphere, the Committee estimated the collective effective dose equivalent commitment at 0.4 man Sv TBq^{-1}, with essentially the same value per TBq released to the aquatic environment. Averaged atmospheric releases from Sellafield are reported to be 3.5 TBq $(GW\ a)^{-1}$, giving a normalized collective effective dose equivalent commitment of 1.4 man Sv $(GW\ a)^{-1}$, compared with 0.69 man Sv $(GW\ a)^{-1}$ quoted in 1982. The difference is accounted for by the reported discharges to the atmosphere being double the values reported in 1975-1979. It would appear that the total throughput of ^{14}C at Sellafield is now accounted for in atmospheric releases. For Cap de la Hague, the assumed release of 0.66 TBq $(GW\ a)^{-1}$ gives a normalized collective effective dose equivalent commitment of 0.3 man Sv $(GW\ a)^{-1}$. Weighted for the fraction of fuel reprocessed, the contribution is 0.04 man Sv $(GW\ a)^{-1}$.

3. Other atmospheric releases

166. Of the other nuclides released to the atmosphere, ^{129}I becomes globally dispersed and makes a contribution to the collective dose commitment over a prolonged period, while the remainder contribute only to the local and regional collective dose commitment. A summary is given in Table 64. The total, averaging over Sellafield and Cap de la Hague, amounts to 1.3 man Sv (GW a)$^{-1}$, compared with the assessment of 3 man Sv (GW a)$^{-1}$ for atmospheric releases made in the UNSCEAR 1982 Report. Some 65% of the dose is now due to ^{14}C discharges, which are reported to be twice the previous levels and which counteract reductions in discharges of other nuclides, particularly actinides. Weighted by the proportion of electric energy generated, the normalized collective effective dose equivalent commitment from atmospheric releases during reprocessing is 0.07 man Sv (GW a)$^{-1}$.

4. Liquid effluents

167. The results are presented in Table 65 for the normalized collective effective dose equivalent commitments for marine discharges from reprocessing at Sellafield and Cap de la Hague. The environmental dosimetric models are appropriate for the specific coastal waters of northern Europe and were fully described in the UNSCEAR 1982 Report. They assume that consumed fish, molluscs and crustacea are the important food pathways to man.

168. For Sellafield, normalized liquid discharges for 1980-1985 were reduced by a factor of 3 since the period 1975-1979. The collective effective dose equivalent commitment per TBq for marine discharges from Sellafield found by the Committee in the UNSCEAR 1982 Report was 0.068 man Sv for ^{137}Cs, 0.034 man Sv for ^{106}Ru and 0.025 man Sv for alpha-emitters. The principal route of exposure is ^{137}Cs in consumed fish, as before, and for 1980-1985 the caesium contribution is some 85% of the total collective dose. The normalized collective effective dose equivalent commitment for 1980-1985 from liquid discharge from Sellafield is 44 man Sv (GW a)$^{-1}$, the estimate made in the UNSCEAR 1982 Report being 124 man Sv (GW a)$^{-1}$. If data for 1985 alone are taken, the normalized release gives a collective effective dose equivalent commitment of 25 man Sv (GW a)$^{-1}$, reflecting the lower discharges after the installation of a new plant to remove radioactive substances from effluent streams.

169. In the case of Cap de la Hague, discharges also seem to have been reduced. The Committee's models used in the UNSCEAR 1982 Report gave dose conversion factors per TBq released from Cap de la Hague of 0.1 man Sv for ^{106}Ru, 0.09 man Sv for ^{137}Cs and 0.4 man Sv for alpha-emitters. The normalized collective effective dose equivalent commitment shown in Table 65 is 11 man Sv (GW a)$^{-1}$, which compares with the figure of 53 man Sv (GW a)$^{-1}$ given in the UNSCEAR 1982 Report. The majority of the dose arises from the discharge of ^{106}Ru.

170. The collective effective dose equivalent commitment weighted for the relative amount of electricity produced by the fuel reprocessed at each plant is thus 25 man Sv (GW a)$^{-1}$ and, after allowing for the proportion of fuel reprocessed commercially, the normalized contribution is 1.2 man Sv (GW a)$^{-1}$. Annual committed effective dose equivalents to the critical group of winkle eaters close to the Sellafield site were reported to be 0.5 mSv in 1985 [B29]. The doses are reduced as discharge levels fall.

C. OCCUPATIONAL EXPOSURES

171. It was noted in the UNSCEAR 1982 Report that experience of fuel reprocessing is limited to a few countries and that plant design and historical operating conditions may not represent the best current potential for new plants. This view is supported by a recent review of the trends in the annual collective and the maximum individual occupational doses in a number of reprocessing plants [B22]. The review covered not only the large operating reprocessing plants at Cap de la Hague and Sellafield, but also the pilot plants WAK at Karlsruhe, Federal Republic of Germany, the Eurochemie plant at Mol, Belgium, the PNC plant at Tokai Mura in Japan, and the Idaho and Savannah River plants in the United States. Although recognizing the differences in sizes and design age of the various plants and that some of them reprocess fuel for military as well as civilian purposes, a downward trend in average doses was observed starting during the period 1971-1973 and ending during the period 1980-1982. The annual average effective dose equivalent dropped from 4-15 mSv in the early 1970s to 2-4 mSv early in the 1980s. Data since the UNSCEAR 1982 Report are summarized in Table 66 for Japan and the United Kingdom [A5, B12, B23, B28, H8]. The data for Japan refer only to the PNC plant at Tokai Mura. An estimate of 0.5 man Sv has been made of the neutron collective dose equivalent at Sellafield in 1982 [B24]. Data for Cap de la Hague and Marcoule from 1973 to 1985 are given in Table 67, taken mainly from the recent comprehensive review by Henry [H13]. This also shows annual average effective dose equivalents of about 2 mSv in the period 1982-1985 at both establishments.

172. In the UNSCEAR 1982 Report the normalized collective effective dose equivalents for the plants at Windscale (now Sellafield), United Kingdom, and Cap de la Hague, France, were estimated to be 18 and 6 man Sv (GW a)$^{-1}$, respectively. Some revised estimates for the United Kingdom are given in Table 66, based on ^{85}Kr discharges related to energy throughput and a fuel content of 14 PBq (GW a)$^{-1}$. The normalized value for Cap de la Hague is reported to have fallen from 6 man Sv (GW a)$^{-1}$ in 1975 to 1 man Sv (GW a)$^{-1}$ in 1985 despite a large increase in reprocessed fuel throughput over this period [B22]. This is in agreement with the data given in the report of a working group [C6] for a period leading up to 1981 and supplemented in a report to the Sizewell B public inquiry in the United Kingdom [Z1] with data for 1982 and 1983. These estimates are in agreement with the detailed results for Cap de la Hague reported by Henry [H13] and given

in Table 67. The Table shows that the normalized collective effective dose equivalent dropped steadily from 2.2 to 0.9 man Sv $(GW\ a)^{-1}$ throughout the period 1980-1985. The difference of nearly an order of magnitude between the normalized values for the two major installations makes it difficult to make a clear estimate. It seems, however, that the estimate in the UNSCEAR 1977 Report of the global collective effective dose equivalent per unit electric energy generated is, at 10 man Sv $(GW\ a)^{-1}$, too high. Based on the trends reported for Cap de la Hague, the estimate for Marcoule in 1980, the experience in Japan, and taking into account the predictions for the new plant at Sellafield, a better estimate for the whole of the 1980s is about 5 man Sv $(GW\ a)^{-1}$. When allowance is made for the proportion of fuel reprocessed commercially, the normalized contribution from occupational exposure is 0.25 man Sv $(GW\ a)^{-1}$.

D. SOLID WASTE DISPOSAL

173. The solid wastes that are generated in the handling, processing and disposal of spent fuels are of two broad categories. Most of the activity in the spent fuel is separated during reprocessing and, after a period of storage as a liquid, will be solidified for eventual disposal as high-level waste (HLW), generating significant decay heat. During the reprocessing operation considerable amounts of solid low-level wastes (LLW) and solid intermediate-level wastes (ILW) are produced, some streams of the latter being characterized by an appreciable content of actinides. If the spent fuel is not reprocessed but stored and prepared for disposal, there will be almost no ILW or LLW. But the packaged spent fuel is then treated as HLW; it contains the actinides that would have been separated for re-use by the reprocessing operation. Since neither spent fuel nor vitrified HLW have been disposed of, they are not considered in this assessment of current operations.

174. Production of other solid wastes in reprocessing plants has been highly dependent on the operational characteristics of the particular plant. In particular, much of the waste produced at the Sellafield plant in the United Kingdom is attributable to the degradation of Magnox fuel in underwater storage and should not be taken to be indicative for other plants now or in the future.

175. Production of ILW from the British Magnox reprocessing programme has been estimated [T11] at 47,000 m^3 from the reprocessing of 30,000 t uranium metal. The activity content is estimated to be about $2\ 10^{16}$ Bq alpha and $2\ 10^{18}$ Bq beta/gamma activity at 1990. Taking an average fuel requirement for Magnox reactors of 200 t $(GW\ a)^{-1}$, these correspond to the quantities shown in Table 68. Comparing with the alpha inventory of the fuel throughput, calculated to be 6,700 TBq $(GW\ a)^{-1}$ at six months and 3,800 TBq $(GW\ a)^{-1}$ at 20 years [G5], the fraction of alpha activity throughput lost to the ILW is about 0.02-0.03. Similarly, taking the beta/gamma inventory of the fuel throughput to be $2.4\ 10^6$ TBq $(GW\ a)^{-1}$ at six months and $1.7\ 10^5$ TBq $(GW\ a)^{-1}$ at 20 years [G5], the fraction of beta/gamma throughput lost to the ILW is about 0.005-0.05. These will be very sensitive to reprocessing chemical conditions for some nuclides, especially ^{237}Np. The generation of ILW from the proposed oxide fuel reprocessing plant for AGR fuel was also estimated by Taylor [T11] to be 11000 m^3 from the reprocessing of 600 t uranium metal in the thermal oxide reprocessing plant (THORP). The activity content is $6\ 10^{15}$ Bq alpha and $5\ 10^{18}$ Bq beta/gamma activity, assuming a cooling period of five years. The average fuel requirement of the AGR is taken to be 30 t $(GW\ a)^{-1}$ to give the quantities in Table 68. Again, comparing with the alpha and beta/gamma inventories of the fuel throughput at five years, calculated to be 3,100 and $2.5\ 10^5$ TBq $(GW\ a)^{-1}$, respectively [G5], the fractions of activity throughput lost to the ILW are 0.01 for alpha and 0.1 for beta/gamma activities.

176. The annual rate of waste generation at Marcoule has been reported [B32] to be about 2,000-3,000 drums containing a total of about 4 TBq alpha and 4,000 TBq beta/gamma activity. Assuming the annual fuel throughput of the facility to be 0.4 GW a and the drum capacity to be 0.2 m^3 gives the quantities shown in Table 68.

177. An alternative method of estimating the activity content of other solid wastes is to assess it directly as a fraction of the throughput of radionuclides in the fuel. This approach has been used by the United States Department of Energy [D4] to give the results in Table 69. The quantities are comparable with those estimated by Hill et al. [H11] but considerably less than those estimated for an operating plant, as shown in Table 68. The difference is about two orders of magnitude for alpha emitters and nearer to three orders of magnitude for beta/gamma emitters.

178. To give some estimate of the consequences of disposal of such wastes, it is assumed that the alpha wastes are entirely ^{239}Pu and the beta/gamma wastes entirely ^{137}Cs, and a typical normalized production from Table 68 is taken to be 100 TBq $(GW\ a)^{-1}$ for alpha wastes and 10,000 TBq $(GW\ a)^{-1}$ for beta/gamma wastes. Using the model for a typical ILW engineered disposal trench from section III.D. and the values for collective effective dose equivalent commitment per unit activity disposed of from Table 55, the normalized collective effective dose equivalent commitment would be 1 man Sv $(GW\ a)^{-1}$. This is reduced to 0.05 man Sv $(GW\ a)^{-1}$ when account is taken of the proportion of fuel reprocessed commercially. Lower losses from throughput to the ILW and LLW waste streams as estimated in paragraph 176 would significantly reduce this estimate; greater losses of the very long-lived radionuclides ^{14}C and ^{129}I would significantly increase it.

V. COLLECTIVE DOSE COMMITMENTS FROM GLOBALLY DISPERSED RADIONUCLIDES

179. The nuclides giving rise to a global collective dose commitment are sufficiently long-lived and migrate

through the environment, thus achieving widespread distribution. Those of interest are ^{3}H, ^{14}C, ^{85}Kr and ^{129}I. The environmental transfer of ^{3}H, ^{14}C and ^{85}Kr is becoming fairly well established, and reliable estimates of collective dose commitments were made by the Committee in the UNSCEAR 1982 Report. Other long-lived nuclides, such as ^{239}Pu, are far less mobile in the environment and therefore become less dispersed after deposition on to soils or sediments, following release into the local region.

180. The very long-lived nuclides, such as ^{129}I, pose a special problem because of the uncertainty in predicting population size, dietary habits and environmental pathways over periods of tens of millions of years. Therefore, little use can be made of these collective dose commitments for decision-making purposes. The incomplete collective dose commitment, however, is useful to demonstrate the time distribution of the dose commitment and to estimate the per caput doses arising per year from a finite duration of a practice. In the following paragraphs, complete and incomplete dose commitments are given for the globally dispersed nuclides up to a maximum of 10^{6} a. The collective dose commitments per unit release were taken from the UNSCEAR 1982 Report of the Committee and scaled for the normalized releases derived for 1980-1984 discharges.

181. In the UNSCEAR 1982 Report, a model reprocessing facility was described and all reactor fuel was assumed to be reprocessed. In this Report, the collective doses assessed for reprocessing plant reported discharges are weighted by the fraction of the energy value of the total nuclear fuel that is reprocessed, namely 5% (paragraph 147). The weighted contribution is added to any contribution from reactor operation to reflect the current normalized exposures.

A. KRYPTON-85

182. Since krypton is an inert gas, it disperses throughout the atmosphere and achieves a uniform concentration in about two years. The Committee, in the UNSCEAR 1982 Report, estimated the collective effective dose equivalent commitment from ^{85}Kr to be 0.17 man Sv PBq^{-1}, assuming a world population of $4\ 10^{9}$. This must be scaled up to 0.2 man Sv PBq^{-1} for the world population of $4.6\ 10^{9}$ during the period 1980-1985. All the dose commitment is delivered within the first 50 years after release. Paragraph 150 gave normalized production of ^{85}Kr as 11.5 PBq $(GW\ a)^{-1}$ for LWRs and 14 PBq $(GW\ a)^{-1}$ for GCRs, leading to 2.3 man Sv $(GW\ a)^{-1}$ and 2.8 man Sv $(GW\ a)^{-1}$ collective effective dose equivalent commitment, respectively. Contributions to the collective effective dose equivalent commitment come almost equally from whole-body gamma-radiation and from beta-irradiation of the skin. Weighting this collective dose by the fraction of fuel currently reprocessed (0.05) leads to 0.12 man Sv $(GW\ a)^{-1}$. The incomplete collective dose commitments are shown in Table 70, which indicates that half of the dose from ^{85}Kr is delivered in the first 10 years after discharge.

B. TRITIUM

183. The models used by the Committee in the UNSCEAR 1982 Report gave a collective effective dose equivalent commitment of $2.8\ 10^{-5}$ man Sv per TBq released. Because of the short half-life of tritium, this applies to the world population at the time of release. For the 1980-1985 world population of $4.6\ 10^{9}$, the dose factor is increased to $3.2\ 10^{-5}$ man Sv per TBq. Releases to the atmosphere and hydrosphere were not distinguished, since the exchange of water between the atmosphere and circulating waters of the globe is rapid, and the models assume immediate mixing and exchange with the hydrogen content of the circulating water.

184. The normalized release of tritium to the atmosphere from reactor operations, weighted by electricity production, is 46 TBq $(GW\ a)^{-1}$, while for liquid discharges the data give 40 TBq $(GW\ a)^{-1}$. Averaged over Sellafield and Cap de la Hague, aquatic and atmospheric releases from reprocessing add up to about 600 TBq $(GW\ a)^{-1}$, and since only 5% of the fuel is reprocessed, this adds 30 TBq $(GW\ a)^{-1}$ to the reactor releases of 86 TBq $(GW\ a)^{-1}$. The total collective effective dose equivalent commitment amounts to 0.004 man Sv $(GW\ a)^{-1}$. The incomplete collective dose commitments shown in Table 70 indicate that essentially all of the dose is received in the first few years after discharge. The local and regional contribution from tritium releases from reactor operation and the fractional reprocessing contribution amount to about 0.6 man Sv $(GW\ a)^{-1}$, which is a factor of over 100 greater than the global contribution.

C. CARBON-14

185. The Committee used in the UNSCEAR 1982 Report a relatively complex compartment model to assess the environmental distribution and behaviour of ^{14}C. This model allows for two hemispheres, each comprising humus, circulating carbon, surface ocean and deep ocean. The circulating carbon represents the carbon in the troposphere and those sectors of the terrestrial biosphere subject to rapid growth and decomposition. Humus represents the carbon content of the terrestrial biosphere which circulates more slowly. Carbon-14 releases are assumed to be instantaneously mixed in the compartment to which release occurs. The results produced by this model are similar to those produced by more complex models; the main area of uncertainty is the rate of transfer of ^{14}C to the deep ocean, from where it is less available.

186. The resulting collective effective dose equivalent commitment is 67 man Sv TBq^{-1} released, averaged over both aquatic and atmospheric releases and assuming a future global population of 10^{10}. Normalized releases from reprocessing plants are averaged over the reported figures for Sellafield (Table 59) and calculated throughput for Cap de la Hague (paragraph 152). Measurements appear to show that all the throughput is measured in airborne effluents, and it is assumed that little is discharged to the sea. The normalized release is 3.5 TBq $(GW\ a)^{-1}$ from Sellafield

(Table 59); Cap de la Hague is assumed to give rise to releases of 0.66 TBq (GW a)$^{-1}$. The collective effective dose equivalent commitment for Sellafield is thus 234 man Sv (GW a)$^{-1}$ and 44 man Sv (GW a)$^{-1}$ for Cap de la Hague. Since about 5% of the annual energy equivalent of fuel is reprocessed, the weighted figure averaged over the two sites is 6 man Sv (GW a)$^{-1}$, the remaining fuel being stored and not giving rise to effluent releases of ^{14}C.

187. HWR releases are about 7.3 TBq (GW a)$^{-1}$ of ^{14}C from reactor operations (Table 25), while those from LWGRs and GCRs are about 1.1 TBq (GW a)$^{-1}$ (paragraph 72). LWR releases at about 0.3 TBq (GW a)$^{-1}$ (paragraph 70) are small in comparison. The normalized collective effective dose equivalent commitment from HWR operation is therefore 490 man Sv (GW a)$^{-1}$. This is nearly a factor of 3 lower than the estimate given in the UNSCEAR 1982 Report, and is entirely due to lower reported discharge figures. About 6% of total nuclear generated electric energy arises from HWRs and about 10% from LWGRs and GCRs, so the electricity production weighted contribution to collective dose is 32 man Sv (GW a)$^{-1}$ from HWRs and an additional 7.7 man Sv (GW a)$^{-1}$ from GCRs and LWGRs. Although LWR releases are lower, because of their larger electric production, LWRs add 17 man Sv (GW a)$^{-1}$. In summary, the present practices of reactor operation and reprocessing lead to a total collective effective dose equivalent commitment of 6 man Sv (reprocessing) plus 57 man Sv (from HWR, LWR, LWGR and GCR operation), i.e., 63 man Sv (GW a)$^{-1}$. This commitment is received over some 10000 years, while the temporal distribution is shown in Table 70 to be 3% in 10 years, 10% in 100 years and 19% in 1,000 years.

D. IODINE-129

188. When released to the atmosphere, iodine, because of its environmental mobility, becomes rapidly incorporated into foodstuffs ingested by individuals. The highest concentrations of iodine occur in sea water and, as with ^{14}C, the greatest uncertainties surround the transfer of ^{129}I to deep oceans and any sedimentation that may remove activity from any biological chain.

189. Assuming again a future global population of 10^{10}, the Committee used a collective effective dose equivalent commitment of $1.4\ 10^{4}$ man Sv TBq^{-1} released [U1]; of this, some 0.003% is delivered within 100 years of release, 0.03% in 10,000 years, 5% in 10^{6} years, thus leaving 95% of the collective dose to be delivered from 1 million years after release, most of it coming between 10 million and 40 million years. For this report incomplete dose commitments to 10^{6} years are used so that the value of ^{129}I is 700 man Sv TBq^{-1}.

190. The normalized releases from Sellafield and Cap de la Hague from 1980-1985 averaged about 40 GBq (GW a)$^{-1}$ to the sea and 4 GBq (GW a)$^{-1}$ to the the atmosphere, giving a total of 44 GBq (GW a)$^{-1}$, which, when weighted for the fraction of fuel that is reprocessed, gives 2.2 GBq (GW a)$^{-1}$. The corresponding incomplete collective effective dose commitment to 10,000 years is 1.5 man Sv (GW a)$^{-1}$. The incomplete value to 10^{4} years is 0.0093 man Sv (GW a)$^{-1}$ and for 100 years 0.0008 man Sv (GW a)$^{-1}$, as shown in Table 70.

VI. TRANSPORT

191. Materials of various types are transported between the installations involved in the entire fuel cycle. The amounts and distances depend on the number of facilities and the degree to which different facilities are located together. An estimate is given in Table 71 of the transport needs in a complete nuclear fuel cycle; this has been adapted from the report of the International Fuel Cycle Evaluation (INFCE) [I15]. In general, mills are located together with mines, and tailings are disposed of close by, so that there is no significant requirement for transport of very large quantities of ore or wastes. The other major transport requirements shown in Table 71 can not be eliminated by co-location, as other factors will dominate the siting requirements. IAEA has continued to work towards a full assessment of the radiological impact of transport and have recently published the preliminary findings of a technical committee [P13]. The general conclusion was that, although the data available were incomplete, the indications were that exposures resulting from normal transport operations were low both for workers and members of the public.

192. The estimates made during the course of the IAEA study of occupational collective doses from the transport of fuel cycle materials were a recognized cautious estimate of 19 man Sv for the United States as a projection for 1985 [N11] and a more realistic estimate of 0.14 man Sv for the United Kingdom in 1981 [G1]. Estimates of less than 0.01 man Sv were made for selected operations in France, Italy and Sweden, but these could not be normalized to energy production. Using the energy production figures for the appropriate years gives normalized collective effective dose equivalents of 0.5 man Sv (GW a)$^{-1}$ for the United States and 0.04 man Sv (GW a)$^{-1}$ for the United Kingdom. Noting that the United States assessment was pessimistic, but that the United Kingdom assessment did not include the transport associated with uranium mining and milling, an overall estimate of 0.2 man Sv (GW a)$^{-1}$ is probably reasonable.

193. Doses to members of the public were also estimated as part of the work of the IAEA committee, based again on submissions from the United States and the United Kingdom. The estimate for the United States was 19 man Sv for 1985 [N11], the same as that for occupational exposure, whereas that for the United Kingdom was several orders of magnitude lower, at 0.001 man Sv for 1981 [G1]. No estimates were available for other countries. Based mainly on the more realistic British assessment, it seems reasonable to conclude that public exposure from transport is less than occupational exposure and to adopt an estimate for the normalized collective effective dose equivalent of 0.1 man Sv (GW a)$^{-1}$.

VII. SUMMARY

194. In the UNSCEAR 1982 Report the Committee carried out a thorough assessment of the exposures to the public from nuclear power production. In this Report the same basic assumptions and environmental transport models are used to carry out a revised assessment based on discharge data for the quinquennium 1980-1984. Some aspects of waste disposal have been treated here in more detail, especially the long-term impact of uranium mill tailings and the disposal of solid low- and intermediate-level wastes by burial on land. The contribution from reprocessing is based more closely on the results being obtained at operating plants rather than on the notional plant used in the previous report. Occupational exposures from the various stages in the fuel cycle are reviewed in this Annex in association with the other exposures from released radioactive materials.

195. A summary of the local and regional normalized collective effective dose equivalent commitments from the nuclear fuel cycle is shown in Table 72. The total of 4 man Sv $(GW\,a)^{-1}$ is essentially the same figure as that derived in the UNSCEAR 1982 Report if the contribution from uranium mine tailings is excluded, although in this Annex reprocessing is added explicitly, whereas a notional plant was used for the UNSCEAR 1982 Report. Contributions other than radon arise mainly from routine atmospheric releases from reactors and the liquid discharges from reprocessing. Effectively, all of these dose commitments are received within one to two years of discharge.

196. The normalized collective effective dose equivalent commitments from the long-term releases from solid waste disposal are shown in Table 73. The dominant contribution, as was recognized in the UNSCEAR 1982 Report, is from mine and mill tailings. The numerical estimate is roughly proportional to the length of time for which release of radon is assumed to occur. The estimate of 150 man Sv $(GW\,a)^{-1}$ corresponds to 10000 years for a tailings pile with a reasonable covering. The estimate for disposals of LLW and ILW are for the release from the disposal sites for all time, but a large proportion of the dose is received within about 10^4 years from the date of disposal. This applies also for the globally dispersed radionuclides shown in Table 73, as these are dominated in terms of the normalized contribution by ^{14}C.

197. The contributions of the various stages of the fuel cycle to occupational doses are summarized in Table 74. The dominant contribution is from reactor operation, itself based mainly on recent experience with LWRs in the United States but with considerable data from many other countries.

198. The per caput doses from existing nuclear power production are estimated from the contributions to collective dose commitment in the short term. This collective dose commitment is from local and regional collective doses and from occupational exposure, i.e., 4 and 12 man Sv $(GW\,a)^{-1}$, respectively (Tables 72 and 74). Assuming a global population of $5\ 10^9$, the per caput dose would be 3 nSv $(GW\,a)^{-1}$. The energy production from nuclear power in 1987 is about 190 GW a (Table 2), so that the annual per caput dose is estimated to be 0.6 μSv.

Table 1

World nuclear generating capacity, 1987
(Net capacity in gigawatts and number of units in parentheses)
[11]

Country	Reactor type						Total capacity	Installed capacity per caput (kw)
	PWR	BWR	GCR	HWR	LWGR	FBR		
Argentina				0.94 (2)			0.94	0.031
Belgium	5.49 (7)						5.49	0.55
Brazil	0.63 (1)						0.63	0.007
Bulgaria	2.59 (5)						2.59	0.29
Canada				12.10 (18)			12.10	0.47
China (Taiwan Province)	1.81 (2)	3.10 (4)					4.92	0.25
Czechoslovakia	3.20 (8)						3.20	0.21
Finland	0.89 (2)	1.42 (2)					2.31	0.47
France	46.46 (46)		2.01 (4)			1.43 (2)	49.80	0.90
German Democratic Rep.	1.69 (5)						1.69	0.10
Germany, Federal Rep.of	11.73 (11)	6.89 (7)	0.30 (2)			0.02 (1)	18.95	0.31
Hungary	1.65 (4)						1.65	0.15
India		0.30 (2)		0.85 (4)			1.15	0.002
Italy	0.26 (1)	0.86 (1)					1.12	0.02
Japan	11.97 (16)	14.64 (19)	0.16 (1)	0.15 (1)			26.90	0.22
Netherlands	0.45 (1)	0.06 (1)					0.51	0.04
Pakistan				0.13 (1)			0.13	0.001
Republic of Korea	4.75 (6)			0.63 (1)			5.38	0.13
South Africa	1.84 (2)						1.84	0.05
Spain	4.68 (6)	1.37 (2)	0.48 (1)				6.53	0.16
Sweden	2.63 (3)	6.83 (9)					9.46	1.14
Switzerland	1.62 (3)	1.31 (2)					2.93	0.45
USSR	16.87 (25)	0.05 (1)			15.98 (27)	0.70 (3)	33.60	0.12
United Kingdom			9.90 (36)	0.09 (1)		0.2 (1)	10.22	0.18
United States	63.57 (70)	29.1 (35)	0.33 (1)				92.98	0.38
Yugoslavia	0.63 (1)						0.63	0.03
Total	183.63 (225)	68.02 (85)	13.07 (45)	14.68 (28)	15.98 (27)	2.38 (7)	297.93 (417)	0.14

Table 2

Electricity generated by nuclear power, 1987
[11]

Country	Electric energy generated (GW a)	Percentage of total electricity generated
United States	51.9	18
France	28.7	70
USSR	21.3	11
Japan	20.8	29
Germany, Federal Republic of	14.1	31
Canada	8.32	15
Sweden	7.35	45
United Kingdom	5.58	18
Belgium	4.52	66
Spain	4.51	31
Republic of Korea	4.27	53
China (Taiwan Province)	3.58	49
Switzerland	2.48	38
Czechoslovakia	2.36	26
Finland	2.11	37
Bulgaria	1.31	29
German Democratic Rep.	1.18	9.7
Hungary	1.18	39
South Africa	0.71	4.5
Argentina	0.68	13
India	0.54	2.6
Yugoslavia	0.49	5.6
Netherlands	0.39	5.2
Brazil	0.10	0.5
Pakistan	0.03	1.0
Italy	0.01	0.1
Total	189	

Table 3

Uranium production by mining, 1980-1984
[04]

Country	Annual quantity of uranium oxide produced (kt)				
	1980	1981	1982	1983	1984
Argentina	0.18	0.12	0.15	0.18	0.13
Australia	0.56	2.92	4.42	3.21	4.39
Brazil	0	0	0.24	0.19	0.12
Canada	7.15	7.72	8.08	7.14	11.17
France	2.63	2.55	2.86	3.27	3.17
Gabon	1.03	1.02	0.97	1.01	0.92
Namibia	4.04	3.97	3.78	3.72	3.70
Niger	4.13	4.36	4.26	3.43	3.28
South Africa	6.15	6.13	5.82	6.06	5.73
Spain	0.19	0.18	0.15	0.17	0.20
United States	16.80	14.79	10.33	8.13	5.72
Total a/	44.0	44.0	41.3	36.7	38.7

a/ Not including centrally planned economy countries.

Table 4

Radon emission from some active uranium mines
[E1, J1, J2, L2, M4, T6, T7, W3]

Location	Mine type	Ore grade (%)	Annual radon emission (TBq)	Normalized radon emission (GBq t^{-1})
New Mexico, United States	Underground	0.1		0.2-3.4 a/
				0.6 b/
Tennessee, United States	Underground		6.7-58	
United States	Underground	0.1	10-17	
Elliot Lake, Canada	Underground		50-100	
Average: United States	Underground		11	0.8 c/
	Surface		12	
Large mine, United States	Surface		46	
Ranger, Australia	Surface	0.3	50-250	

a/ Range for seven mines.
b/ Average for seven mines.
c/ Average for 27 mines.

Table 5

Airborne emissions from a mill processing 2000 tonnes of ore per day
[U1]

Radionuclide	Annual emissions (GBq)
U-238	1-4
Th-230	0.2-2
Ra-226	0.2-2
Pb-210	0.2-2
Rn-222	1000-7000

Table 6

Radon emanation from uranium mill tailings piles
[B25, B34, C10, H14, L1, N5, R2, R3]

Location	Tailings management	Radon emanation rate per unit area ($Bq\ m^{-2}\ s^{-1}$)	Area (ha)	Annual radon emanation (TBq)
Argentina				
Chubut (1984)		2.7		
Malargue (1984)		5.8		
Malargue (1985)		10.8		
Cordoba (1985)		0.8		
Salta a/ (1984)		21		
St. Rafael (1983)		7.8		
Australia				
Rum Jungle	Uncovered	1.3	30	13
	3 m capping	< 0.07	30	< 0.7
Canada				
Elliot Lake	Vegetated	1.2-4.0	400	300
	Unvegetated	3.5		
India				
Jaduguda	Uncovered	1.1	12	4
United States				
(Temperate)	Uncovered	10	40	120
	1 m clay	0.3	40	4
(Semi-arid)	Uncovered	10	90	300
	Impermeable dam	0.1	90	3
Salt Lake City	Uncovered	18		
	Covered	7		
Ambrosia Lake	-	4		

a/ This mine and mill is at an altitude of 2000 m.

Table 7

Options for treatment of uranium mill tailings piles and relative effects on radon release rates
[N5]

Option	Predicted radon exhalation rate at 1000 years relative to base case a/ initial value
(0) Bare tailings pile	1.4
(1) 1 m top cover of silt/sand	0.73
(2) 3 m top cover of silt/sand	0.58
(3) 1 m top cover of clay	0.45
(4) As (1) with erosion protection b/	0.29
(5) As (2) with erosion protection b/	0.039
(6) As (3) with erosion protection b/	0.0027
(7) Bare tailings below ground level covered with 3 m clay-shale	$2.7\ 10^{-7}$
(8) As (7) with rock surround and gravel capping	$2.5\ 10^{-7}$

a/ Base case is the bare tailings pile.
b/ Crushed rock around exterior slopes and a gravel cap over the top surface.

Table 8

Collective effective dose equivalent commitments per unit activity released in airborne releases from uranium mines and mills

Radionuclide	Normalized collective effective dose equivalent commitment [man Sv $(TBq)^{-1}$]
U-238	7
U-234	8
Th-230	30
Ra-226	0.6
Pb-210	1
Po-210	1
Rn-222	0.015

Table 9

Truncated collective dose commitments from radionuclides released from tailings piles a/ [N5]

Management scenario a/	Truncated collective effective dose equivalent commitment (man Sv)							
	100 years		1000 years		5000 years		10000 years	
	Region	Continent	Region	Continent	Region	Continent	Region	Continent
Base case	46	380	480	4000	3200	26500	7800	64000
Option 1	12	96	200	1650	2200	18500	6450	53000
Option 2	2.8	23	120	990	1750	14500	5400	44500
Option 3	0.6	4.9	88	720	1900	15500	6000	50000
Option 4	12	96	120	960	580	4800	1200	9700
Option 5	1.6	13	16	130	80	660	160	1300
Option 6	0.1	0.9	1.0	8.8	5.6	47	12	100

a/ Treatment options are listed in Table 7. The base case is the bare tailings pile. The collective effective dose equivalent commitments from options (7) and (8) are less than 0.01 man Sv.

Table 10

Occupational exposures of underground uranium miners
[A4, B11, B37, E3, S8, Z2]

Country	Year	Number of workers monitored		Annual collective effective dose equivalent (man Sv)		Annual average effective dose equivalent a/ (mSv)
		Gamma exposed	Radon exposed	External radiation	Radon and its daughters	
Canada	1980	-	7124	- b/	40	5 b/
	1981	6140	6837	7.0 b/	58	10 b/
	1982	7160	6159	18.7	51	11
	1983	6290	4428	18.0	40	12
	1984	5850	3970	15.6	33	11
	1985	5810	3930	12.5	29	10
France	1980	1609		6.2	25	23 c/
	1981	1380		4.7	18	21 c/
	1982	1301		5.6	19	23 c/
	1983	1281	1281	5.0	16	25 c/
	1984	1384	1384	4.6	15	19 c/
	1985	1388	1388	4.8	13	18 c/
United States	1980	7600	7556	27.0	68	12
	1981		3790		27	7 d/
	1982		2120		14	7 d/

a/ Average for those exposed to gamma-radiation plus average for those exposed to radon and its daughters, except where noted. This procedure might lead to an overestimate.

b/ There was no monitoring for external radiation in 1980; data for 1981 are not for a complete year.

c/ Includes the following values of annual collective effective dose equivalent (man Sv) from mineral dust, for which a conversion of 34 Bq mSv^{-1} was taken: 1980, 6.0; 1981, 5.6; 1982, 5.4; 1983, 10.5; 1984, 6.4; 1985, 7.7.

d/ Radon and daughter exposure only.

Table 11

Radon and radon daughter exposures
of Canadian and Australian surface uranium miners
[A4, A8, M9]

Country	Year	Number of workers monitored	Annual collective effective dose equivalent (man Sv)	Annual average effective dose equivalent (mSv)
Canada	1980	124	0.005	- a/
	1981	138	0.006	- a/
	1982	219	0.15	0.7
	1983	535	0.6	1.1
	1984	500	1.0	2.0
	1985	510	0.9	1.7
Australia				
Nabarlek	1981/82	131	0.042	0.3
Ranger	1985/86	60	0.030	0.5

a/ Less than 0.05 mSv.

Table 12

Effluent discharges
from selected fuel conversion, enrichment and fabrication plants, 1980-1985
[A4, B1, B2, B3, B8, B16, B29, L1, M2]

Location and nuclide	Liquid discharges (GBq)					
	1980	1981	1982	1983	1984	1985
United Kingdom						
Capenhurst (enrichment)						
U-234	0.64	0.93	0.54	0.26	0.54	0.46
U-235	0.03	0.04	0.03	0.01	0.03	0.02
U-238	0.64	0.93	0.54	0.26	0.54	0.46
Th-234	0.65	0.95	0.55	0.27	0.55	0.47
Tc-99	11	6.9	20.5	3.7	2.0	0.35
Springfields (conversion, fabrication)						
Uranic alpha	900	600	700	700	800	700
Uranic beta	131000	37000	174000	215000	152000	160000
Canada (fabrication) (average values per year)						
Port Hope Uranium	0.09					
Toronto Uranium	3.9					
Peterborough Uranium	0.002					
Varennes Uranium	0					
Moncton Uranium	< 58					

Location and nuclide	Atmospheric discharges (GBq)					
	1980	1981	1982	1983	1984	1985
United Kingdom						
Capenhurst (enrichment)						
U-234	0.15	0.26	0.20	0.35	0.05	0.003
U-235	0.01	0.01	0.01	0.02	0.002	0.0001
U-238	0.15	0.26	0.20	0.35	0.05	0.003
Th-234	0.15	0.27	0.20	0.36	0.05	0.003
Springfields (conversion, fabrication)						
Uranic alpha	10.0	2.0	2.0	1.0	1.0	1.0
Uranic beta	10.0	2.0	2.0	1.0	1.0	1.0
Canada (fabrication) (average values per year)						
Port Hope Uranium	0.002					
Toronto Uranium	0.004					
Peterborough Uranium	0.0002					
Varennes Uranium	0					
Moncton Uranium	< 8					

Table 13

Normalized effluent discharges from model fuel conversion, enrichment and fabrication facilities
[MBq $(GWa)^{-1}$]

Radionuclide	Atmosphere			Aquatic		
	Conversion	Enrichment	Fabrication	Conversion	Enrichment	Fabrication
U-238	130	1.3	0.34	94	10	170
U-235	6.1	0.06	0.0014	4.3	0.5	1.4
U-234	130	1.3	0.34	94	10	170
Th-234	130	1.3	0.34	-	-	170
Th-232	0.022	-	-	-	-	-
Th-230	0.4	-	-	-	-	-
Th-228	0.022	-	-	-	-	-
Ra-226	-	-	-	0.11	-	-

Table 14

Normalized collective effective dose equivalent commitment from the model fuel fabrication facility

Radionuclide	Normalized collective effective dose equivalent commitment [10^{-3} man Sv (GW a)$^{-1}$]	
	Inhalation	Deposited activity
U-238	1.1	0.1
U-234	1.1	0.1
Th-230	0.001	-
Ra-226	-	-
Rn-222	0.4	-
Total (rounded)	2.6	0.2

Table 15

Occupational exposure from uranium fuel fabrication
[A4, B12, B23, H8, I3, I4, I5, I6, I16, J3, N2, P14]

Country	Year	Number of workers monitored	Annual collective effective dose equivalent (man Sv)	Annual average effective dose equivalent (mSv)	Normalized collective effective dose equivalent [man Sv (GW a)$^{-1}$]
Argentina	1981			0.3	0.35
	1982			0.2	0.12
	1983			0.2	0.10
	1984			0.3	0.15
	1985			0.2	0.11
	1986			0.3	0.15
Canada	1980	717	1.03	1.4	0.24
	1981	702	0.87	1.2	0.19
	1982	686	0.89	1.3	0.20
	1983	621	0.96	1.5	0.17
	1984	504	1.03	2.0	
	1985	509	1.21	2.4	
Japan	1981	1269	0.71	0.6	0.073
	1982	1465	0.83	0.6	0.073
	1983	1611	1.16	0.7	0.095
	1984	1654	0.92	0.6	0.064
United Kingdom	1980	3806	6.95	1.8	2.1
	1981	3816	6.38	1.7	1.8
	1982	3835	5.38	1.4	1.2
	1983	3626	3.94	1.1	0.9
	1984	3492	5.12	1.5	
United States	1980	5900	11.11	1.9	0.23
	1981	5942	9.40	1.6	0.18

Table 16

Occupational external exposures from plutonium fuel fabrication, 1977-1982
[A5]

Country	Year	Number of workers monitored	Annual collective effective dose equivalent (man Sv)	Annual average effective dose equivalent (mSv)
Japan	1977	114	0.9	0.13
	1978	134	0.8	0.10
	1979	225	1.0	0.23
	1980	198	1.7	0.33
	1981	200	2.5	0.50
	1982	266	2.2	0.58

Table 17

Noble gases discharged in airborne effluents from PWRs, 1980-1985
[A1, A6, B5, B19, B36, C4, F1, F4, J3, K1, S1, S2, S3, S5, S9, S10, T3, T4, T5, T8, T9, T10]

Country and reactor	Start-up year	Electricity generating capacity (GW)	Activity (TBq)					
			1980	1981	1982	1983	1984	1985
Belgium								
Doel 1,2,3	1974/75	1.68	94.1	76.4	112.1			
Tihange 1,2	1975	1.78	100.6	303.8	96.4			
France								
Blayais 1,2,3,4	1981/83	3.64	-	67	111	250	350	431
Bugey 2,3,4,5	1978/79	3.65	112	141	189	107	103	105
Chinon B1,B2	1982/83	1.74	-	-	-	120	150	170
Chooz	1967	0.30	98	76	170	170	210	163
Cruas 1,2,3,4	1983/84	3.42	-	-	-	79	210	270
Dampierre 1,2,3,4	1980/81	3.56	48	167	210	200	290	275
Fessenheim 1,2	1977	1.78	91	82	92	95	94	93
Gravelines 1-6	1980/85	5.46	81	211	185	230	240	330
Paluel 1,2	1984/85	5.32	-	-	-	-	110	336
St. Laurent B1,2	1981	1.76	-	104	120	121	128	130
Tricastin 1,2,3,4	1980/81	3.6	81	178	285	248	200	130
Germany, Federal Rep. of								
Biblis A,B	1974/76	2.39	50.0	18.0	14.0	18.5	22.5	14.0
Grafenrheinfeld	1981	1.23	-	0.0003	0.0007	7.4	0.58	0.012
Neckarwestheim	1976	0.79	49.0	1.8	2.9	5.0	14.0	11.0
Obrigheim	1968	0.33	3.8	5.0	12.6	17.0	1.8	0.97
Stade	1972	0.63	11.0	4.8	2.1	1.8	1.7	34.0
Unterweser	1978	1.23	21.0	11.9	7.4	6.0	4.2	5.6
Grohnde	1984	1.30	-	-	-	-	0.1	0.051
Philippsburg 2	1984	1.27	-	-	-	-	-	5.3
Italy								
Trino	1964	0.24						
Japan								
Genkai 1,2	1975/81	1.12	1.4	2.2	1.9	2.2	1.6	1.4
Ikata 1,2	1977/82	1.13	3.3	3.7	0.67	14	0.48	0.043
Mihama 1,2,3	1970/72/76	1.17	29.0	4.4	1.5	1.6	2.5	1.3
Ohi 1,2	1979	2.35	1.4	2.8	2.1	1.9	1.5	1.7
Sendai 1,2	1984	1.78	-	-	-	-	0.008	0.031
Takahama 1,2	1974/75	1.65	1.9	0.78	2.5	3.7	1.7	2.3
Netherlands								
Borssele	1973	0.45	13.6	8.9	45.9	8.9	0.7	
Sweden								
Ringhals 2	1975	0.80	9.8	13.0	27.9	2.35	9.04	3.6
Ringhals 3	1980	0.92	0.009	52.98	19.9	11.11	20.3	9.1
Ringhals 4	1982	0.92	-	-	0.36	0.44	0.92	0.49

Table 17, continued

Country and reactor	Start-up year	Electricity generating capacity (GW)	Activity (TBq)					
			1980	1981	1982	1983	1984	1985
USSR								
Armenian 1,2	1976/80	0.816	91.8	90.5	89.1	58.1	-	66
Kalinin 1	1984	1.000	-	-	-	-	-	-
Kola 1,2	1973/74	0.94	74.3	75.6	63.5	75.6	-	-
Kola 3	1981	0.440	-	-	-	-	-	-
Nikolaev 1	1982	1.000	-	-	-	162	190	200
Novovoronezh 1,2	1964/69	0.575	46.2	72.0	107	97.8	40	-
Novovoronezh 3,4	1971/72	0.834	78.7	32.7	127	46.3	14	-
Novovoronezh 5	1980	1.00	19.7	72.4	28.1	74.7	30	13
Rovno 1,2	1980/81	0.808	-	28.6	45.1	81.4	83	88
Zaporozhe 1	1984	1.000	-	-	-	-	-	110
United States								
Arkansas One-1	1974	0.84	1406	138.01	77.7	36.3	107.21	261.8
Arkansas One-2	1979	0.91	346.69	160.95	361.86	49.28	120.49	329.7
Beaver Valley	1976	0.81	3.197	29.82	4.85	7.31	42.97	0.113
Calvert Cliffs 1,2	1974	1.76	109.5	80.66	296	361.1	140.31	150
Crystal River	1977	0.82	1350.5	1465.2	253.45	125.06	72.55	99.04
Davis Besse	1977	0.91	123.95	37.37	19.8	33.88	18.53	4.38
Diablo Canyon	1984	2.15	-	-	-	-	0.0022	21.17
Donald C. Cook 1,2	1975	2.09	139.12	200.54	143.5	12.16	129.67	183
Farley 1	1978	0.83	710.4	8.18	1409.7	812.1	138.06	37.69
Farley 2	1981	0.87	-	0.096	130.98	31.33	147.76	1.25
Fort Calhoun	1973	0.46	10.99	45.14	12.8	32.52	56.56	
H.B. Robinson	1970	0.66	21.5	18.98	6.47	10.83	1.81	78.93
Haddam Neck	1968	0.58	99.16	67.71	27.9	102.28	269.79	
Indian Point 1,2	1973/76	1.77	347.06	337.81	268.99	354.87	139.86	
Indian Point 3	1976	0.965	41.07	243.09	95.46	20.70	69.41	
Kewaunee	1974	0.53	4.51	4.37	6.14	6.17	1.01	0.345
Maine Yankee	1972	0.77	14.32	12.14	56.61	1.45	4.53	15.95
McGuire 1,2	1981	1.18	-	0.0006	61.0	205.97	168.58	142.9
Millstone Pt. 2	1975	0.80	49.21	82.88	336.33	341.22	1087.57	
North Anna	1978	0.90	129.5	196.1	160.58	635.96	653.75	11.35
Oconee	1973/74	2.58	710.4	603.1	891.7	889.71	843.27	867
Palisades	1971	0.68	5.18	111.0	273.06	110.86	0.26	136.1
Point Beach 1,2	1970/72	0.99	23.7	22.7	36.7	28.45	3.45	4.29
Prairie Island 1,2	1973/74	1.04	9.62	1.72	20.2	10.26	2.80	1.69
R.E. Ginna	1969	0.47	31.86	20.2	72.15	26.52	10.96	
Rancho Seco 1	1974	0.91	58.46	50.69	54.76	25.61	143.57	173
Salem 1	1976	1.09	2.89	39.22	8.55	4.00	5.21	26.95
Salem 2	1980	1.12	0.29	22.53	41.07	19.88	7.16	10.47
San Onofre 1	1967	0.43	38.85	15.43	3.19	0.39	3.19	
San Onofre 2,3	1982	1.10	-	-	0.24	274.59	1485.46	937.4
Sequoyah	1980	1.15	111.37	334.11	212.38	145.05	247.16	
St. Lucie 1	1976	0.80	331.89	851.0	862.1	796.1	1323.74	
St. Lucie 2	1983	0.84	-	-	-	46.39	277.38	42.56
Surry 1,2	1972/73	0.57	228.29	521.7	780.7	203.02	256.29	
Three Mile Island 1	1974	0.79	1.717	2.15	2.8	0.74	0.013	
Three Mile Island 2	1978	0.91	1550.3	16.66	18.09	6.44	7.66	
TMI 2/EPICOR	1978	0.89	0.079	6.808	15.76	1.34	1.48	
Trojan	1975	1.10	14.47	42.92	31.3	8.45	30.92	
Turkey Point	1972/73	1.39	156.88	160.2	740.0	598.7	428.50	
Virgil C. Summer	1982	0.90	-	-	5.18	14.34	0.61	
Wolf Greek	1985	1.13	-	-	-	-	-	44.56
Yankee Rowe	1960	0.17	2.616	6.364	5.735	32.59	64.72	
Zion 1,2	1973/74	2.08	213.9	255.7	595.7	177.96	91.97	
Total annual electric energy generated (GW a)			35.50	43.75	48.17	56.29	64.94	60.22
Normalized activity [TBq (GW a)$^{-1}$]			275.83	179.64	211.57	254.07	171.42	108.79
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]					218 ± 40			

Table 18

Isotopic composition of noble gas discharges from PWRs in the United States, 1982
[T5]

Reactor	Start-up year	Electric energy generated (GW a)	Activity (TBq)									
			Ar-41	Kr-85m	Kr-85	Kr-87	Kr-88	Xe-131m	Xe-133m	Xe-133	Xe-135m	Xe-135	Xe-138
Arkansas One-1	1974	0.424	-	0.006	0.768	-	0.001	0.285	0.289	111.333	-	3.978	-
Arkansas One-2	1978	0.435	-	0.815	0.653	-	0.002	1.390	0.720	336.404	-	25.226	-
Beaver Valley 1	1976	0.307	-	-	0.093	-	-	0.086	0.028	4.627	0.004	0.009	-
Calvert Cliffs 1,2	1975/76	1.182	0.010	2.039	-	1.001	0.021	2.520	0.859	290.742	-	2.923	-
Crystal River	1977	0.561	5.439	1.380	50.690	0.747	0.451	21.241	1.910	176.585	2.819	12.841	15.133
Davis-Besse 1	1977	0.367	-	-	0.234	-	-	0.090	0.030	18.907	0.059	0.385	0.158
Donald Cook 1,2	1975/78	1.409	0.053	0.231	0.807	0.134	0.096	0.437	0.522	139.120	-	2.205	-
Farley 1	1977	0.595	0.433	6.438	0.958	6.882	6.882	0.633	1.432	335.069	0.052	895.403	0.981
Farley 2	1981	0.605	1.084	0.002	0.718	0.201	0.074	0.060	0.238	119.311	-	9.253	-
Fort Calhoun 1	1973	0.397	0.237	0.010	0.422	0.004	0.007	0.181	0.071	11.707	0.001	0.164	0.005
H.B. Robinson	1970	0.257	0.296	0.005	0.006	-	0.002	0.014	0.327	5.588	-	0.247	-
Haddam Neck	1967	0.518	0.001	0.042	3.811	0.013	0.031	0.046	0.240	22.388	0.008	1.269	0.031
Indian Point 1,2	1973/77	0.508	-	0.651	5.476	2.113	0.688	2.287	2.309	289.346	0.158	6.957	0.002
Indian Point 3	1976	0.164	-	0.275	0.514	0.005	0.015	0.722	0.662	90.661	0.006	2.802	0.002
Kewaunee	1974	0.436	0.262	-	0.154	-	-	-	0.001	0.151	-	0.095	-
Maine Yankee	1972	0.516	-	-	-	-	-	0.254	0.056	3.569	-	0.267	-
McGuire	1981	0.491	17.464	0.298	0.154	-	-	-	0.271	36.112	0.012	6.586	-
Millstone 2	1975	0.572	0.080	5.661	2.938	3.474	5.587	0.032	2.017	289.984	3.752	41.904	0.751
North Anna 1,2	1978/80	0.736	0.008	0.032	0.566	0.007	0.015	0.418	0.597	128.164	0.013	6.847	0.006
Oconee 1,2,3	1973/74	1.221	0.004	0.367	16.293	0.010	-	6.439	6.550	851.126	-	13.211	-
Palisades	1971	0.382	9.250	0.011	0.154	0.011	0.016	0.088	0.008	272.321	0.037	0.073	-
Point Beach 1,2	1970/72	0.720	0.273	1.806	2.586	1.465	3.226	0.012	0.310	15.877	0.899	7.920	2.409
Prairie Island 1,2	1973/74	0.887	0.004	0.085	0.207	0.035	0.049	0.084	0.088	19.580	0.010	0.355	0.005
R.E. Ginna	1969	0.275	0.002	0.003	0.581	0.004	0.004	0.629	0.235	70.300	0.042	0.525	0.005
Rancho Seco 1	1974	0.384	0.028	0.142	0.139	0.040	0.078	0.002	0.336	51.430	-	2.542	-
Salem 1	1976	0.467	0.001	0.015	0.021	-	0.009	-	0.081	8.329	0.283	0.047	-
Salem 2	1981	0.906	-	-	0.011	-	-	-	0.043	41.159	-	0.003	-
San Onofre 1	1967	0.058	-	0.001	0.500	-	-	-	0.005	2.623	-	0.058	-
San Onofre 2,3	1982/83	0.014	0.025	0.001	-	-	0.001	-	0.003	0.215	0.002	0.009	-
Sequoyah 1,2	1980/82	0.560	0.548	0.622	0.055	-	-	0.441	3.811	195.738	-	10.954	-
St. Lucie 1	1976	0.773	1.228	11.507	2.087	7.289	12.987	11.470	4.700	747.435	3.959	55.130	4.736
Surry 1,2	1972/73	1.251	0.149	0.670	2.472	0.012	0.125	-	3.885	751.100	-	20.646	-
Three Mile Island 1	1974	-	-	-	-	-	-	-	-	-	-	-	-
Three Mile Island 2	1978	-	-	-	18.056	-	-	-	-	-	-	-	-
TMI 2/EPICOR	1978	-	-	-	15.762	-	-	-	-	-	-	-	-
Trojan	1975	0.548	0.011	0.082	0.269	0.061	0.076	0.196	0.205	28.864	0.247	1.758	0.138
Turkey Point 3,4	1972/73	0.868	1.706	0.282	0.622	0.074	0.286	1.913	2.605	728.900	0.172	4.144	0.034
Virgil C. Summer	1982	0.022	-	-	5.180	-	-	-	-	-	-	-	-
Yankee Rowe	1960	0.101	0.079	0.068	0.033	0.80	0.126	0.087	0.037	1.985	1.735	1.302	0.154
Zion 1,2	1973	1.125	2.316	0.944	0.480	48.470	1.188	0.153	13.507	477.514	2.224	10.408	0.095
Total annual electric energy generated (GW a)		21.042											
Normalized activity [TBq (GW a)$^{-1}$]			1.95	1.64	6.39	3.38	1.52	2.48	2.33	317.21	0.78	54.58	1.17

Table 19

Noble gases discharged in airborne effluents from BWRs, 1980-1985
[B5, B20, F1, J3, K1, S1, S2, S3, S5, S9, S10, T3, T4, T5, T8, T9, T10]

Country and reactor	Start-up year	Electricity generating capacity (GW)	Activity (TBq)					
			1980	1981	1982	1983	1984	1985
Finland								
Olkiluoto	1978/79	1.36	0.14	0.00002	0.0	0.0	0.0	0.0
Germany, Federal Rep. of								
Brunsbüttel	1976	0.770	6.1	25.1	23.4	1.9	30.0	19.0
Gundremmingen	1984	2.49	-	-	-	-	0.16	0.021
Isar	1977	0.870	28.0	20.0	13.0	22.0	26.0	27.0
Krummel	1983	1.26	-	-	-	0.0008	0.15	0.95
Philippsburg 1	1979	0.864	21.0	0.2	17.0	28.0	5.3	0.035
Würgassen	1971	0.640	290.0	118.0	17.0	23.0	43.0	11.0
Italy								
Caorso	1978	0.548	3.7	4.0	8.1			
Japan								
Fukushima I-1,2	1971/74							
3,4,5,6	76/78/79	4.696	74.0	63.0	67.0	78.0	1.6	2.4
Fukushima II-1,2	1982/84	2.200	-	-	0.0004	0.0056	0	0
Hamaoka 1,2	1976/78	1.380	0	0	0	0	0	0
Onagawa	1984	0.50	-	-	-	-	0	0
Shimane	1974	0.46	0	0	0	0	0	0
Tokai II-1	1978	1.100	0.12	0.078	0	0.067	0	0
Tsuruga 1	1970	0.357	0.20	0.0044	0.32	0.067	0.003	0.00022
Netherlands								
Dodewaard	1968	0.052	74.4	38.4	38.5	23.68	24.79	
Sweden								
Barsebäck 1	1975	0.59	2.1	400.39	662.43	70.1	0.71	0.16
Barsebäck 2	1976	0.59	2.3	1.702	1.405	7.297	0.90	0.29
Forsmark 1	1980	0.90	0	0.40	11.89	0.22	1.941	71
Forsmark 2	1981	0.90	-	0	0.147	11.5	23.38	232
Oskarshamn 1	1970	0.46	5247	3696.12	1417.02	870	680	533
Oskarshamn 2,3	1974/85	1.65	309	421.22	110	53.51	45	47.6
Ringhals 1	1974	0.75	2800	15000	20000	1500	1040	1280
USSR								
VK-50	1965	0.050	175					
United States								
Big Rock Point	1963	0.075	795.5	728.9	477.3	356.82	5175	2264
Browns Ferry	1973/77	3.195	6142.0	1672.4	10212.0	17761.85	24619.31	979
Brunswick	1975/77	1.58	2564.1	19314.0	17205.0	17857.13	6056.61	313
Cooper	1974	0.764	186.11	91.76	525.4	56.94	51.28	
Dresden 1	1960	0.20	2.6	0	0	0.0	0.0	
Dresden 2,3	1971/72	1.545	1591.0	1383.8	348.8	312.06	68.07	108.7
Duane Arnold 1	1975	0.515	99.9	18.02	3.7	17.16	15.15	8.9
Fitzpatrick	1975	0.8	2841.6	7400.0	7807.0	3118.05	1171.17	511
Grand Gulf	1982	1.198	-	-	0	1.66	4.20	5.60
Hatch 1	1975	0.717	1413.4	1024.9	156.51	709.06	373.16	360.9
Hatch 2	1979	0.795	10.9	7.622	38.48	467.66	84.48	100.4
Humbolt Bay	1963	0.065	0.0	0.0	0.0	0.0	0.0	0.0
Lacrosse	1969	0.050	174.3	186.1	157.6	261.93	407.02	328.7
Lasalle	1982	1.078	-	-	0.128	0.43	20.93	6.158
Millstone 1	1971	0.655	440.3	529.1	308.2	234.40	103.69	
Monticello	1971	0.652	141.71	138.38	267.14	117.92	19.03	100.7
Nine Mile Point	1969	0.61	21.72	22.57	1.89	9.95	37.85	36.42
Oyster Creek	1969	0.62	1154.4	1953.6	847.3	79.32	145.37	
Peach Bottom	1974	2.086	556.1	584.6	484.7	2064.87	2990.85	
Pilgrim	1972	0.664	969.4	196.1	717.8	739.66	0.68	99.83
Quad Cities	1973	1.538	795.5	1184.0	432.9	445.81	95.51	109.3
Susquehanna	1983	1.050	0	0	20.76	3.20	4.36	
Vermont Yankee	1972	0.514	60.31	117.29	113.59	115.66	115.61	
Total annual electric energy generated (GW a)			18.39	19.06	21.38	20.71	22.97	17.09
Normalized activity [TBq (GW a)$^{-1}$]			1576	2958	2000	2300	1893	460
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]					2150 ± 523			

Table 20

Isotopic composition of noble gas discharged from BWRs in the United States, 1982
[T5]

Reactor	Start-up year	Electric energy generated (GW a)	Activity (TBq)							
			Ar-41	Kr-83m	Kr-85m	Kr-85	Kr-87	Kr-88	Kr-89	Kr-90
Big Rock Point	1963	0.041	-	7.067	8.140	-	33.485	22.829	19.980	22.422
Browns Ferry	1973/77	1.964	418.100	-	950.900	492.100	355.940	2471.600	-	-
Brunswick	1975/77	0.551	703.000	-	777.000	-	1883.300	1842.600	-	-
Cooper	1974	0.602	-	16.983	53.280	0.718	78.070	111.000	0.699	-
Dresden 1	1960	-	-	-	-	-	-	-	-	-
Dresden 2,3	1971	1.029	-	-	19.462	0.008	6.068	48.100	-	-
Duane Arnold	1975	0.260	0.013	-	0.035	0.002	0.006	0.032	-	-
Fitzpatrick	1975	0.566	11.803	-	425.500	0.024	714.000	880.600	-	-
Hatch 1	1975	0.329	2.479	-	27.084	-	1.746	10.138	-	-
Hatch 2	1979	0.426	1.702	-	0.178	-	1.173	0.833	-	-
Humbolt Bay	1963	-	-	-	-	-	-	-	-	-
Lacrosse	1968	0.016	-	-	6.993	-	6.845	15.133	-	-
Lasalle	1982	0.053	-	-	-	-	-	-	-	-
Millstone 1	1971	0.465	-	-	6.179	-	16.169	9.731	-	-
Monticello	1971	0.276	-	0.581	0.592	24.383	3.104	1.739	56.610	1.894
Nine Mile Point	1969	0.129	-	-	-	-	-	-	-	-
Oyster Creek	1969	0.229	-	-	36.149	-	122.840	120.990	-	-
Peach Bottom	1974	1.519	-	-	2.875	-	0.335	0.836	0.001	-
Pilgrim	1972	0.375	-	-	62.160	0.002	11.618	68.450	-	-
Quad Cities	1973	0.947	-	-	32.042	-	9.324	41.440	-	-
Susquehanna 1	1983	0.037	-	-	-	0.021	1.177	1.136	-	-
Vermont Yankee	1972	0.476	-	-	0.463	0.002	2.201	1.476	-	-
Total annual electric energy generated (GW a)		10.293								
Normalized activity [TBq (GW a)$^{-1}$]			110.505	2.394	234.114	50.268	315.598	548.947	7.511	2.363

Reactor	Start-up year	Electric energy generated (GW a)	Activity (TBq)							
			Xe-131m	Xe-133m	Xe-133	Xe-135m	Xe-135	Xe-137	Xe-138	Xe-139
Big Rock Point	1963	0.041	0.002	0.115	2.664	32.560	32.079	31.746	182.410	29.933
Browns Ferry	1973/77	1.964	-	-	4551.019	174.640	212.018	-	569.800	-
Brunswick	1975/77	0.551	254.930	240.500	1383.811	1343.100	3163.520	39.960	5143.000	-
Cooper	1974	0.602	0.258	1.587	43.662	14.763	125.061	1.832	74.740	-
Dresden 1	1960	-	-	-	-	-	-	-	-	-
Dresden 2,3	1971	1.029	-	-	61.790	43.660	239.390	-	187.220	-
Duane Arnold	1975	0.260	-	-	0.389	0.429	1.088	-	1.632	-
Fitzpatrick	1975	0.566	1054.500	15.614	747.400	347.430	2208.901	-	939.800	-
Hatch 1	1975	0.329	9.583	1.269	62.533	16.021	10.435	3.123	5.624	-
Hatch 2	1979	0.429	0.061	0.001	3.775	5.106	6.882	0.039	3.959	-
Humbolt Bay	1963	-	-	-	-	-	-	-	-	-
Lacrosse	1968	0.016	0.260	1.380	32.746	2.072	92.131	0.977	7.104	-
Lasalle	1982	0.053	-	-	0.128	-	-	-	-	-
Millstone 1	1971	0.465	-	-	79.187	36.408	51.808	58.090	50.690	-
Monticello	1971	0.276	0.251	0.131	33.855	4.329	2.757	74.000	56.980	5.735
Nine Mile Point	1969	0.129	-	-	0.792	-	1.099	-	-	-
Oyster Creek	1969	0.229	-	-	21.238	66.600	229.030	31.265	218.670	-
Peach Bottom	1974	1.519	0.001	6.401	336.346	3.891	119.147	-	2.209	-
Pilgrim	1972	0.375	-	9.546	373.700	1.136	187.960	-	3.700	-
Quad Cities	1973	0.947	-	-	52.917	103.230	68.111	-	122.470	-
Susquehanna 1	1983	0.037	-	4.185	1.584	6.328	0.455	-	5.772	-
Vermont Yankee	1972	0.476	-	-	1.850	20.572	2.205	-	85.100	-
Total annual electric energy generated (GW a)		10.293								
Normalized activity [TBq (GW a)$^{-1}$]			128.23	27.27	756.96	215.90	656.18	23.42	744.28	3.47

Table 21

Noble gases discharged in airborne effluents from HWRs and LWGRs, 1980-1985
[A1, B19, B36, L1]

Country and reactor	Start-up year	Electricity generating capacity (GW)	Activity (TBq)					
			1980	1981	1982	1983	1984	1985
H W R s								
Argentina								
Atucha 1	1974	0.335	250	46	19	47	4.7	5.5
Embalse	1983		0.0	0.0	0.0	0.0	40.9	156
Canada								
Bruce A	1976/79	2.96	830	610	510	670	800	800
Bruce B	1984	0.75	-	-	-	-	29.0	106
Gentilly	1983	0.685	-	-	-	10.7	25.9	121
Pickering A	1971/73	2.92	240.0	250	220	350	170	190
Pickering B	1983/84	1.03	-	-	-	137.0	170	270
Point Lepreau	1983	0.630	-	-	0.1	3.9	0.0	0.8
Total annual electric energy generated (GW a)			4.531	4.810	4.665	5.752	5.837	7.37
Normalized activity [TBq (GW a)$^{-1}$]			290	188	161	212	212	223
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]					212 ± 48			
L W G R s								
USSR								
Chernobyl 1,2	1977/78	2.00	10400	14900	8940	7360	-	-
Chernobyl 3,4	1981/83	2.00	-	-	1770	2190	-	-
Ignalino 1	1983	1.50	-	-	-	-	-	-
Kursk 1,2	1976/79	2.00	4730	9490	6410	5830	8300	6600
Leningrad 1,2	1973/75	2.00	-	4700	6200	6300	5600	5200
Leningrad 3,4	1979/81	2.00	-	1400	3000	4100	4000	2600
Smolensk 1	1982	1.00	-	-	-	2030	2600	2500
Total annual electric energy generated (GW a)			3.172	3.077	3.583	4.355	3.5	3.5
Normalized activity [TBq (GW a)$^{-1}$]			4710	7927	4778	3998	5857	4828
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]					5466 ± 1365			

Table 22

Activation gases discharged in airborne effluents from GCRs, 1980-1985
[B1, B2, B3, B16, B29, F1, F4, H1, H2, H3, H4, J3, K1, P5, P9, S11]

Country and reactor	Start-up year	Electricity generating capacity (GW)	Activity (TBq)					
			1980	1981	1982	1983	1984	1985
France								
Chinon 2,3	1965/66	0.540	108.8	111	111	140	130	25
Bugey 1	1972	0.540	114.7	113	31	113	123	130
St. Laurent A1,2	1971/72	0.930	126	87	150	151	272	244
Italy								
Latina	1964	0.150	86.7	83.3	82.9			
Japan								
Tokai	1966	0.166	352.0	352.0	293.0	322.0	300.0	270
United Kingdom								
Berkeley	1962	0.276	370	37	74	222	222	310
Bradwell	1962	0.250	37	-	370	481	666	740
Chapelcross	1959	0.140	1200	1200	1200	1200	1200	1200
Dungeness A	1965	0.410	18.5	37	1110	1850	1110	1200
Dungeness B	1983	1.200	-	-	-	22.2	10	20
Hartlepool	1983	1.200	-	-	-	3.7	10	10
Heysham	1983	1.200	-	-	-	10	10	10
Hinkley Point A	1965	0.430	2960	2960	2960	3330	2960	3100
Hinkley Point B	1976	1.040	148	111	111	148	111	70
Hunterston A	1964	0.300	732.6	640.1	647.5	699.3	710.4	
Hunterston B	1976	1.040	113.7	122.0	116.6	112.9	79.9	
Oldbury	1967	0.534		185	222	111	148	130
Sizewell	1966	0.420	2220	1480	1110	1850	1110	1700
Trawsfynydd	1965	0.390	1850	3330	3330	3330	3330	5000
Wylfa	1971	0.840	37	74	74	74	70	
Total annual electric energy generated (GW a)			3.868	4.847	5.382	5.885	6.223	5.738
Normalized activity [TBq (GW a)$^{-1}$]			2692	2246	2228	2407	2018	2432
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]					2318 ± 224			

Table 23

Tritium discharged in airborne effluents from reactors, 1980-1985
[B5, B17, B18, D3, F1, H1, H2, H3, H4, K1, L1, P5, P9, S1, S2, S3, S5, S10, S11, T3, T4, T5, T8, T9, T10]

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
P W R s						
Belgium						
Doel 1-3	-	-	0.1			
Finland						
Lovilsa	-	3.5	3.0	11.0	2.0	2.9
France						
Chooz	-	-				
Blayais 1,2	-					

Table 23, continued

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
Germany, Federal Rep. of						
Biblis A,B	4.6	2.5	2.7	2.3	2.2	3.6
Grafenrheinfeld	-	0.00015	0.05	0.23	0.58	0.64
Grohnde	-	-		-	0.0027	0.045
Neckarwestheim	1.9	0.60	0.70	0.80	0.71	0.42
Obrigheim	0.3	0.20	0.20	0.18	0.28	0.34
Philippsburg 2	-	-	-	-	-	0.14
Stade	1.6	0.70	1.1	1.5	1.4	0.83
Unterweser	0.4	0.40	1.5	1.6	1.5	1.7
Netherlands						
Borssele	0.63	0.68	0.40	0.59	0.59	
United States						
Arkansas 1	4.773	4.625	0.204	0.167	0.008	0.355
Arkansas 2	0.075	0.115	0.244	0.104	0.152	0.127
Beaver Valley	0.176	0.004	0.004	0.729	0.540	0.21
Callaway	-	-	-	-	0.041	0.19
Calvert Cliffs 1,2	1.032	0.215	0.252	1.54	0.097	0.12
Crystal River	0.784	0.574	0.910	0.858	0.603	0.762
Davis-Besse	0.220	0.320	1.314	0.459	0.429	0.607
Diablo Canyon	-	-	-	-	0.0005	0.366
Donald C Cook 1,2	0.041	0.202	0.189	1.35	1.88	0.803
Farley 1	22.681	2.516	4.847	5.18	3.00	0.677
Farley 2	-	4.625	4.218	14.7	7.03	6.14
Fort Calhoun	0.048	3.016	0.192	0.052	0.250	
H.B. Robinson	0.225	0.392	0.045	0.141	0.082	3.275
Haddam Neck	2.313	3.201	1.883	9.10	5.00	
Indian Point 1,2	0.407	0.157	0.229	0.076	0.119	
Indian Point 3	0.182	0.137	0.062	0.040	0.064	
Kewaunee	0.770	0.141	0.298	0.041	0.035	0.315
Maine Yankee	0.117	0.166	0.150	0.196	0.178	0.101
McGuire	-	0.002	0.140	0.084	1.09	1.565
Millstone Pt. 2	31.450	5.180	2.512	5.07	7.84	
North Anna	2.076	1.162	0.307	1.41	0.239	0.284
Oconee	0.395	2.135	0.459	0.470	15.4	1.584
Palisades	0.190	0.238	0.166	0.233	0.209	0.158
Point Beach 1,2	24.161	17.760	37.740	27.8	12.1	2.483
Prairie Island	3.193	2.720	3.574	2.56	3.23	2.697
R.E. Ginna	1.465	2.594	3.574	2.24	3.49	
Rancho Seco	6.549	5.217	2.220	6.48	7.18	1.225
Salem 1	-	0.101	12.173	77.0	4.22	1.894
Salem 2	-	0.042	0.131	31.8	6.51	1.11
San Onofre 1	1.365	0.444	0.203	0.145	0.0	
San Onofre 2,3	-	-	0.049	0.470	8.36	0.295
Sequoyah 1	-	0.037	8.066	31.3	7.99	
St. Lucie 1	13.801	13.690	26.270	3.74	29.9	
St. Lucie 2	-	-	-	15.44	10.7	5.994
Surry 1,2	0.677	2.290	3.141	0.865	1.42	
Three Mile Island 1	0.670	0.00005	0.0000008	0.00003	0.000002	0.00002
Three Mile Island 2	14.578	2.427	4.107	1.50	0.518	0.733
TMI 2/EPICOR	21.616	0.002	0.038	0.001	0.011	
Trojan	0.551	1.491	4.773	5.14	1.80	
Turkey Point 3,4	0.043	0.025	0.052	0.032	0.012	
Virgil C Summer	-	-		0.134	0.008	
Wolf Creek	-	-	-	-	1.53	
Yankee Rowe	0.054	0.114	0.199	0.190	0.350	
Zion 1,2	-	-	0.315	0.685	3.22	
Total annual electric energy generated (GW a)	22.29	26.18	28.46	28.99	32.75	24.19
Normalized activity [TBq (GW a)$^{-1}$]	7.453	3.310	4.740	9.215	4.702	1.9
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			5.9 ± 2.4			

Table 23, continued

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
B W R s						
Finland						
Olkiluoto	0.17	0.41	0.34	0.21	0.17	0.13
Germany, Federal Rep. of						
Brunsbüttel	0.01	0.2	0.3	0.014	0.49	0.47
Gundremmingen	-	-		-	0.026	0.076
Isar	3.1	0.5	0.07	0.57	0.24	0.52
Krummel	-	-		0.0075	0.087	0.51
Philippsburg 1	0.02	0.04	0.35	0.20	0.18	0.12
Würgassen	0.6	1.0	0.8	0.052	0.76	1.0
Italy						
Caorso	-	-	0.15			
Netherlands						
Dodewaard			0.11	0.11		
United States						
Big Rock Point	0.466	0.377	0.232	0.714	1.57	0.932
Browns Ferry	1.876		1.536	1.51	0.570	0.277
Brunswick	0.326	0.651	0.265	0.481	0.368	0.076
Cooper	0.145	0.168	0.268	0.139	0.171	0.0
Dresden 2,3	43.66	11.692	11.581	9.55	3.02	1.795
Duane Arnold	0.134		0.145	0.144	0.252	0.729
Fitzpatrick	0.162	0.246	0.195	0.544	0.295	0.067
Grand Gulf	-	-	-	0.0003	0.0042	0.078
Hatch 1	0.318	0.130	2.638	1.22	1.92	71.04
Hatch 2	1.635	0.225	0.792	0.781	0.321	14.1
Humbolt Bay	0.001	0.001	0.001	0.0014	0.0007	0.0015
Lacrosse	0.240	0.866	0.629	0.851	1.57	1.288
Lasalle	-	-	-	0.0002	0.229	0.085
Millstone 1	3.533	3.504	1.991	2.81	2.85	
Monticello	5.476	4.07	2.405	1.78	0.740	2.705
Nine Mile Point 1	3.996	2.346	1.610	7.44	1.35	1.221
Oyster Creek	0.346	0.119	0.252	0.107	0.113	
Peach Bottom	0.477	1.054	0.921	0.566	0.966	
Pilgrim	1.621	2.845	0.707	2.22	0.065	0.098
Quad Cities	1.628	3.178	4.551	0.788	0.892	1.931
Susquehanna	-	-	1.143	0.109	1.36	
Vermont Yankee	0.611	0.725	0.722	0.544	0.440	
WNP-2	-	-		-	0.0034	0.286
Total annual electric energy generated (GW a)	11.76	9.245	12.28	11.91	12.50	15.94
Normalized activity [TBq (GW a)$^{-1}$]	5.984	3.671	2.881	2.791	1.659	6.249
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			3.4 ± 1.6			

Table 23. continued

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
H W R s						
Argentina						
Atucha	240	210	300	630	200	250
Embalse	-	-		-	7.33	29.5
Canada						
Bruce A	1554	3404	1517	3700	2553	1500
Bruce B	-	-		-	3	99
Gentilly	-	-		0.69	14	53.2
Pickering A	660	592	666	629	430	390
Pickering B	-	-		25	46	144
Point Lepreau	-	-		25	68	110
Total annual electric energy generated (GW a)	4.531	4.810	4.665	5.752	6.408	7.370
Normalized activity [TBq (GW a)$^{-1}$]	541.5	874.5	532.3	871.0	518.3	348.9
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			670 ± 190			
G C R s						
Italy						
Latina	0.07	0.07	0.90			
United Kingdom						
Hunterston A	2.2	2.0	1.6	1.4	1.3	
Hunterston B	2.2	4.1	3.4	4.6	4.6	
Oldbury	0.5	1.6	0.3	1.1	1.0	0.43
Sizewell						
Trawsfynydd	3.7					
Wylfa						
Total annual electric energy generated (GW a)	1.346	1.252	1.41	1.39	1.45	0.379
Normalized activity [TBq (GW a)$^{-1}$]	6.44	6.21	4.40	5.10	4.76	1.135
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			5.4 ± 0.9			

Table 24

Tritium discharged in liquid effluents from reactors, 1980-1985
[B5, B6, B7, B17, B18, B29, D3, F4, H1, H2, H3, H4, K1, L1, P1, P5, P9, S1, S2, S3, S5, S9, S10, S11, T3, T4, T5, T8, T9, T10]

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
P W R s						
Belgium						
Doel 1,2	24.8	22	20	35.6	30.9	47
Tihange	12.4	27.2	38.5	35.3	30.9	49
Finland						
Lovilsa 1,2	3.7	11.0	9.7	9.5	7.1	9.4
France						
Blayais 1,2,3,4	-	4	23	35	78	80
Bugey 2,3,4,5	53	49	61	73	66	79
Chinon B1,2	-	-	-	10	27	22
Chooz	110	111	110	104	111	98
Cruas 1,2,3,4	-	-	-	1	23	57
Dampierre 1,2,3,4	7	51	47	64	72	67
Fessenheim 1,2	28	34	25	33	41	30
Gravelines 1,2,3,4	4	29	32	51	78	95
Paluel 1,2	-	-	-	-	6	31
St. Laurent B1,2	-	5	5	21	26	30
Tricastin 1,2,3,4	10	42	57	55	76	72
Germany, Federal Rep. of						
Biblis A,B	35	28.2	27.5	36.0	32.0	33.0
Grafenrheinfeld	-	-	7.0	19.0	21.0	22.0
Grohnde	-	-		-	0.091	7.2
Neckarwestheim	3	2.8	6.4	11.8	11.0	13.0
Obrigheim	3.3	4.5	4.5	3.2	5.0	5.3
Phillippsburg 2	-	-	-	-	0.0005	13.0
Stade	2.3	2.9	5	8.0	12.0	6.2
Unterweser	8.8	10.0	14.6	19.2	25.0	27.0
Italy						
Trino	37.4	2.2	0.8			
Netherlands						
Borssele	6.3	6.0	7.5	6.18	4.63	
Sweden						
Ringhals 2	13	10	13	11	17	19.5
Ringhals 3	0.7	11	3.9	8.3	16	10.8
Ringhals 4	-	-	0.6	6.2	13	14.6
USSR						
Armenian	1.9					
United States						
Arkansas 1	7.844	16.35	7.622	4.03	0.37	0.36
Arkansas 2	10.693	9.028	5.143	8.81	11.4	8.92
Beaver Valley	1.473	5.18	6.808	17.2	15.2	1.45
Callaway	-	-		-	1.07	21.8
Calvert Cliffs 1,2	18.167	37.0	16.095	27.97	29.1	17.9
Crystal River	7.215	10.027	6.734	7.36	15.5	1.64
Davis Besse	3.996	5.809	2.102	4.22	4.51	2.49
Diablo Canyon	-	-	-	-	0.040	15.8
Donald Cook 1,2	28.934	33.855	45.51	32.75	50.7	42.2
Farley 1	21.090	6.105	12.469	15.24	15.7	13.7
Farley 2	-	23.458	13.238	11.73	13.2	6.73
Fort Calhoun	2.013	8.954	11.369	5.66	8.73	
Fort St. Vrain	-	-	-	13.65	4.59	0.57
Grand Gulf	-	-	-	0.0001	0.027	0.19
H.B. Robinson	6.993	6.882	3.519	8.88	0.496	11.4
Haddam Neck	121.73	195.73	149.85	144.30	135.4	
Indian Point 1,2	10.212	8.917	6.365	12.69	8.21	
Indian Point 3	15.799	23.754	7.178	1.18	21.7	
Kewaunee	8.621	9.287	11.766	10.80	16.3	14.0
Maine Yankee	8.060	7.992	6.845	10.62	6.36	6.81
McGuire	-	0.231	5.92	5.51	23.9	29.8
Millstone Pt. 2	9.916	13.727	10.767	4.48	14.7	
North Anna	14.911	44.4	21.127	59.57	22.9	31.0
Oconee	26.344	18.759	13.098	47.36	47.36	45.9

Table 24, continued

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
Palisades	2.764	10.286	6.623	8.70	2.57	15.9
Point Beach 1,2	28.157	24.124	18.611	19.94	77.7	29.8
Prairie Island	20.091	20.794	22.200	19.24	23.7	25.8
R.E. Ginna	5.920	8.880	11.396	12.95	17.0	
Rancho Seco	0.0005	3.089	2.39	2.75	11.0	3.33
Salem 1	-	18.241	26.714	7.70	12.2	68.5
Salem 2	-	31.191	19.425	8.25	11.4	20.4
San Onofre 1	38.110	10.989	20.165	0.581	1.25	
San Onofre 2,3	-	-	0.33	8.81	16.84	17.6
Sequoya	-	-	-	27.20	67.34	
St. Lucie 1	10.064	12.025	11.877	12.80	8.18	
St. Lucie 2	-	-	-	1.39	8.18	13.5
Surry 1,2	14.245	19.647	33.670	26.53	30.04	
Three Mile Island 1	1.206	0.263	1.45	0.114	0.064	0.021
Three Mile Island 2	0.000022	0.0019	0.0026	0.0014	0.000006	0.00008
Trojan	4.588	3.811	7.4	0.851	6.92	
Turkey Point	27.713	7.215	23.199	26.34	32.75	
Virgil C. Summer	-	-	0.0118	8.40	8.33	
Waterford						0.94
Wolf Creek						6.77
Yankee Rowe	2.161	3.811	6.882	6.22	6.07	
Zion 1,2	27.565	32.190	56.610	15.91	25.38	
Total annual electric energy generated (GW a)	29.69	40.55	44.97	53.09	61.74	57.84
Normalized activity [TBq (GW a)$^{-1}$]	29.43	28.40	25.60	23.42	25.95	25.00
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			27.0 ± 1.8			

Table 24, continued

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
B W R s						
Finland						
Olkiluoto	0.58	0.84	0.77	0.82	1.0	1.2
Germany, Federal Rep. of						
Brunsbüttel	0.09	0.8	1.9	1.1	2.6	0.87
Gundremmingen	-	-	-	-	0.41	1.2
Isar	8.8	10.1	14.6	3.1	1.8	0.47
Krummel	-	-		0.043	0.59	0.76
Philippsburg 1	0.4	0.05	0.8	1.7	2.0	0.90
Würgassen	2.1	1.9	1	0.43	0.79	0.71
Italy						
Caorso	0.2	0.1	9.4			
Netherlands						
Dodewaard	0.6	0.5	0.3	0.22	0.15	
Sweden						
Barsebäck 1,2	0.79	0.75	0.52	0.68	1.0	0.58
Forsmark 1,2	0.14	1.2	1.7	1.0	1.2	1.4
Oskarshamn 1,2	0.97	0.6	0.56	0.71	0.54	0.63
Ringhals 1	1.7	1.8	1.9	1.0	0.7	0.525
United States						
Big Rock Point	0.229	0.116	0.110	0.821	0.041	0.047
Browns Ferry	0.807		0.884	1.18	1.18	1.23
Brunswick	0.474	0.836	1.806	3.85	1.25	0.25
Cooper	0.324	0.309	0.336	0.281	0.266	
Dresden 2,3	0.294	0.224	0.05	0.00005	1.45	0.28
Duane Arnold	-	-	0.00000008		0.00000005	0.0013
Fitzpatrick	0.104	0.152	0.024	0.101	0.176	0.11
Hatch 1	0.525	0.429	3.811	3.50	2.97	1.45
Hatch 2	0.396	0.343	1.362	1.26	0.788	0.67
Humboldt Bay	0.0036	0.006	0.002	0.002	0.0006	0.04
Lacrosse	2.664	2.864	2.190	4.59	4.63	4.74
Lasalle	-	-	0.034	0.157	0.041	0.014
Millstone 1	1.01	0.097	0.229	0.310	0.317	
Monticello	-	0.00002	0.0000001	0.000		
Nine Mile Point	-	0.187	0.215	0.292		
Oyster Creek	5.698	0.988	0.183	0.324	0.381	
Peach Bottom	1.38	1.362	0.877	0.747	1.32	
Pilgrim	1.48	1.262	0.219	0.577	0.544	0.16
Quad Cities	0.381	0.44	0.289	0.144	0.201	0.13
Susquehanna	-	-	0.032	0.332	0.414	
Vermont Yankee	-	0.111	0.259			
WNP	-	-		-	0.02	0.055
Total annual electric energy generated (GW a)	14.17	14.77	15.85	15.78	16.80	18.0
Normalized activity [TBq (GW a)$^{-1}$]	2.227	1.864	2.877	1.801	1.58	1.01
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			2.1 ± 0.5			

Table 24, continued

Country and reactor	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
G C R s						
France						
Bugey 1	1	14	1	1	0.2	17
Chinon A2,3	4	4	2	2	2	0.5
St. Laurent A1,2	16	1	1	6	13	5
Italy						
Latina	0.03	0.3	0.5			
United Kingdom						
Berkeley	2.812	2.96	1.739	0.074	1.30	0.5
Bradwell	2.331	3.108	1.961	0.999	7.99	1.3
Chapelcross	0.09	1.3	0.7	0.555	0.24	
Dungeness A	0.294	0.148	0.74	2.295	0.37	0.8
Dungeness B	-	-		0.111	5.55	46.4
Hartlepool	-	-		0.037	18.87	22.4
Heysman	-	-	-	0.037	16.65	24.7
Hinkley Point A	1.961	1.961	0.666	0.703	0.52	22.6
Hinkley Point B	163.91	204.24	231.62	313.39	338.92	336
Hunterston A	0.777	2.183	1.48	2.183	1.26	
Hunterston B	109.85	144.078	256.41	264.328	301.55	
Oldbury	0.296	0.518	0.962	1.073	1.04	0.8
Sizewell	1.036	1.295	1.295	0.814	1.26	9.9
Trawsfynydd	0.999	11.322	3.7	0.962	0.78	2.4
Wylfa	11.322	11.47	18.574	13.505	12.21	7.0
Total annual electric energy generated (GW a)	3.868	4.847	5.382	5.885	6.223	5.738
Normalized activity [TBq (GW a)$^{-1}$]	81.5	83.3	95.7	103.7	116.4	86.75
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			96 ± 13			
L W G R s						
USSR						
Chernobyl	3.5					
Kursk	2.0					
Total annual electric energy generated (GW a)	0.317	0.308	0.356	0.376	0.580	
Normalized activity [TBq (GW a)$^{-1}$]	1.734					
H W R s						
Argentina						
Atucha	290	410	310	240	410	320
Embalse	-	-		-	3.48	16.1
Canada						
Bruce A	888	740	962	1600	604	1060
Bruce B	-	-	-	-	0.6	21.5
Gentilly	0.4	8.0	0.05	0.78	14	30.7
Pickering A	481	278	370	370	330	330
Pickering B	-	-	-	44	330	380
Point Lepreau	-	-	-	9.1	68	24
Total annual electric energy generated (GW a)	4.531	4.810	4.665	5.752	6.408	7.370
Normalized activity [TBq (GW a)$^{-1}$]	365.8	296.3	351.4	212.6	234.6	296.1
Average normalized activity, 1980-1984 [TBq (GW a)$^{-1}$]			290 ± 68			

Table 25

Carbon-14 discharged from reactors, 1980-1985
[B17, 03, R1, W1]

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
P W R s a/						
Finland						
Loviisa 1					150	
Germany, Federal Rep. of						
Biblis A			50 (110)	41	41	17
Biblis B			22	22	50	11
Grafenrheinfeld	-	-	23	310	78	91
Grohnde	-	-	-	-	0.09	17
Neckarwestheim	11 (96)	22 (110)	44	26	15	30
Obrigheim	22	22	26	40	17	13
Philippsburg 2	-	-	-	-	-	5.6
Stade	41 (78)	80	100	130	66	49
Unterweser	26	33	41	22	42	75
USSR						
Armenian	400-500	400-500	400-500	400-500	400-500	
Kola 3		780	780	780	780	
Kola 4					510	
Novovoronezh 3	140	140	140	140	140	
Novovoronezh 4	140-540	140-540	140-540	140-540	140-540	
Total annual electric energy generated (GW a)	3.654	4.186	7.285	7.020	7.270	7.856
Normalized activity [GBq (GW a)$^{-1}$]	329	482	297	333	283	39
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			345 ± 80			
B W R s						
Finland						
TVO 1					300	
Germany, Federal Rep. of						
Brunsbüttel	30	240	80	0.9	240	260
Gundremmingen	-	-	-	-	300	770
Isar	-	180	4.8	340	310	320
Krummel	-	-	-	-	550	190
Philippsburg 1	-	6.3	91	200	220	250
Würgassen	270	270	88	15	280	360
Total annual electric energy generated (GW a)	1.195	1.521	1.275	1.880	3.106	4.717
Normalized activity [GBq (GW a)$^{-1}$]	251.2	457.7	206.9	295.7	434.6	455.8
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			330 ± 110			

Table 25, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
H W R s						
Argentina						
Atucha	2300	2700	1800	590	450	370
Total annual electric energy generated (GW a)	0.2488	0.3021	0.2001	0.2668	0.195	0.1678
Normalized activity [GBq (GW a)$^{-1}$]	9244	8938	8996	2195	2308	8766
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			6336 ± 3333			
L W G R s						
USSR						
Chernobyl						
Ignalino 1						
Kursk 1,2,3						
Leningrad 1,2,3,4						
Smolensk 1						
Average normalized activity, 1980-1983 [GBq (GW a)$^{-1}$]			1300			

a/ Carbon dioxide, carbon monoxide and hydrocarbon bound values in parentheses.

Table 26

Iodine-131 discharged in airborne effluents from reactors, 1980-1985
[A1, B5, B17, B18, B19, B36, D1, F1, H1, H2, H3, H4, J3, K1, L1, P5, P9, S1, S2, S3, S5, S9, S10, S11, T3, T4, T5, T8, T9, T10]

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
P W R s						
Belgium						
Doel 1,2	0.26	0.009	0.33			
Tihange	1.4	4.6	1.9			
Finland						
Loviisa	0.002	0.081	0.063	0.49	0.002	0.0067
Germany, Federal Rep. of						
Biblis A,B	0.44	0.31	0.17	0.15	0.12	0.10
Grafenrheinfeld	-	-	-	0.00005	0.0001	0.00007
Grohnde	-	-	-	-	0.0008	-
Neckarwestheim	0.23	0.001	0.15	0.008	0.033	0.018
Obrigheim	0.002	0.003	0.01	0.056	0.005	0.005
Philippsburg 2	-	-	-	-	-	0.003
Stade	0.004	0.003	0.008	0.005	0.009	0.039
Unterweser	0.08	0.005	0.005	0.005	0.005	0.0005
Japan						
Genkai	n/d	0.0024	n/d	n/d	0.006	-
Ikata	0.0052	0.0078	0.0029	0.0008	0.03	0.00048
Mihama	1.3	0.089	0.067	0.034	0.09	0.028
Ooi	0.016	0.26	0.063	0.0056	0.003	0.0049
Sendai	-	-	-	n/d	n/d	n/d
Takahama	0.010	0.0021	0.0034	0.0002	0.09	0.021
Netherlands						
Borssele	-	0.002	0.02			
Sweden						
Ringhals 2	0.01	0.088	0.0011	0.022	1.1	0.053
Ringhals 3	-	0.01	0.0022	0.0093	0.025	0.0024
Ringhals 4	-	-	0.00012	0.00006	0.000096	0.0036
USSR						
Armenian 1,2	7.8	2.1	3.4	8.8	18	7.5
Kola 1,2	0.257	0.270	0.365	0.945	-	-
Nikolaev 1	-	-	-	-	0.67	0.035
Novovoronezh 1,2	14	7.0	3.3	4.6	2.1	-
Novovoronezh 3,4	0.424	0.043	0.046	0.17	0.078	-
Novovoronezh 5	-	0.83	0.42	0.24	0.16	-
Rovno 1,2	-	0.048	0.196	1.480	1.30	0.086
United States						
Arkansas One-1	6.14	0.2042	0.0477	0.0151	0.043	0.118
Arkansas One-2	0.236	0.5069	0.1702	0.202	0.008	0.111
Beaver Valley	0.0164	0.2372	0.1461	1.813	0.194	0.012
Callaway	-	-	-	-	0.00003	0.011
Calvert Cliffs 1,2	2.05	1.3764	1.4467	3.522	2.20	1.92
Crystal River	0.246	0.3345	0.0339	0.033	0.0047	0.010
Davis Besse	0.0744	1.9906	0.1943	0.272	0.061	0.019
Diablo Canyon	-	-		-	0.0	0.0089
Donald Cook 1,2	0.470	1.7242	3.8480		0.437	3.81
Farley 1	0.00696	0.1110	3.3559	0.70	0.213	0.206
Farley 2	-	0.0010	0.0007	0.002	0.055	0.0096
Fort Calhoun 1	0.0847	0.1306	0.0559	0.033	0.466	
H.B. Robinson	0.0006	0.0007	0.0164	0.485	0.0083	0.50
Haddam Neck	0.0747	0.3275	0.0061	0.181	2.024	
Indian Point 1,2	2.24	1.1988	1.3394	0.596	0.178	
Indian Point 3	0.455	0.0614	0.0755	0.002	0.141	
Kewaunee	0.00673	0.0032	0.0005	0.0005	0.126	0.0014
Maine Yankee	0.0577	0.0155	0.0032	0.0009	0.089	0.011
McGuire	-	-	0.0017	0.051	0.640	0.603
Millstone Pt. 2	0.233	3.9220	3.9960	0.862	4.07	
North Anna	0.444	17.2420	0.8066	3.55	3.02	0.108
Oconee	-	9.2500	7.7330	2.52	0.377	0.138
Palisades	0.929	1.4985	0.8362	1.22	0.031	0.759
Point Beach 1,2	0.0369	0.1661	0.3130	0.659	0.017	0.127
Prairie Island 1,2	0.0651	0.0134	0.1369	0.488	0.050	0.271
R.E. Ginna	0.129	0.0414	0.0324	0.184	0.060	

Table 26, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
Rancho Seco	0.2557	0.1421	0.0142	0.075	0.870	0.237
Salem 1	0.1447	0.3445	0.1332	0.892	0.0191	0.407
Salem 2	0.0020	0.0309	0.0958	0.009	0.047	0.017
San Onofre 1	0.0086	0.0884	.	0.0001	0.0003	
San Onofre 2,3	-	-	0.0006	5.77	15.10	16.40
Sequoyah	0.0015	0.0296	0.0362	0.025	0.186	
St. Lucie 1,2	1.1951	2.1645	10.1750	3.73	20.05	4.81
Surry 1,2	0.5994	1.6650	2.1127	2.76	27.90	
Three Mile Island 1	-	-	-	0.0	0.0	
Three Mile Island 2	-	-	-	0.0	0.0	
TMI 2/EPICOR	-	-	-	0.0	0.0	
Trojan	0.4218	1.3949	0.2035	0.068	0.143	
Turkey Point 3,4	1.9203	1.0360	8.1030	5.25	1.01	
Virgil C. Summer	-	-	-	0.0008	0.00007	
Wolf Creek	-	-	-	-	-	0.002
Yankee Rowe	0.0023	0.0062	0.0111	0.114	0.231	
Zion 1,2	0.0220	0.1905	0.2409	0.189	0.177	
Total annual electric energy generated (GW a)	29.34	32.90	36.79	41.09	44.94	35.65
Normalized activity [GBq (GW a)$^{-1}$]	1.54	1.91	1.63	1.37	2.31	1.07
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			1.75 ± 0.33			

Table 26, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
B W R s						
Finland						
Olkiluoto	0.0042	0.0096	0.0036	0.075	0.0038	0.0027
Germany, Federal Rep. of						
Brunsbüttel	0.04	0.23	0.41	0.006	0.14	0.20
Gundremmingen						0.093
Isar	0.08	0.002	0.004	0.003	0.002	0.011
Krummel	-			0.00003	0.006	0.011
Philippsburg 1	-	-	0.02	2.1	0.011	0.019
Würgassen	2.2	1.4	0.54	0.76	1.9	0.8
Italy						
Caorso	0.01	0.06	0.01			
Japan						
Fukushima I, 1-6	1.9	2.3	1.9	1.4	0.35	0.31
Fukushima II, 1,2	-	n/d	n/d	0.0063	0.0002	0.000006
Hamaoka 1,2	0.010	0.0067	0.0048	0.0033	0.004	0.0016
Shimane	n/d	n/d	n/d	n/d	n/d	n/d
Tokai 2	0.067	0.036	0.0078	0.0078	n/d	n/d
Tsuruga	0.027	0.0093	0.011	0.0059	0.0002	0.0004
Netherlands						
Dodewaard	0.09	0.07	0.04			
Sweden						
Barsebäck 1	0.06	0.11	0.48	0.005	0.019	0.027
Barsebäck 2	0.02	0.031	0.02	0.028	0.0016	0.01
Forsmark 1	0.02	0.12	0.0028	0.0079	0.013	0.013
Forsmark 2	-	0.002	0.00055	0.068	0.12	0.66
Forsmark 3	-	-	-	-	-	0.034
Oskarshamn 1	0.4	0.19	1.4	0.68	0.4	0.20
Oskarshamn 2,3	0.3	0.36	0.26	0.14	0.044	0.051
Ringhals 1	1.4	11	15	1.3	2.5	5.7
United States						
Big Rock Point	0.0396	0.0881	0.0093	0.067	4.59	2.56
Browns Ferry	2.4346	1.4800	3.9590	7.25	5.51	0.76
Brunswick	9.9160	9.8420	32.7450	108.04	11.14	0.81
Cooper	0.6327	0.1946	4.1440	0.673	0.288	
Dresden 1	0.1343	0.0966	0.0002	0.00002	0.023	
Dresden 2,3	130.6100	70.9400	21.4970	13.43	1.39	2.63
Duane Arnold	1.6576	0.5254	0.2068	0.0870	0.075	0.030
Fitzpatrick	2.8379	4.2550	16.0580	10.43	3.74	1.21
Grand Gulf	-	-	-	0.000001	0.0001	0.0081
Hatch 1	47.3600	7.4000	6.5490	8.88	1.61	0.112
Hatch 2	0.5328	0.3189	2.5012	0.448	30.8	0.110
Humboldt Bay	-	-	-	0.0	0.0	0.0
Lacrosse	0.1617	0.1510	0.1025	0.228	0.147	1.83
Lasalle	-	-	-	0.009	0.131	0.314
Millstone 1	7.9180	2.7824	3.6704	1.08	0.788	
Monticello	0.7511	0.5735	2.6936	0.925	0.221	2.49
Nine Mile Point	0.4403	0.2383	0.0792	0.164	0.400	0.833
Oyster Creek	34.9650	33.8550	32.5600	0.345	12.25	
Peach Bottom	1.0878	1.2358	1.1174	1.43	3.46	
Pilgrim	3.2486	1.9388	0.8732	1.20	0.0029	1.22
Quad Cities	12.2470	18.9440	10.6560	7.25	1.80	1.78
Susquehanna	-	-	-	0.027	0.448	
Vermont Yankee	0.4107	0.0603	0.0053	0.005	0.153	
WNP-2	-	-	-	-	-	0.075
Total annual electric energy generated (GW a)	17.33	17.63	19.19	18.63	19.97	29.18
Normalized activity [GBq (GW a)$^{-1}$]	15.23	9.690	8.316	9.044	4.230	0.824
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			9.3 ± 4.9			

Table 26, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
H W R s						
Argentina						
Atucha 1	0.20	0.42	0.019	0.14	0.0092	0.59
Embalse	-	-	-	-		0.231
Canada						
Bruce A	0.130	0.104	1.184	0.359	2.801	0.05
Bruce B	-	-	-	-	0.004	0.052
Gentilly						
Pickering A	0.155	0.063	0.070	0.070	0.13	0.056
Pickering B	-	-	-	0.0	0.157	0.040
Point Lepreau				0.0096	0.0	0.0042
Total annual electric energy generated (GW a)	4.531	4.810	4.586	5.645	5.729	7.006
Normalized activity [GBq (GW a)$^{-1}$]	0.1070	0.1220	0.2776	0.1027	0.5378	0.1461
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			0.23 ± 0.08			
L W G R s						
USSR						
Chernobyl 1,2,3	189	300	118	41.4	-	-
Kursk 1,2,3	25.7	28.8	112	45.1	40	47
Smolensk 1	-	-	-	72.1	-	-
Leningrad 1,2,3,4	-	120	110	74	89	40
Total annual electric energy generated (GW a)	3.172	3.077	3.583	4.355	3.5	3.5
Normalized activity [GBq (GW a)$^{-1}$]	67.69	150.0	94.89	53.27	36.86	24.86
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			80 ± 40			

Table 26, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
G C R s						
France						
Bugey 1						
Chinon 2,3						
St. Laurent A1,2						
Italy						
Latina	0.003	0.0001	0.002			
Japan						
Tokai 1	0.0018	0.0035	0.003	0.0021	0.0004	0.0017
United Kingdom						
Berkeley						
Bradwell						
Chapelcross						
Dungeness A						
Dungeness B	-	-	-	1.7	1.8	1.9
Hartlepool	-	-	-	0.11	0.22	0.2
Heysham	-	-	-	0.89	1.22	0.9
Hinkley Point A						
Hinkley Point B	0.518	0.481	0.481	0.518	0.407	0.4
Hunterston A						
Hunterston B					0.37	
Oldbury						
Sizewell						
Trawsfynydd						
Wylfa						
Total annual electric energy generated (GW a)	0.8215	0.8899	0.8871	1.363	2.372	1.647
Normalized activity [GBq (GW a)$^{-1}$]	0.64	0.55	0.91	3.21	1.86	2.07
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			1.4 ± 1.1			

Table 27

Isotopic composition of iodine discharged from reactors in the United States, 1982
[T5]

Reactor	Activity (GBq)				
	I-131	I-132	I-133	I-134	I-135
P W R s					
Arkansas 1	0.0477	-	0.0016	-	-
Arkansas 2	0.1702	0.0003	0.0003	-	0.0019
Beaver Valley	0.1462	-	0.2982	-	-
Calvert Cliffs 1,2	1.4467	0.0002	0.3996	-	0.9324
Crystal River	0.0339	-	1.5873	-	-
Davis-Besse	0.1943	-	0.0062	-	-
Donald Cook 1,2	3.8480	-	0.3996	-	-
Farley 1	3.3559	-	15.2810	-	-
Farley 2	0.0007	-	103.9794	-	-
Fort Calhoun	0.0559	-	0.0123	-	0.0015
H.B. Robinson	0.0164	-	0.0005	-	0.0001
Haddam Neck	0.0061	-	0.0054	-	-
Indian Point 1,2	1.3394	-	0.5846	-	4.1440
Indian Point 3	0.0755	-	0.0267	-	-
Kewaunee	0.0005	0.0001	0.0005	-	0.0001
Maine Yankee	0.0032	-	0.0148	-	-
McGuire	0.0017	-	0.0113	-	-
Millstone Pt. 2	3.9960	0.0001	0.5328	-	0.2002
North Anna	0.8066	0.0062	0.1598	0.0044	0.0140
Oconee	7.7330	-	1.1100	-	0.6179
Palisades	0.8362	0.0662	0.3485	0.0048	0.1258
Point Beach 1,2	0.3130	0.1021	0.0540	0.0011	0.0081
Prairie Island 1,2	0.1369	-	0.0244	-	-
R.E. Ginna	0.0324	0.4181	0.0411	0.0037	0.0064
Rancho Seco	0.0142	0.2834	0.0112	-	-
Salem 1	0.1332	-	0.1502	-	-
Salem 2	0.0958	-	0.0245	-	-
San Onofre 1	-	-	-	-	-
San Onofre 2,3	0.0006	-	0.0003	-	-
Sequoyah	0.0362	-	0.0091	-	-
St. Lucie	10.1750	0.0013	7.1780	-	1.7168
Surry 1,2	2.1122	0.1010	0.3996	-	0.0685
Three Mile Island 1	-	-	-	-	-
Three Mile Island 2	-	-	-	-	-
Trojan	0.2035	0.0810	0.1702	-	0.3774
Turkey Point	8.1030	-	3.8480	-	0.3885
Virgil C. Summer	-	-	-	-	-
Yankee Rowe	0.0111	-	0.0028	-	0.0022
Zion 1,2	0.2409	0.0030	0.0234	-	0.0052
Total annual electric energy generated (GW a)	21.042				
Normalized activity [GBq (GW a)$^{-1}$]	2.60	0.05	6.50	0.00067	0.41

Table 27. continued

Reactor	Activity (GBq)				
	I-131	I-132	I-133	I-134	I-135
B W R s					
Big Rock Point	0.0929	0.7400	0.8695	1.5429	1.7723
Browns Ferry	3.9590	-	1.8241	-	2.1053
Brunswick	32.7450	4.0330	148.3700	6.7340	83.9900
Cooper	4.1440	-	1.0693	-	4.0330
Dresden 1	0.0002	-	0.0001	-	0.0796
Dresden 2,3	21.4970	-	120.6200	-	202.0200
Duane Arnold	0.2068	-	0.3508	-	0.3052
Fitzpatrick	16.0580	-	68.4500	-	82.8300
Hatch 1	6.5490	-	1.5318	-	16.2430
Hatch 2	2.5012	-	0.5217	-	0.0263
Humbolt Bay	-	-	-	-	-
Lacrosse	0.1025	-	0.0892	-	0.0537
Lasalle	-	-	-	-	-
Millstone 1	3.6702	-	16.0210	-	32.5970
Monticello	2.6936	-	3.1487	-	1.7279
Nine Mile Point	0.0792	-	0.2875	-	0.3774
Oyster Creek	32.5600	-	137.2700	-	216.4500
Peach Bottom	1.1174	-	28.3050	-	17.6860
Pilgrim	0.8732	-	3.3929	-	3.9590
Quad Cities	10.6560	-	46.9900	-	743.7000
Susquehanna	-	-	-	-	-
Vermont Yankee	0.0053	-	0.0357	-	0.7141
Total annual electric energy generated (GW a)	10.29				
Normalized activity [GBq (GW a)$^{-1}$]	13.56	0.46	56.27	0.80	137.03

Table 28

Particulates discharged in airborne effluents from reactors, 1980-1985
[A1, B5, B9, B17, B18, B19, B36, D3, F1, F4, H1, H2, H3, H4, J3, K1, L1, P5, P9, S1, S2, S3, S5, S9, S10, S11, T3, T4, T5, T8, T9, T10]

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
P W R s						
Finland						
Loviisa	0.136	0.0442	0.145	0.0568	0.136	0.178
France a/						
Blayais 1,2,3,4	-	0.2	0.3	0.5	1.1	2.5
Bugey 2,3,4,5	0.9	0.9	0.7	0.7	1.9	0.7
Chinon B1,2	-	-		0.002	1.9	0.3
Chooz	2.6	0.4	0.6	0.2	0.2	0.07
Cruas 1,2,3,4	-	-	-	0.02	0.07	0.07
Dampierre 1,2,3,4	0.1	0.3	0.8	3.0	1.0	1.5
Fessenheim 1,2	0.5	0.3	0.7	0.04	0.07	0.12
Gravelines 1-6	1.6	0.3	0.2	0.3	0.4	1.9
Paluel 1,2	-	-	-	-	0.06	0.26
St. Laurent B1,2	-	0.1	0.4	0.9	1.6	0.7
Tricastin 1,2,3,4	0.1	0.1	0.3	0.4	0.8	0.6
Germany, Federal Rep. of						
Biblis A,B	0.080	0.006	0.054	1.0	0.084	0.31
Grafenrheinfeld	-	-	-	0.0004	0.002	0.002
Neckarwestheim	0.025	0.012	0.019	0.15	0.016	0.014
Obrigheim	0.072	0.17	0.10	0.12	0.14	0.024
Stade	0.022	0.009	0.016	0.27	0.045	0.028
Unterweser	0.080	0.056	0.025	0.014	0.004	0.008
Netherlands						
Borssele	0.037	0.037	0.037	0.037	0.037	
Sweden						
Ringhals 2	0.016	0.01196	0.00955	0.04338	0.0122	0.0077
Ringhals 3	-	0.00907	0.00058	0.00379	0.0024	0.0039
Ringhals 4	-	-	0.00182	0.00246	0.0022	0.0011
USSR						
Armenian 1,2	4.3	2.1	3.4	8.8	18	17.5
Kalinin 1	-	-	-	-	-	-
Kola 1,2	0.26	1.35	0.65	0.30	9.6	0.42
Kola 3,4	-	0.10	0.09	-	9.8	0.19
Nikolaev 1,2	-	-	-	0.15	0.22	0.19
Novovoronezh 1,2	9.6	29	6.7	3.7	3.2	-
Novovoronezh 3,4	3.77	3.9	1.8	3.7	2.4	-
Novovoronezh 5	0.044	0.022	0.081	0.036	0.02	0.074
Rovno 1,2	-	0.10	1.30	0.56	0.37	3.6
Zaporozhe 1	-	-	-	-	-	-
United States						
Arkansas 1	-	0.002	0.022	0.106	0.0325	0.0118
Arkansas 2	0.019	0.015	0.012	0.00414	0.00304	0.0066
Beaver Valley	0.054	0.016	0.023	0.087	0.036	0.0051
Callaway	-	-	-	-	0.00007	0.0005
Calvert Cliffs 1,2	0.703	0.359	5.361	11.55	1.753	0.393
Crystal River	0.004	0.324	0.085	0.013	0.00058	1.089
Davis Besse	-	0.152	0.001	0.0015	0.00070	0.0
Diablo Canyon	-	-	-	-	0.00044	0.0
Donald Cook 1,2	2.076	11.411	0.888	1.37	0.338	2.756
Farley 1	0.081	22.977	0.007	0.0047	0.0044	0.0003
Farley 2	-	0.118	0.002	0.0003	0.00202	0.0003
Fort Calhoun	0.005	0.002	0.002	0.0009	0.0093	
H.B. Robinson	0.041	0.012	0.005	0.0012	0.00087	0.301
Haddam Neck	0.222	0.146	0.014	0.0147	0.0669	
Indian Point 1	0.133	0.437	0.204	0.175	0.152	
Indian Point 3	0.481	0.073	0.083	0.00174	0.0059	
Kewaunee	0.003	0.001	0.002	0.0075	0.0262	0.383
Maine Yankee	0.012	0.007	0.001	0.0016	0.805	0.0031
McGuire	-	-	0.034	0.0094	0.0204	0.0148
Millstone Pt. 2	0.485	-	7.807	0.378	31.9	
North Anna	0.022	0.555	0.485	322.6	51.5	7.69
Oconee	1.288	2.738	8.662	0.628	0.036	0.044
Palisades	0.092	0.037	0.015	0.046	0.352	0.444
Point Beach 1,2	0.010	7.345	-	39.49	0.201	0.209

Table 28, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
Prairie Island	0.003	0.003	0.001	0.0012	0.0031	0.0148
R.E. Ginna	0.204	0.176	0.471	0.0008	0.00302	
Rancho Seco	0.113	0.030	0.959	0.0083	0.0046	0.0559
Salem 1	7.884	17.564	0.157	1.55	0.000	2.889
Salem 2	-	0.203	0.048	1.30	0.152	3.294
San Onofre 1	31.108	0.348	-	0.00009	0.0001	
San Onofre 2,3	-	-	0.001	0.0027	0.631	2.263
Sequoyah	0.094	0.451	4.515	0.0576	0.599	
St. Lucie 1	1.099	0.681	5.180	0.0029	0.0020	
St. Lucie 2	-	-	-	-	0.0002	
Surry 1,2	0.085	0.751	6.092	2.21	1.05	
Three Mile Island 1	0.011	0.019	0.006	0.0024	0.00000005	
Three Mile Island 2	0.021	0.001	0.002	0.000009	0.0002	
TMI 2/EPICOR	-	-	-	0.000006	0.000	
Trojan	0.507	1.443	0.333	0.0901	0.0588	
Turkey Point	0.688	0.052	0.037	0.0692	172.06	
Virgil C. Summer	-	-		0.001	0.0003	
Wolf Creek	-			-		0.0472
Yankee Rowe	0.001	0.002	0.010	15.95	114.71	
Zion 1,2	0.089	0.272	2.930	0.813	2.065	
Total annual electric energy generated (GW a)	29.10	39.35	41.77	49.81	56.54	53.82
Normalized activity [GBq (GW a)$^{-1}$]	2.514	2.703	1.306	8.40	7.80	1.01
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			4.5 ± 2.9			

Table 28, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
B W R s						
Finland						
Olkiluoto	0.511	0.203	0.203	0.348	0.125	1.027
Germany, Federal Rep. of						
Brunsbüttel	0.47	0.015	0.029	0.059	0.056	0.021
Isar	0.64	0.31	0.88	0.43	0.20	0.150
Krummel	-	-	-	0.0001	0.020	0.052
Philippsburg 1	0.14	0.17	0.32	0.037	0.065	0.11
Würgassen	1.6	1.2	1.9	2.1	0.51	0.22
Netherlands						
Dodewaard	0.074	0.037	0.037	0.037	0.037	
Sweden						
Barsebäck 1	0.43	1.452	2.071	1.040	0.112	3.965
Barsebäck 2	0.11	0.201	0.234	0.0994	0.0622	0.201
Forsmark 1	0.03	0.0731	0.0321	2.784	0.108	15.438
Forsmark 2,3	-	0.0124	0.465	0.797	6.37	4.655
Oskarshamn 1	0.031	28.425	240.49	844.37	26.4	6.146
Oskarshamn 2	0.025	6.866	15.105	6.927	1.88	1.499
Ringhals 1	0.26	39.876	1.216	2.303	11.9	0.933
United States						
Big Rock Point	1.048	0.138	0.081	47.4	828.0	126.6
Browns Ferry	0.354	2.116	0.777	4.25	0.898	0.202
Brunswick	74.185	23.051	40.885	13.9	1.65	0.506
Cooper	4.991	0.209	1.591	0.181	0.145	
Dresden 1	0.406	0.271	0.012	0.028	0.0389	
Dresden 2,3	276.39	194.25	13.653	9.56	3.411	3.146
Duane Arnold	1.487	0.677	0.174	0.334	0.428	0.299
Fitzpatrick	1.754	6.105	12.469	3.633	4.03	0.66
Grand Gulf	-	-	-	-	0.0067	0.0196
Hatch 1	0.370	0.444	0.259	0.335	0.725	0.293
Hatch 2	0.007	0.030	0.026	0.147	0.0122	0.113
Humboldt Bay	0.019	0.014	0.004	0.0101	0.0047	0.0051
Lacrosse	0.327	0.474	0.206	0.172	0.108	0.0956
Lasalle	-	-	0.154	0.656	107.2	82.18
Millstone 1	4.366	2.694	4.063	1.194	1.474	
Monticello	0.296	0.703	0.581	0.709	0.863	1.189
Nine Mile Point 1	0.503	0.313	0.924	0.231	0.245	0.450
Oyster Creek	11.285	49.025	5.920	5.34	4.31	
Peach Bottom	-	0.315	0.326	3.82	6.60	
Pilgrim	0.599	0.603	0.770	0.536	0.188	0.114
Quad Cities	9.583	28.046	4.588	213.9	1.39	1.702
Susquehanna	-	-	0.032	0.0078	0.098	
Vermont Yankee	0.218	0.107	0.048	0.194	0.181	
WNP 2	-	-	-	8.495	8.495	6.956
Total annual electric energy generated (GW a)	13.99	14.53	15.15	15.25	16.29	16.40
Normalized activity [GBq (GW a)$^{-1}$]	28.13	26.72	23.14	76.59	61.99	15.85
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			43.3 ± 24.4			

Table 28, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
G C R s						
France						
Bugey 1	0.8	0.5	0.4	0.3	0.3	0.2
Chinon 2,3	0.3	0.1	0.3	0.3	0.2	0.1
St. Laurent A1,2	7.4	1.7	1.0	0.8	1.2	1.2
United Kingdom						
Berkeley	0.111	0.037	0.037	0.037	0.037	0.02
Bradwell	0.037	0.037	0.037	0.074	0.185	0.09
Dungeness A	0.037	0.037	0.333	0.296	0.259	0.24
Dungeness B	-	-	-	0.111	0.111	0.12
Hartlepool	-	-		0.37	0.037	0.03
Heysham	-	-	-	0.37	0.185	0.04
Hinkley Point A	0.333	0.296	0.296	0.333	0.296	0.34
Hinkley Point B	0.925	1.132	0.629	0.518	0.518	0.51
Hunterston A	0.074	0.037	0.037	0.037	0.037	
Hunterston B	2.96	2.59	0.74	0.74	0.740	
Oldbury	0.148	0.333	0.185	0.148	0.111	0.23
Sizewell	0.37	0.333	0.222	0.444	0.296	0.51
Trawsfynydd	0.296	0.407	0.37	0.333	0.370	0.51
Wylfa	0.296	0.259	0.111	0.074	0.074	0.14
Total annual electric energy generated (GW a)	3.868	4.847	5.382	5.885	6.223	5.738
Normalized activity [GBq (GW a)$^{-1}$]	2.86	1.61	0.87	0.79	0.80	0.74
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			1.39 ± 0.79			
L W G R s						
USSR						
Chernobyl 1,2	9.3	-	-	-	-	-
Kursk 1,2	-	-	-	140	120	140
Leningrad 1,2,3,4		6.5	8.6	10	6.8	9.4
Smolenks 1	-	-	-	1.61	-	-
Total annual electric energy generated (GW a)	3.172	3.077	3.583	4.355	3.5	3.5
Normalized activity [GBq (GW a)$^{-1}$]	2.93	2.11	2.40	34.81	36.22	42.68
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			15.69 ± 16.19			

Table 28, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
H W R s						
Argentina						
Atucha 1	0.016	0.014	0.0074	0.0086	0.0046	0.022
Canada						
Bruce A	0.056	0.093	0.100	0.048	0.060	0.044
Bruce B	-	-	-	-	0.117	0.210
Gentilly						
Pickering A	0.118	0.170	0.089	0.020	0.022	0.027
Pickering B	-	-	-	-	0.012	0.013
Point Lepreau						
Total annual electric energy generated (GW a)	4.283	4.508	4.386	4.742	4.964	5.956
Normalized activity [GBq $(GW\ a)^{-1}$]	0.0406	0.0584	0.0431	0.0143	0.0425	0.0531
Average normalized activity, 1980-1984 [GBq $(GW\ a)^{-1}$]			0.040 ± 0.016			

a/ Reported data for France includes halogens with particulates.

Table 29

Liquid releases excluding tritium from reactors, 1980-1985
[B5, B9, B16, B17, B18, B29, C4, F1, F4, H1, H2, H3, H4, J3, K1, L1, P1, P5, P9, S1, S2, S3, S5, S9, S10, S11, T3, T4, T5, T8, T9, T10, V5]

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
P W R s						
Finland						
Loviisa	17.819	2.757	13.474	22.318	20.463	18.167
Belgium						
Doel 1,2,3	98.0	57.0	33.0			
Tihange	56.0	294.0	21.5			
France						
Blayais 1,2	-	81	109	176	213	87
Bugey 2,3,4,5	566	392	130	140	139	177
Chinon B.1,2	-	-		40	78	61
Chooz	9	14	11	9	13	8
Cruas 1-4	-	-	-	45	16	26
Dampierre 1-4	59	337	137	292	226	222
Fessenheim 1,2	262	122	52	105	76	37
Gravelines 1-4	148	218	303	370	380	120
Paluel 1,2	-	-		-	5	98
St. Laurent B1,2	-	100	78	128	187	369
Tricastin 1-4	77	446	170	130	120	140
Germany, Federal Rep. of						
Biblis A,B	11.1	5.9	5.5	3.3	3.7	2.4
Grafenrheinfeld	-	0.0016	0.05	0.10	0.065	0.035
Grohnde	-	-	-	-	0.054	0.11
Neckarwestheim	0.3	0.36	0.06	0.12	0.13	0.30
Obrigheim	3.0	1.7	1.5	2.9	2.0	0.77
Philippsburg 2	-	-		-	0.0006	0.047
Stade	3.0	1.6	1.0	1.1	0.92	1.2
Unterweser	1.8	0.38	0.42	0.65	0.017	0.72
Italy						
Trino	0.44	12.6	15.5			
Japan						
Genkai 1,2	n/d	n/d	n/d	n/d	n/d	n/d
Ikata 1,2	0.011	0.0026	0.0004	n/d	n/d	n/d
Mihama 1,2,3	0.17	0.1	0.1	0.096	0.04	0.034
Ooi 1,2	0.06	0.20	0.024	0.021	0.03	6.021
Sendai	-	-	-	n/d	n/d	n/d
Takahama 1,2	0.052	0.02	0.0078	0.0074	0.009	0.0081
Netherlands						
Borssele	4.44	7.03	12.58	7.03	22.2	
Sweden						
Ringhals 2	120	78.7	57.7	69.8	153.2	47.72
Ringhals 3	0.7	22.2	11.4	41.1	106.2	21.09
Ringhals 4	-	-	2.1	36.0	47.2	59.02
USSR						
Armenian	0.039					
Novovoronezh 5	0.16					
United States						
Arkansas 1	126.540	277.500	214.600	2678	160.7	139.2
Arkansas 2	152.810	109.150	218.300	863.9	566.4	237.6
Beaver Valley	3.848	5.328	5.439	15.94	25.22	0.681
Callaway	-	-	-	-	0.197	55.49
Calvert Cliffs 1,2	167.610	99.160	194.620	165.1	71.12	97.26
Crystal River	5.402	4.773	3.959	78.69	442.6	173.3
Davis Besse	7.659	29.304	8.103	19.78	6.99	6.861
Diablo Canyon	-	-	-	-	0.43	119.2
Donald Cook 1,2	50.690	68.820	70.300	44.58	419.7	129.1
Farley 1	2.287	4.847	2.198	248.64	214.8	5.332
Farley 2	-	0.995	1.073	116.18	109.5	6.626
Fort Calhoun 1	18.648	6.105	6.623	26.46	135.2	
H.B. Robinson	13.246	68.080	44.400	39.75	14.88	12.29
Haddam Neck	10.212	26.344	2.564	110.5	40.43	
Indian Point 1,2	46.620	209.790	89.170	132.1	113.5	

Table 29, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
Indian Point 3	107.300	96.940	20.202	23.00	687.9	
Kewaunee	22.829	30.155	56.240	19.59	41.47	23.0
Maine Yankee	10.989	16 132	26.011	9.09	3.84	7.205
McGuire	-	14.578	64.750	72.24	118.3	51.33
Millstone Pt. 2	103.970	154.660	51.430	109.9	201.1	
North Anna	38.850	25.012	48.840	607.0	742.6	421.4
Oconee	56.980	64.750	38.480	325.1	677	763.3
Palisades	0.323	1.225	4.699	3.694	1.26	6.637
Point Beach 1,2	23.273	37.370	109.150	46.86	448.5	70.33
Prairie Island	0.488	0.337	0.083	4.36	5.48	3.098
R.E. Ginna	0.725	1.424	22.829	7.11	7.44	
Rancho Seco	0.140	21.904	7.992	9.63	24.93	0.0002
Salem 1	98.050	103.600	119.140	121.1	143.75	877.6
Salem 2	36.593	55.870	118.770	112.5	138.5	142
San Onofre 1	414.400	204.610	79.550	45.14	109.5	
San Onofre 2,3	-	-	23.384	215.6	482.7	425
Sequoyah	-	102.120	363.340	170.6	246.5	
St. Lucie 1	87.320	91.020	113.590	2160	168.9	
St. Lucie 2	-	-	-	18.86	168.9	529.3
Surry 1,2	142.450	226.070	247.160	842.7	363	
Three Mile Island 1	6.771	3.215	1.957	3.01	1.26	0.3478
Three Mile Island 2	0.001	0.001	0.002	0.003	0.024	0.0053
Trojan	29.119	36.778	31.672	13.43	33.62	
Turkey Point	25.086	11.211	62.160	41.71	33.34	
Virgil C. Summer	-	-	0.005	53.66	167.3	
Wolf Creek	-	-		-	-	29.51
Yankee Rowe	0.647	0.529	0.353	27.71	141.9	
Zion 1,2	17.538	98.420	87.690	177.23	643.8	
Total annual electric energy generated (GW a)	35.50	40.55	44.97	53.09	61.74	57.02
Normalized activity [GBq (GW a)$^{-1}$]	92.10	111.5	82.87	214.7	160.9	102.7
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			132.4 ± 49.5			

Table 29, continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
B W R s						
Finland						
Olkiluoto	11.4	17.062	9.877	8.822	14.782	13.805
Germany, Federal Rep. of						
Brunsbüttel	9.6	2.1	1.5	0.91	0.77	0.81
Gundremmingen	-	-	-	-	6.3	2.7
Isar	5.9	1.9	2.1	0.47	0.69	0.54
Krummel	-	-	-	1.3	1.5	0.36
Philippsburg 1	4.1	0.44	1.5	3.3	4.7	0.78
Würgassen	10	9.3	9.7	6.2	5.4	1.9
Italy						
Caorso	0.44	12.6	15.5			
Japan						
Fukushima I						
1,2,3,4,5,6	1.9	1.3	0.59	0.25	0.15	0.035
Fukushima II-1,2	-	n/d	n/d	n/d	n/d	n/d
Hamaoka 1,2	2.6	0.85	0.34	0.14	0.09	0.051
Onagawa	-	-	-	-	n/d	n/d
Shimane	0.037	0.02	0.024	0.018	0.011	0.0043
Tokai II-1	0.31	0.26	0.34	0.25	0.08	0.12
Tsuruga	0.28	0.15	0.023	0.029	0.018	0.025
Netherlands						
Dodewaard	17.76	24.79	30.34	18.5	10.73	
Sweden						
Barsebäck 1,2	57.4	105	151.3	79.979	81.3	64.757
Forsmark 1,2	0.25	19	248.1	413.02	2293.0	382.93
Oskarshamn 1,2	99	123	171	86.085	82.1	62.174
Ringhals 1	99	153.4	153.4	93.6	49.3	54.891
United States						
Big Rock Point	28.934	14.467	9.620	2.887	4.30	5.423
Browns Ferry	347.060	82.880	1983.200	0.189	205.2	49.23
Brunswick	46.620	81.400	85.840	69.92	72.22	16.02
Cooper	407.000	133.570	201.280	458.3	233.5	
Dresden 1	-	-	-	-	-	74.1
Dresden 2,3	26.492	2.264	0.707	0.459	4.27	0.02277
Fitzpatrick	55.870	92.870	24.050	28.34	3.57	6.747
Grand Gulf	-	-	-	0.164	1.17	7.893
Hatch 1	2.527	13.801	25.900	35.73	44.48	18.33
Hatch 2	1.691	6.031	6.771	14.27	14.44	13.04
Humboldt Bay	5.143	5.735	12.802	6.44	2.136	4.64
Lacrosse	78.810	8.362	215.710	142.2	122.2	71.22
Lasalle	-	-	36.334	314.2	3.146	1.48
Limerick	-	-	-	-	0.024	
Millstone 1	26.788	14.578	42.550	38.93	2.104	
Monticello	-	-	-	-	-	-
Nine Mile Point	-	197.950	0.093	0.413		
Oyster Creek	18.722	9.176	2.997	0.464	0.253	
Peach Bottom	70.300	72.890	345.210	86.77	227.8	
Pilgrim	101.010	71.780	32.264	34.62	175.1	38.75
Quad Cities	4.847	120.990	14.911	5.033	5.716	54.87
Susquehanna	-	-	7.363	103.6	5.532	0.3766
Vermont Yankee	-	0.377	-	-	-	
WNP 2	-	-	-	-	-	0.4991
Total annual electric energy generated (GW a)	19.05	19.64	21.92	21.35	23.45	23.57
Normalized activity [GBq (GW a)$^{-1}$]	78.23	71.31	175.3	95.86	156.5	40.9
Average normalized activity, 1980-1984 [GBq (GW a)-1]			115 ± 47			

Table 29. continued

Country and reactor	Activity (GBq)					
	1980	1981	1982	1983	1984	1985
G C R s						
France						
Bugey 1	118	70	40	41	42	43
Chinon A1,2	38	17	19	11	14	5
St. Laurent A1,2	407	237	200	135	131	57
Italy						
Latina	59.2	86.2	162.8			
Japan						
Tokai 1	0.31	0.22	0.23	0.14	0.12	0.1
United Kingdom						
Berkeley	2190	1321	662	667.1	366.3	290
Bradwell	1443	1735	981	962	817.7	1510
Chapelcross	322	1776	4144	3071	481	2000
Dungeness A	629	714	840	864.2	1750.1	2190
Dungeness B	-	-	-	7.4	51.8	174
Hartlepool	-	-	-	7.4	118.4	218
Heysham	-	-	-	7.4	59.2	60
Hinkley Point A	5106	3367	2660	1306.1	2249.6	3720
Hinkley Point B	141	100	44.4	812	928.7	840
Hunterston A	13500	8436	8695	2812	2664	
Hunterston B	1554	2160	3016	2653	3319	
Oldbury	1406	2290	1994	2620	1713.1	1210
Sizewell	1924	1143	1036	658	889.1	1010
Trawsfynydd	518	281	485	350.9	370	430
Wylfa	74	51.8	111	78.3	96.2	48
Total annual electric energy generated (GW a)	3.868	4.847	5.382	5.885	6.223	5.738
Normalized activity [GBq (GW a)$^{-1}$]	7609	4890	4632	2901	2582	2406
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			4520 ± 1790			
H W R s						
Argentina						
Atucha 1	81	51	37	51	51	51
Embalse	-	-	-	-	25.9	1.91
Canada						
Bruce A	163	81	78	74	72	-
Bruce B	-	-	-	-	7	7
Gentilly	2	2	0.6	4	1	9.7
Pickering A	13	8	18	22	27	32
Pickering B	-	-	-	11	27	9
Point Lepreau				18.71	13.10	1.6
Total annual electric energy generated (GW a)	4.283	4.508	4.386	5.283	5.729	7.370
Normalized activity [GBq (GW a)$^{-1}$]	41.10	19.74	21.89	23.79	22.21	14.86
Average normalized activity, 1980-1984 [GBq (GW a)$^{-1}$]			25.7 ± 8.7			

n/d = Discharge not detected.

Table 30

Radionuclide composition of liquid releases excluding tritium from reactors in the United States, 1982
[T5]

A. PWRs

Reactor	Activity (TBq)										
	I-131	I-132	I-133	I-134	I-135	Na-24	Cr-51	Mn-54	Mn-56	Co-57	Co-58
Arkansas 1	14.356	0.013	0.122	0.005	0.026	0.038	14.430	3.885	-	0.243	90.650
Arkansas 2	49.950	0.019	4.107	0.001	0.256	0.029	24.420	0.929	-	0.035	23.680
Beaver Valley	0.389	-	0.064	-	0.003	-	0.013	0.081	-	0.151	1.754
Calvert Cliffs 1,2	24.790	0.059	6.253	-	-	0.059	13.135	1.602	-	0.061	55.500
Crystal River	0.640	0.081	0.168	-	0.188	0.069	0.280	0.069	-	0.001	0.436
Donald Cook 1,2	9.805	-	4.625	-	1.018	0.281	0.685	0.607	-	0.042	20.424
Farley 1	0.032	0.005	0.009	-	-	0.001	0.047	0.030	-	-	0.235
Farley 2	0.032	0.001	0.009	-	0.002	0.001	0.063	0.038	-	-	0.319
Fort Calhoun	0.266	-	0.077	-	-	-	0.614	0.093	-	0.204	0.588
H.B. Robinson	0.239	-	0.086	-	-	39.220	0.002	0.182	-	-	1.469
Haddam Neck	0.001	-	-	-	-	-	-	0.028	-	0.002	0.065
Indian Point 1,2	1.780	-	-	-	-	2.964	5.365	0.559	-	0.570	2.690
Indian Point 3	0.345	0.037	0.097	0.004	0.018	0.001	1.025	0.296	0.002	0.015	2.046
Kewaunee	-	-	-	-	-	0.050	0.362	0.346	-	0.006	31.894
Maine Yankee	1.151	0.019	1.861	-	0.157	-	-	0.119	-	0.003	19.055
McGuire	0.014	-	0.035	-	0.094	0.167	5.291	0.389	-	0.013	40.330
Millstone Pt. 2	164.280	19.536	95.830	5.217	34.299	4.329	10.138	4.884	-	0.023	14.430
North Anna	10.730	-	2.438	-	0.124	0.007	1.132	0.759	-	0.010	13.098
Oconee	0.522	0.004	0.040	0.031	0.007	0.004	0.342	0.215	0.001	0.010	4.625
Palisades	0.488	-	0.007	-	-	-	-	0.347	-	0.004	1.066
Point Beach 1,2	24.827	8.288	21.793	3.922	15.540	0.525	0.076	0.036	-	0.004	0.091
Prairie Island	-	-	-	-	-	-	-	-	-	-	0.010
R.E. Ginna	0.003	0.007	0.018	0.003	0.012	-	-	1.336	-	-	2.653
Rancho Seco	0.585	-	0.051	-	0.005	0.005	0.248	0.389	-	-	4.662
Salem 1	2.394	0.027	0.204	0.006	0.013	0.071	5.106	7.585	-	0.011	62.900
Salem 2	4.958	0.028	0.222	-	-	0.043	3.959	7.474	-	-	62.530
San Onofre 1	-	-	-	-	-	-	1.709	1.314	-	-	17.982
San Onofre 2,3	0.092	-	0.206	-	-	5.809	6.919	0.234	0.033	-	4.736
Sequoyah	3.737	-	0.304	-	0.010	15.799	24.938	6.401	-	0.411	178.710
St. Lucie	7.030	0.003	0.463	0.043	0.455	0.023	7.955	1.103	0.086	0.041	14.208
Surry 1,2	75.850	0.041	8.399	-	0.670	16.206	4.699	1.151	-	0.020	33.004
Three Mile Island 1	-	-	-	-	-	-	-	-	-	-	-
Three Mile Island 2	-	-	-	-	-	-	-	-	-	-	-
Trojan	1.243	0.031	1.191	-	0.143	0.009	0.947	0.263	-	0.134	1.535
Turkey Point	24.309	0.400	7.030	0.129	1.284	8.621	0.858	0.178	-	0.020	6.327
Virgil C. Summer	-	-	-	-	-	0.003	-	-	-	-	-
Yankee Rowe	0.102	-	0.063	-	-	-	0.005	0.013	-	-	0.004
Zion 1	0.044	-	-	-	-	0.005	0.090	0.014	-	-	7.696
Zion 2	0.011	-	0.008	-	-	-	6.253	0.068	-	-	14.652
Total annual electric energy generated (GW a)	21.042										
Normalized activity [GBq (GW a)$^{-1}$]	28.57	1.33	7.41	0.55	8.47	4.48	6.19	2.54	0.01	0.09	34.25

Table 30, continued

Reactor	Activity (TBq)										
	Fe-59	Co-60	Zn-65	Sr-89	Sr-90	Zr-95	Zr-97	Nb-95	Nb-97	Mo-99	Tc-99m
Arkansas 1	1.439	39.590	0.011	0.058	0.004	2.708	0.199	4.736	0.336	0.023	-
Arkansas 2	1.154	5.994	0.038	0.028	-	3.234	0.018	4.884	0.132	0.185	-
Beaver Valley	-	2.416	-	0.003	0.001	0.018	-	-	0.042	-	0.067
Calvert Cliffs 1,2	-	6.956	0.053	1.188	2.290	3.012	-	5.328	-	0.696	-
Crystal River	0.144	0.340	0.154	0.021	0.007	0.201	0.001	-	-	0.249	0.074
Donald Cook 1,2	0.004	7.437	0.088	0.080	0.076	0.622	0.011	-	-	-	-
Farley 1	0.003	0.385	-	0.027	-	0.008	0.011	0.014	-	-	0.009
Farley 2	0.006	0.088	-	0.015	-	0.003	0.004	0.007	-	-	0.004
Fort Calhoun	0.127	0.633	0.166	-	-	0.124	-	0.071	-	0.061	0.030
H.B. Robinson	-	2.046	-	0.718	0.040	-	-	-	0.003	-	-
Haddam Neck	-	1.199	-	0.134	0.276	0.001	-	-	-	-	-
Indian Point 1,2	1.194	5.846	1.265	0.098	0.022	0.895	-	-	-	7.918	0.940
Indian Point 3	0.100	2.893	0.079	0.008	0.002	0.046	-	0.045	0.319	0.171	0.018
Kewaunee	0.165	13.727	-	0.002	0.004	0.030	-	0.092	-	-	-
Maine Yankee	-	0.581	-	0.047	0.002	-	-	-	-	0.003	-
McGuire	0.426	4.255	0.008	0.006	-	0.088	-	-	0.002	-	0.004
Millstone Pt. 2	0.074	33.411	-	0.507	0.006	1.158	-	1.939	5.032	0.071	0.312
North Anna	0.364	8.029	-	0.065	-	3.267	0.065	-	-	-	0.083
Oconee	0.002	1.214	-	0.259	0.284	0.002	0.006	0.064	0.077	0.157	0.005
Palisades	-	0.662	-	-	-	-	-	-	-	-	-
Point Beach 1,2	0.003	0.186	-	0.130	0.041	-	-	-	-	-	-
Prairie Island	-	-	-	-	-	-	-	-	-	-	-
R.E. Ginna	-	4.662	-	-	-	0.128	-	0.474	-	-	-
Rancho Seco	0.086	0.818	-	-	-	0.080	-	-	-	-	-
Salem 1	0.317	33.633	-	-	-	0.422	-	1.047	0.352	0.038	-
Salem 2	0.208	31.857	-	-	-	0.358	-	0.844	0.400	0.006	-
San Onofre 1	0.459	30.969	-	0.002	0.019	-	-	0.021	-	-	-
San Onofre 2,3	0.279	0.161	0.004	-	-	0.389	0.024	0.207	0.024	0.019	0.072
Sequoyah	3.522	49.950	0.414	3.774	0.110	4.810	-	-	0.033	0.036	0.072
St. Lucie	0.312	51.800	0.008	0.035	0.004	1.365	0.529	1.920	0.319	0.197	0.339
Surry 1,2	0.233	32.634	-	-	-	-	0.396	-	-	-	-
Three Mile Island 1	-	0.121	-	-	0.006	-	-	-	-	-	-
Three Mile Island 2	-	-	-	-	-	-	-	-	-	-	-
Trojan	0.001	4.440	-	2.675	1.787	3.027	-	4.477	-	0.015	0.018
Turkey Point	0.022	6.290	0.019	0.349	0.011	0.032	-	0.186	-	0.219	-
Virgil C. Summer	-	-	-	-	-	-	-	-	-	-	-
Yankee Rowe	0.002	0.012	0.004	0.007	0.001	0.001	-	-	-	0.006	0.001
Zion 1	-	7.733	-	1.406	0.007	-	-	0.074	-	-	-
Zion 2	-	16.539	-	15.170	0.004	0.118	-	1.188	-	-	-
Total annual electric energy generated (GW a)	21.042										
Normalized activity [GBq (GW a)$^{-1}$]	0.50	19.09	0.11	1.26	0.23	1.24	0.04	1.30	0.34	0.47	0.09

Table 30, continued

Reactor	Activity (TBq)										
	Ru-103	Ru-106	Ag-110m	Sb-124	Sb-125	Cs-134	Cs-136	Cs-137	Ba/La-140	Ce-141	Ce-144
Arkansas 1	1.380	1.680	6.031	-	-	9.250	0.009	21.090	1.883	-	2.264
Arkansas 2	2.083	0.596	0.577	-	-	32.190	0.291	59.940	2.364	-	0.573
Beaver Valley	-	-	0.008	-	-	0.156	-	0.259	-	-	-
Calvert Cliffs 1,2	0.299	0.940	8.066	1.854	10.064	16.317	-	29.711	0.906	-	-
Crystal River	0.001	-	0.176	0.073	0.178	0.303	0.009	0.407	0.165	0.071	0.459
Donald Cook 1,2	-	-	0.503	0.714	-	6.734	0.474	13.246	-	-	-
Farley 1	0.010	0.001	0.020	-	-	0.072	0.005	0.180	0.047	-	0.124
Farley 2	0.002	0.002	0.008	-	-	0.073	0.015	0.101	0.021	-	0.078
Fort Calhoun	0.075	-	-	0.088	-	0.851	0.102	1.972	0.261	0.134	-
H.B. Robinson	-	-	-	0.004	-	0.031	-	0.222	-	-	-
Haddam Neck	0.001	0.109	-	0.002	0.183	0.078	-	0.455	-	-	0.024
Indian Point 1,2	-	-	0.265	-	2.812	9.139	1.025	20.498	3.848	0.655	-
Indian Point 3	0.096	0.014	0.206	0.107	4.403	0.290	0.060	1.428	0.240	0.033	0.014
Kewaunee	-	-	5.476	1.066	0.692	0.422	-	1.650	-	-	-
Maine Yankee	-	-	-	0.170	-	0.090	-	1.273	-	-	-
McGuire	-	-	0.147	1.232	-	-	-	0.017	-	-	0.012
Millstone Pt. 2	-	0.814	3.271	0.026	0.110	32.338	1.388	46.990	0.022	-	0.241
North Anna	0.236	-	1.787	0.065	-	2.642	0.004	5.254	0.030	0.028	0.103
Oconee	-	0.336	0.331	-	0.057	11.063	0.011	17.353	0.165	-	0.231
Palisades	-	-	-	-	0.035	0.640	-	1.384	-	-	-
Point Beach 1,2	0.001	0.123	0.002	-	0.026	7.992	0.110	11.544	0.094	0.011	0.004
Prairie Island	-	-	-	-	-	-	-	-	-	-	-
R.E. Ginna	-	-	0.001	-	-	0.433	-	0.792	12.284	-	-
Rancho Seco	-	-	-	-	-	0.356	0.011	0.692	0.005	-	-
Salem 1	0.026	-	0.174	0.295	0.252	1.462	-	2.179	0.277	0.050	-
Salem 2	-	-	0.145	0.374	0.377	1.876	0.021	2.827	0.249	0.026	-
San Onofre 1	-	-	0.024	0.020	-	7.215	-	20.017	-	-	-
San Onofre 2,3	-	-	-	0.002	-	1.088	-	-	0.006	-	1.561
Sequoyah	0.012	0.010	0.367	0.226	-	0.733	0.019	3.482	-	-	0.607
St. Lucie	-	-	0.659	2.157	1.869	6.660	0.108	11.729	0.092	0.002	0.314
Surry 1,2	-	-	-	2.087	2.446	72.150	1.872	107.300	0.210	-	-
Three Mile Island 1	-	-	-	-	0.019	0.305	-	1.532	-	-	-
Three Mile Island 2	-	-	-	-	-	-	-	0.001	-	-	-
Trojan	1.221	0.012	0.100	0.084	0.906	0.925	-	1.624	2.757	0.352	1.502
Turkey Point	-	-	0.262	0.374	0.662	1.132	0.123	2.250	0.032	-	-
Virgil C. Summer	-	-	-	-	-	-	-	-	-	-	-
Yankee Rowe	0.001	-	0.002	0.001	-	0.017	-	0.038	0.004	0.002	0.009
Zion 1	-	-	2.231	0.414	0.381	0.451	0.014	1.658	0.164	-	-
Zion 2	-	-	2.164	1.709	-	0.588	0.001	0.698	-	-	-
Total annual electric energy generated (GW a)	21.042										
Normalized activity [GBq (GW a)$^{-1}$]	0.26	0.20	1.60	0.62	1.21	10.23	0.27	17.86	1.23	0.06	0.37

Table 30, continued

B. B W R s

Reactor	Activity (TBq)										
	I-131	I-132	I-133	I-134	I-135	Na-24	Cr-51	Mn-54	Mn-56	Co-57	Co-58
Big Rock Point	-	-	-	-	-	-	0.012	2.168	-	-	0.071
Browns Ferry	8.325	-	2.457	-	0.559	3.885	3.885	3.497	10.471	-	0.644
Brunswick	4.255	0.011	0.736	2.135	0.056	1.565	20.202	10.582	0.002	-	1.388
Cooper	5.143	-	-	-	-	0.636	9.842	14.578	0.002	-	8.806
Dresden 1	-	-	-	-	-	-	-	-	-	-	-
Dresden 2,3	-	-	-	-	-	-	0.008	0.084	-	-	-
Duane Arnold	-	-	-	-	-	-	-	-	-	-	-
Fitzpatrick	0.033	-	0.038	-	0.005	0.063	0.102	2.501	-	-	1.088
Hatch 1	2.956	0.004	0.282	-	0.008	0.182	0.548	0.313	-	-	0.085
Hatch 2	0.777	0.001	0.074	-	0.002	0.206	0.437	0.045	0.001	-	0.027
Humboldt Bay	-	-	-	-	-	-	-	0.005	-	-	-
Lacrosse	0.400	0.020	0.255	0.004	0.059	-	2.316	28.416	0.004	0.111	15.466
Lasalle	-	-	-	-	-	0.214	0.175	0.659	-	-	0.267
Millstone 1	5.402	-	1.054	-	0.381	0.048	1.158	1.469	-	-	0.189
Monticello	-	-	-	-	-	-	-	-	-	-	-
Nine Mile Point	-	-	-	-	-	-	-	-	-	-	-
Oyster Creek	-	-	-	-	-	-	0.003	0.477	-	-	0.001
Peach Bottom	5.476	0.235	8.695	0.045	2.782	131.720	9.176	0.235	0.116	-	4.884
Pilgrim	0.002	-	-	-	-	-	0.245	1.502	-	-	0.199
Quad Cities	0.429	-	0.892	-	0.414	0.335	0.181	4.292	-	-	0.075
Susquehanna	0.064	-	0.018	-	-	0.307	2.830	0.149	0.028	-	0.892
Vermont Yankee	-	-	-	-	-	-	-	-	-	-	-
Total annual electric energy generated (GW a)	10.29										
Normalized activity [GBq/(GW a)]	3.23	0.03	1.42	0.21	0.42	13.52	4.97	6.90	1.03	0.01	3.31

Reactor	Activity (TBq)										
	Fe-59	Co-60	Zn-65	Sr-89	Sr-90	Zr-95	Zr-97	Nb-95	Nb-97	Mo-99	Tc-99m
Big Rock Point	1.099	1.869	0.064	0.009	0.145	-	-	-	-	-	-
Browns Ferry	6.771	54.760	27.232	0.385	0.228	0.977	-	0.977	-	0.326	0.315
Brunswick	1.029	25.271	0.111	0.433	0.444	0.003	0.007	-	0.662	0.033	0.400
Cooper	0.448	65.490	1.395	7.437	0.264	1.251	-	-	-	1.487	0.492
Dresden 1	-	-	-	-	-	-	-	-	-	-	-
Dresden 2,3	-	0.474	-	0.014	0.003	-	-	-	-	-	-
Duane Arnold	-	-	-	-	-	-	-	-	-	-	-
Fitzpatrick	0.128	13.542	0.363	0.021	0.007	0.002	-	-	-	0.005	0.001
Hatch 1	0.032	0.521	3.533	0.174	0.014	0.041	-	0.083	0.001	0.555	0.681
Hatch 2	0.003	0.356	1.010	0.024	-	0.038	0.001	0.057	0.008	0.025	0.062
Humboldt Bay	-	0.836	-	-	0.014	-	-	-	-	-	-
Lacrosse	7.289	98.420	3.034	0.001	0.312	0.217	-	3.504	-	0.242	0.533
Lasalle	0.006	0.107	0.076	0.002	0.004	-	-	-	-	0.003	0.015
Millstone 1	0.246	5.661	0.013	0.165	0.051	-	-	0.011	-	0.028	0.195
Monticello	-	-	-	-	-	-	-	-	-	-	-
Nine Mile Point	-	0.078	-	-	-	-	-	-	-	-	-
Oyster Creek	-	2.505	-	-	-	-	-	-	-	-	-
Peach Bottom	-	24.013	72.520	0.403	0.022	-	-	8.288	-	0.356	2.257
Pilgrim	0.021	13.283	0.206	0.096	0.026	0.044	-	-	-	0.002	-
Quad Cities	0.001	2.072	0.051	0.053	0.021	0.009	-	2.886	-	0.165	0.216
Susquehanna	0.143	0.055	0.157	0.005	0.003	-	-	-	-	0.304	0.799
Vermont Yankee	-	-	-	-	-	-	-	-	-	-	-
Total annual electric energy generated (GW a)	10.29										
Normalized activity [GBq (GW a)$^{-1}$]	1.67	30.16	10.67	0.9	0.15	0.25	0.00	1.54	0.07	0.34	0.58

Table 30, continued

Reactor	Activity (TBq)								
	Ru-103	Ag-110m	Sb-124	Cs-134	Cs-136	Cs-137	Ba/La-140	Ce-144	Np-239
Big Rock Point	-	-	0.053	0.063	-	2.157	0.007	-	-
Browns Ferry	-	5.920	0.470	5.735	0.511	8.029	0.407	-	-
Brunswick	0.011	0.012	-	5.143	0.040	6.438	2.161	-	0.308
Cooper	-	2.401	0.108	28.157	0.332	32.782	1.661	-	-
Dresden 1	-	-	-	-	-	-	-	-	-
Dresden 2,3	-	-	-	0.024	0.001	0.096	-	-	-
Duane Arnold	-	-	-	-	-	-	-	-	-
Fitzpatrick	-	-	0.020	2.024	-	2.575	0.067	0.001	0.696
Hatch 1	-	0.005	0.003	3.312	2.864	4.884	0.031	0.219	0.015
Hatch 2	-	0.014	-	0.777	0.027	1.236	0.002	0.007	0.001
Humboldt Bay	-	-	-	0.685	-	11.063	-	-	-
Lacrosse	1.543	-	-	4.255	-	37.000	-	4.329	0.607
Lasalle	-	-	-	-	-	-	-	-	-
Millstone 1	0.020	-	-	-	2.246	0.026	23.865	0.366	-
Monticello	-	-	-	-	-	-	-	-	-
Nine Mile Point	-	-	-	-	-	-	-	-	-
Oyster Creek	-	-	-	-	-	0.004	-	0.004	0.001
Peach Bottom	0.003	0.001	-	16.835	-	24.013	1.099	-	0.818
Pilgrim	-	0.030	-	0.607	-	4.736	0.003	0.001	-
Quad Cities	0.003	0.021	0.001	0.124	0.012	1.654	0.284	-	0.027
Susquehanna	-	-	0.001	0.262	-	0.065	-	0.365	0.105
Vermont Yankee	-	-	-	-	-	-	-	-	-
Total annual electric energy generated (GW a)	10.29								
Normalized activity [GBq (GW a)$^{-1}$]	0.15	0.82	0.06	6.61	0.59	13.29	2.88	0.51	0.25

Table 31

Radionuclide composition of liquid releases excluding tritium from GCRs in the United Kingdom, 1982
[H3]

Reactor	Activity (GBq)						
	S-35	Ca-45	Mn-54	Fe-55	Co-60	Sr-89	Sr-90
Berkeley	19.869	13.246	< 0.331	< 3.311	< 3.311	-	112.591
Bradwell	9.805	< 4.902	< 4.902	29.415	< 4.902	-	98.05
Chapelcross	44.4	-	-	-	0.74	-	925.0
Dungeness A	23.902	< 5.975	< 0.598	< 5.975	< 0.598	< 5.975	239.02
Hinkley A	212.824	<13.301	< 1.330	<13.301	<13.301	-	292.633
Hinkley B	839.3	11.544	< 0.222	4.44	2.22	-	< 0.222
Hunterston A	177.6	-	-	-	7.4	-	384.8
Hunterston B	-	51.8	3.7	-	22.2	-	18.5
Oldbury	698.005	< 9.972	< 0.997	39.886	< 9.972	-	19.943
Sizewell	124.32	< 0.518	< 0.518	< 5.18	< 5.18	< 0.518	31.08
Trawsfynydd	315.055	< 2.424	< 2.424	< 2.424	< 2.424	-	29.082
Wylfa	17.76	1.11	1.11	21.09	4.44	2.22	1.11
Total annual electric energy generated (GW a)	4.19						
Normalized activity [GBq (GW a)$^{-1}$]	821.0	27.41	3.60	29.84	18.13	2.08	292.9

Reactor	Activity (GBq)					
	Ru-106	Sb-125	Cs-134	Cs-137	Ce-144	Pm-147
Berkeley	< 3.311	< 3.311	39.738	364.265	< 3.311	< 3.311
Bradwell	< 4.902	< 4.902	98.05	637.325	< 4.902	< 4.902
Chapelcross	7.4	3.7	88.8	1073.0	3.7	-
Dungeness A	< 0.598	11.951	35.853	633.403	< 0.598	< 5.975
Hinkley A	133.015	79.809	106.412	655.072	186.221	212.824
Hinkley B	1.332	0.444	0.888	1.332	1.776	0.888
Hunterston A	7.4	-	1480.0	4562.1	299.7	-
Hunterston B	7.4	-	7.4	7.4	-	-
Oldbury	< 9.972	< 9.972	59.829	1076.922	< 9.972	< 0.997
Sizewell	< 5.18	< 5.18	41.44	756.28	< 5.18	10.36
Trawsfynydd	4.847	29.082	4.847	19.388	4.847	4.847
Wylfa	1.11	< 0.555	1.11	22.2	< 0.555	1.11
Total annual electric energy generated (GW a)	4.19					
Normalized activity [GBq (GW a)$^{-1}$]	42.75	34.66	447.7	2085.0	123.4	58.54

Table 32

Normalized local and regional collective effective dose equivalent commitment from noble gases released from the model PWR site

Radionuclide	Normalized collective effective dose equivalent commitment [10^{-4} man Sv $(GW\ a)^{-1}$]
Ar-41	4.0
Kr-85m	0.8
Kr-85	1.0
Kr-87	2.6
Kr-88	8.8
Xe-131m	0.4
Xe-133m	1.1
Xe-133	165
Xe-135m	0.1
Xe-135	73
Xe-138	0.3
Total	257

Table 33

Normalized local and regional collective effective dose equivalent commitment from noble gases released from the model BWR site

Radionuclide	Normalized collective effective dose equivalent commitment [man Sv $(GW\ a)^{-1}$]
Ar-41	0.02
Kr-85m	0.014
Kr-85	0.0008
Kr-87	0.024
Kr-88	0.32
Xe-131m	0.004
Xe-133	0.042
Xe-135m	0.004
Xe-135	0.090
Xe-138	0.052
Total	0.56

Table 34

Normalized local and regional collective effective dose equivalent commitments from tritium released to the hydrosphere

Reactor type	Normalized activity release [TBq $(GW\ a)^{-1}$]	Collective effective dose equivalent commitment [man Sv $(GW\ a)^{-1}$]
PWR	27	0.022
BWR	2.1	0.002
HWR	290	0.23
GCR	97	0.088
LWGR	1.7	0.001
Weighted average 0.033 man Sv $(GW\ a)^{-1}$		

Table 35

Normalized local and regional collective effective dose equivalent commitments from carbon-14 released to the atmosphere

Reactor type	Normalized activity release [GBq (GW a)$^{-1}$]	Collective effective dose equivalent commitment [man Sv (GW a)$^{-1}$]
PWR	345	0.62
BWR	330	0.59
HWR	6336	11.4
GCR	1100	2.0
LWGR	1300	2.3
Weighted average 1.6 man Sv (GW a)$^{-1}$		

Table 36

Normalized local and regional collective effective dose equivalent commitments from releases of iodine

Reactor type	Normalized iodine-131 activity [GBq (GW a)$^{-1}$]	Collective effective dose equivalent commitment [man Sv (GW a)$^{-1}$]		
		Iodine-131	Other iodine isotopes	Total
PWR	1.7	$0.54\ 10^{-3}$	$0.04\ 10^{-3}$	$0.58\ 10^{-3}$
BWR	9.3	$3.61\ 10^{-3}$	$1.2\ 10^{-3}$	$4.8\ 10^{-3}$
GCR	1.4	$0.54\ 10^{-3}$	$0.18\ 10^{-3}$	$0.72\ 10^{-3}$
HWR	0.23	$0.089\ 10^{-3}$	$0.013\ 10^{-3}$	$0.12\ 10^{-3}$
LWGR	80	$30\ 10^{-3}$	$10\ 10^{-3}$	$40\ 10^{-3}$
Average weighted collective dose: $3.3\ 10^{-3}$ man Sv (GW a)$^{-1}$				

Table 37

Normalized local and regional collective effective dose equivalent commitments from particulates released to atmosphere from reactors

Pathway	Collective dose per unit activity (10^{-3} man Sv GBq^{-1})	Collective effective dose equivalent commitment [10^{-3} man Sv (GW a)$^{-1}$]				
		PWR	BWR	HWR	GCR	LWGR
Direct cloud	0.001	0.005	0.043	0.00004	0.0015	0.016
Inhalation	0.12	0.48	5.2	0.005	0.15	2.0
Ingestion	2.0	9	87	0.08	2.4	33.6
Ground-deposits	3.3	14.8	143	0.13	4.2	56
Resuspension	0.004	0.018	0.17	0.0002	0.005	0.08
Total	5.4	24	230	0.22	6.8	90
Average weighted collective dose: 75 10^{-3} man Sv (GW a)$^{-1}$						

Table 38

Normalized collective effective dose equivalent commitments for radionuclides in liquid effluents from reactors discharged to the model river

Reactor type	Radionuclide	Normalized released activity [GBq (GW a)$^{-1}$]	Collective effective dose equivalent commitment [10^{-4} man Sv (GW a)$^{-1}$]		
			Pathway		
			Drinking water	Fish	External
PWR	I-131	30	9.3	0.67	-
	Co-58	33	0.17	0.03	0.0003
	Co-60	20	0.93	0.07	0.007
	Sr-90	0.22	0.23	0.01	-
	Cs-134	10	1.33	0.83	0.002
	Cs-137	18	1.67	1.03	0.01
Total:	16.3		13.63	2.64	0.02
BWR	I-131	3	1.0	0.07	-
	Co-58	3	0.02	0.003	-
	Co-60	30	1.4	0.1	0.01
	Sr-90	0.2	0.2	0.01	-
	Cs-134	8	1.0	0.6	0.002
	Cs-137	15	1.2	0.8	0.01
Total:	6.6		5	1.6	0.02

Table 39

Normalized collective effective dose equivalent commitments for radionuclides in liquid effluents from reactors discharged to coastal waters

Reactor type	Radionuclide	Normalized released activity	Collective effective dose equivalent commitment [10^{-4} man Sv (GW a)$^{-1}$]		
			Pathway		
		[GBq (GW a)$^{-1}$]	Fish	Crustacea	Molluscs
PWR	Co-58	33	0.2	0.1	1.3
	Co-60	20	3.3	1.2	8.7
	Ag-110m	0.2	0.3	0.07	5.3
	Cs-134	10	4.7	0.1	0.83
	Cs-137	18	8.3	0.17	1.17
Total:		35.7	16.8	1.64	17.30
BWR	Zn-65	10	15	1	320
	Co-58	3	0.02	0.01	0.12
	Co-60	30	5	1.8	13
	Ag-110m	0.2	0.3	0.1	5
	Cs-134	8	4	0.08	1
	Cs-137	15	8	0.15	1
Total:		375	32	3	340
GCR	Co-60	20	3	1	10
	Sr-90	360	10	3	20
	Ru-106	50	5	1	40
	Sb-125	40	7	0.2	0.4
	Cs-134	500	150	4	30
	Cs-137	2500	1300	27	180
	Ce-144	150	1.5	7.5	75
Total:		1875	1476	44	355

Table 40

Occupational exposures at LWRs
[A5, B5, B13, B35, E8, E9, I3, I4, I5, I6, I7, K1, M3, N2, N3, P2, P3, P7, S1, S2, S3, S5, S14, S16, T12, V1]

Country, reactor type and year	Number of units	Annual collective effective dose equivalent (man Sv)	Number of workers monitored	Energy generated in the year (GW a)	Annual average effective dose equivalent (mSv)	Normalized collective effective dose equivalent [man Sv $(GW\ a)^{-1}$]
Finland (2 PWRs, 2 BWRs)						
1980	4	2.1		0.48		
1981	4	1.4		1.57		0.9
1982	4	3.3	1900	1.80	1.7	1.8
1983	4	2.3	2600	1.90	0.9	1.2
1984	4	3.2	1800		1.8	
1985	4	2.2	1500		1.4	
France (PWR)						
1980		12.2		3.7		3.3
1981		20.9		8.9		2.3
1982		27.3		10.0		2.7
1983		43.1		12.9		3.3
1984		46.4		17.7		2.6
1985		57.3		21.3		2.7
Japan (ratio PWRs : BWRs ~ 1 to 1)						
1980	24	134	72000	9.1	1.9	15
1981	24	136	88000	9.3	1.6	14
1982	27	116	82000	11.6	1.4	10
1983	27	112	87000	11.9	1.3	9.4
1984	30	117	102000	13.6	1.2	8.6
1985	31	113	118000	14.5	1.0	7.8
Netherlands (1 PWR; 1 BWR)						
1980	2	2.7	790	0.45	3.5	5.9
1981	2	6.4	1350	0.39	4.7	16
1982	2	8.9	1560	0.42	5.7	21
1983	2	7.8	1400	0.39	5.6	20
1984	1 (PWR)	5.2	1040		5.0	
Sweden (3 PWRs; 9 BWRs in 1985)						
1981	9	13	4200	4.3	3.2	3.0
1982	10	9.6	3800	4.3	2.5	2.2
1983	10	14.7	4800	4.6	3.1	3.2
1984	10	11.6	4600	5.8	2.5	2.0
1985	12	11.0	5300	6.5	2.1	1.7
Switzerland (2 PWRs; 2 BWRs)						
1980	4	8.9	1900		4.6	
1981	4	9.1	2050	1.6	4.4	5.5
USSR (PWR)						
1980s					5.6	11
United States (Ratio PWRs : BWRs ~ 2 to 1)						
1980	68	538	80300	29	6.7	18
1981	70	541	82200	31	6.6	17
1982	74	522	84400	33	6.2	16
1983	75	565	85600	33	6.6	17
1984	78	552	98100	37	5.6	15

Table 41

Collective occupational exposures at PWRs and BWRs in the United States and Japan
[B35, T12]

Country and reactor type	Year	Annual collective effective dose equivalent (man Sv)	Number of reactors	Energy generated in the year (GW a)	Annual collective effective dose equivalent per reactor (man Sv)	Normalized collective effective dose equivalent [man Sv (GW a)$^{-1}$]
United States						
(PWR)	1980	243	42	18	5.8	13
	1981	287	44	21	6.5	14
	1982	278	48	22	5.8	13
	1983	290	49	23	5.9	12.5
	1984	281	51	26	5.5	10.5
(BWR)	1980	295	26	11	11.4	27
	1981	255	26	11	9.8	23
	1982	244	26	11	9.4	23
	1983	275	26	10	10.6	28
	1984	271	27	10	10.0	27
Japan						
(PWR)	1980	25.1	13	4.0	1.9	6.3
	1981	28.4	13	4.3	2.2	6.6
	1982	29.2	13	5.4	2.2	5.4
	1983	32.4	13	5.8	2.5	5.6
	1984	34.9	15	5.9	2.3	5.9
	1985	36.4	15	6.7	2.4	5.5
(BWR)	1980	109	11	5.1	9.9	21.5
	1981	108	11	5.0	9.8	21.7
	1982	87	14	6.2	6.2	14.0
	1983	80	14	6.1	5.7	13.0
	1984	83	15	7.7	5.5	10.8
	1985	76	16	7.8	4.8	9.8

Table 42

Occupational exposures at HWRs and GCRs
[A4, B15, B18, H1, H2, H3, H4, I3, I4, I5, I6,]
I7, K1, N3, P5, P8, P14, T12, W2]

Country, reactor type and year	Number of units	Annual collective effective dose equivalent (man Sv)	Number of workers monitored	Energy generated in the year (GW a)	Annual average effective dose equivalent (mSv)	Normalized collective effective dose equivalent [man Sv (GW a)$^{-1}$]
Argentina (HWR)						
1983	2	5.0	980	0.34	5	13
1984	2	3.6	1000	0.44	4	6
1985	2	6.4	860	0.72	8	9
1986	2				13	18
Canada (HWR)						
1980	12	19.1	6780	4.34	2.8	4.4
1981	12	15.7	6640	4.65	2.4	3.4
1982	13	14.9	8100	4.55	1.8	3.3
1983	13	21.7	7190	5.54	3.0	3.9
1984	13	16.5	6290		2.6	
1985	13	11.2	6330		1.8	
Japan (GCR)						
1980	1	1.1		0.12	0.3	9.2
1981	1	0.8		0.12	0.2	7.0
1982	1	0.8		0.10	0.1	8.0
1983	1	0.8		0.11	0.2	7.5
1984	1	1.0		0.11	0.2	9.9
1985	1	1.4		0.09	0.3	15.5
USSR (LWGR)						
1980	2	2.3	-	0.41	-	1.9
United Kingdom (GCR)						
1980	22	23.0	11100	3.33	2.1	7.1
1981	22	22.6	17400	3.48	1.3	6.5
1982	22	19.9	17700	4.28	1.1	4.6
1983	25	18.7	19800	4.57	0.9	4.1
1984	25	19.1	20300	5.00	0.9	3.8
United States (GCR)						
1980	1	0.03	58	0.08	0.05	0.4
1981	1	0.01	31	0.09	0.03	0.1
1982	1	0.04	22	0.07	0.02	0.1
1983	1	0.01	48	0.09	0.02	0.1

Table 43

Normalized occupational exposures at reactors
for the quinquennium 1980-1984

Reactor type	Collective effective dose equivalent per unit energy generated [man Sv (GW a)$^{-1}$]
LWR	13
HWR	4
GCR	5
HTGR	0.1
LWGR	2

Table 44

Estimated typical volumes and activities of conditioned solid wastes from LWRs in the 1970s [E5]

Reactor type	[M7]	[M8]	[B30]	[E5]
	Annual volume (m^3)			
BWR	1000	1500	1000	1000-2000
PWR	600	1100	400	200- 500
	Annual activity (TBq)			
BWR	a/	a/	150	110
PWR	a/	a/	70	20-55

a/ Not estimated.

Table 45

Estimated volumes and activity concentrations of conditioned solid wastes from LWRs

Reactor type	ILW	LLW
Volume per unit energy generated $[m^3 (GW\ a)^{-1}]$		
PWR	50	200
BWR	100	500
Activity concentration ($GBq\ m^{-3}$)		
PWR	100	1
BWR	50	1

Table 46

Estimated typical radionuclide composition of conditioned solid wastes from LWRs after about ten years of interim storage

Radionuclide	Activity percentage	
	ILW	LLW
H-3	0.5	
C-14	0.1	
Ni-59	0.01	
Ni-63	1	
Co-60	20	20
Sr-90 a/	5	5
Tc-99	0.001	
I-129	0.0001	
Cs-134	2	
Cs-136	0.000001	
Cs-137 a/	30	35
U-234	0.00001	
U-235	0.00001	
U-238	0.0001	
Pu-238	0.01	
Pu-239	0.001	
Pu-240	0.01	
Pu-241	0.1	
Am-241 b/	0.001	
Cm-244	0.01	

a/ These radionuclides have daughters that will be in equilibrium. Only the percentage of the parent is reported.
b/ The radionuclide will build up as plutonium-241 decays.

Table 47

Estimated typical volumes and activity concentrations of conditioned solid wastes from HWRs and GCRs
[B31, F3]

Reactor type	ILW	LLW
Volume per unit energy generated $[m^3 (GW\ a)^{-1}]$		
HWR	50	250
GCR	20	1000
Activity concentration $(GBq\ m^{-3})$		
HWR a/	100	1
GCR	1000	10

a/ Excluding tritium.

Table 48

Some major closed and operating shallow burial sites
[C8, C9, H15, N9]

Country and site	Started operation	Status	Approximate total capacity $(10^3\ m^3)$
United States			
Beatty, Nevada	1962	open	600
Maxey Flats, Kentucky	1962	closed	135
West Valley, New York	1963	closed	65
Richland, Washington	1965	open	90
Sheffield, Illinois	1967	closed	85
Barnwell, South Carolina	1971	open	2400
United Kingdom			
Drigg, Cumbria	1971	open	1000
France			
Centre de la Manche	1969	open	400

Table 49

Summary of recorded disposals of packaged solid waste into the north-east Atlantic, 1949-1982
[N6]

Gross weight	(t)	142 000
Alpha activity	(TBq)	680
Beta/gamma activity	(TBq)	38 000
Tritium a/	(TBq)	15 000

a/ Recorded separately for 1975-1982; included in beta/gamma activity for earlier years.

Table 50

Assumptions for assessment of individual and collective doses for releases from land repositories
[L4]

Foodstuff or pathway	Maximum annual exposure (h)	Maximum annual consumption (kg)	Food yield: Terrestrial (kg km^{-2})	Food yield: Marine (kg)
Drinking water		0.6 a/		
Freshwater fish		20		
Beef		60	$1.6\ 10^{4}$	
Cow liver		20	$6.4\ 10^{2}$	
Milk		300	$6.3\ 10^{5}$	
Mutton		30	$1.3\ 10^{3}$	
Sheep liver		20	$6.9\ 10^{1}$	
Green vegetables		80	$1.0\ 10^{6}$	
Grain		130	$4.0\ 10^{5}$	
Root vegetables		120	$2.5\ 10^{6}$	
Marine fish		220		$1.0\ 10^{4}$
Crustacea		36		$2.1\ 10^{4}$
Molluscs		36		$1.0\ 10^{4}$
Seaweed		36		$1.0\ 10^{2}$
Beach occupancy b/	1000			
Fishing gear exposure	880			
Farm ploughing	300			
Other inhalation	8760			

a/ m^3.
b/ Inhalation rate: 1 m^3 h^{-1}.

Table 51

Collective dose equivalent rate per unit activity at closure in an engineered facility as a function of time from 100 to 2,000 years

Time after closure (years)	Collective dose equivalent rate per unit activity [man Sv (TBq a)$^{-1}$]: ^{14}C	^{129}I
100	0 a/	0
200	$1.0\ 10^{-7}$	$1.0\ 10^{-6}$
300	$3.1\ 10^{-3}$	$8.5\ 10^{-3}$
400	$1.0\ 10^{-1}$	$1.7\ 10^{-1}$
500	$3.2\ 10^{-1}$	$2.7\ 10^{-1}$
600	$3.3\ 10^{-1}$	$9.2\ 10^{-2}$
700	$3.2\ 10^{-1}$	$2.0\ 10^{-1}$
800	$4.5\ 10^{-1}$	$3.1\ 10^{-1}$
900	$4.6\ 10^{-1}$	$1.5\ 10^{-1}$
1000	$3.2\ 10^{-1}$	$1.9\ 10^{-1}$
1200	$1.5\ 10^{-1}$	$4.1\ 10^{-2}$
1400	$8.5\ 10^{-2}$	$2.7\ 10^{-2}$
1600	$3.5\ 10^{-2}$	$4.4\ 10^{-3}$
1800	$1.2\ 10^{-2}$	$8.2\ 10^{-4}$
2000	$3.0\ 10^{-3}$	$2.0\ 10^{-4}$

a/ $< 10^{-10}$.

Table 52

Collective dose equivalent rate per unit activity at closure in an engineered facility as a function of time from 10,000 to 250,000 years

Time after closure	Collective dose equivalent rate per unit activity [man Sv (TBq a)$^{-1}$]	
(years)	^{241}Pu a/	^{241}Am a/
10,000	$3.1\ 10^{-16}$	$9.5\ 10^{-15}$
20,000	$1.8\ 10^{-9}$	$5.4\ 10^{-8}$
30,000	$2.9\ 10^{-7}$	$8.8\ 10^{-6}$
40,000	$1.0\ 10^{-6}$	$3.1\ 10^{-5}$
50,000	$2.0\ 10^{-6}$	$6.1\ 10^{-5}$
60,000	$1.4\ 10^{-6}$	$4.3\ 10^{-5}$
70,000	$4.2\ 10^{-7}$	$1.3\ 10^{-5}$
80,000	$6.1\ 10^{-6}$	$1.8\ 10^{-5}$
90,000	$1.6\ 10^{-6}$	$4.9\ 10^{-5}$
100,000	$2.1\ 10^{-6}$	$6.3\ 10^{-5}$
120,000	$1.3\ 10^{-6}$	$3.8\ 10^{-6}$
140,000	$1.1\ 10^{-7}$	$3.5\ 10^{-6}$
160,000	$1.3\ 10^{-7}$	$3.8\ 10^{-6}$
180,000	$2.5\ 10^{-7}$	$7.7\ 10^{-6}$
200,000	$1.3\ 10^{-7}$	$3.8\ 10^{-6}$
250,000	$5.1\ 10^{-10}$	$1.5\ 10^{-8}$

a/ Most of the dose is delivered by ^{237}Np daughter.

Table 53

Collective dose equivalent rate per unit activity at closure in an engineered facility as a function of time from 100,000 to 2,000,000 years

Time after closure	Collective dose equivalent rate per unit activity [man Sv (TBq a)$^{-1}$]	
(years)	^{235}U a/	^{239}Pu a/
100,000	$2.5\ 10^{-12}$	$8.4\ 10^{-17}$
200,000	$4.3\ 10^{-7}$	$1.4\ 10^{-11}$
300,000	$6.9\ 10^{-5}$	$2.3\ 10^{-9}$
400,000	$2.8\ 10^{-4}$	$9.5\ 10^{-9}$
500,000	$6.3\ 10^{-4}$	$2.1\ 10^{-8}$
600,000	$5.5\ 10^{-4}$	$1.9\ 10^{-8}$
700,000	$2.2\ 10^{-4}$	$7.6\ 10^{-9}$
800,000	$1.8\ 10^{-4}$	$6.3\ 10^{-9}$
900,000	$4.9\ 10^{-4}$	$1.6\ 10^{-8}$
1,000,000	$7.1\ 10^{-4}$	$2.4\ 10^{-8}$
1,200,000	$4.9\ 10^{-4}$	$1.6\ 10^{-8}$
1,400,000	$7.1\ 10^{-5}$	$2.4\ 10^{-9}$
1,600,000	$4.0\ 10^{-5}$	$1.4\ 10^{-9}$
1,800,000	$8.6\ 10^{-5}$	$2.9\ 10^{-9}$
2,000,000	$5.4\ 10^{-5}$	$1.8\ 10^{-9}$

a/ Most of the dose is from the daughters ^{231}Pa and ^{227}Ac.

Table 54

Collective effective dose equivalent rate and commitment for disposal of 1 TBq of each radionuclide by shallow burial (LLW)

Radionuclide	Collective effective dose equivalent commitment (man Sv)	Maximum collective effective dose equivalent rate (man Sv a^{-1})	Time at which the specified percentage of the maximum collective effective dose equivalent rate is reached				
			1%	10%	100%	10%	1%
^{3}H	10^{-3}	$3\ 10^{-5}$	15	20	30	50	70
^{3}H a/	10^{-6}	10^{-9}	15	20	30	50	90
^{14}C	1	$4\ 10^{-3}$	15	20	40	600	1000
^{14}C a/	100	$5\ 10^{-2}$	25	45	150	750	20000
^{60}Co	- b/	- b/					
^{63}Ni	10^{-7}	$2\ 10^{-10}$	900	1000	1500	2000	2500
^{90}Sr	- b/	- b/					
^{99}Tc	0.7	$3\ 10^{-3}$	15	20	40	600	1000
^{106}Ru	- b/	- b/					
^{129}I	100	$6\ 10^{-1}$	15	20	40	600	1000
^{129}I a/	700	$6\ 10^{-3}$	25	45	150	750	10^{5}
^{137}Cs	- b/	- b/					
^{237}Np	2000	$3\ 10^{-2}$	$3\ 10^{3}$	$4\ 10^{3}$	$7\ 10^{3}$	$2\ 10^{5}$	$4\ 10^{5}$
^{238}U	400	$2\ 10^{-3}$	$3\ 10^{4}$	$4\ 10^{4}$	$6\ 10^{4}$	$3\ 10^{5}$	$5\ 10^{5}$
^{239}Pu	5	$7\ 10^{-5}$	$9\ 10^{4}$	$1.1\ 10^{5}$	$1.5\ 10^{5}$	$2\ 10^{5}$	$3\ 10^{5}$
^{241}Pu	0.01	$2\ 10^{-7}$	$3\ 10^{3}$	$4\ 10^{3}$	$7\ 10^{3}$	$2\ 10^{5}$	$4\ 10^{5}$
^{241}Am	0.4	$6\ 10^{-6}$	$3\ 10^{3}$	$4\ 10^{3}$	$7\ 10^{3}$	$2\ 10^{5}$	$4\ 10^{5}$

a/ Global circulation.
b/ Collective effective dose equivalent rate less than 10^{-12} man Sv a^{-1}, or collective dose commitment less than 10^{-10} man Sv.

Table 55

Collective effective dose equivalent rate and commitment for disposal of 1 TBq of each radionuclide by engineered trench (ILW)

Radionuclide	Collective effective dose equivalent commitment (man Sv)	Maximum collective effective dose equivalent rate (man Sv a^{-1})	Time at which the specified percentage of the maximum collective effective dose equivalent rate is reached				
			1%	10%	100%	10%	1%
^{3}H	- b/	- b/					
^{3}H a/	- b/	- b/					
^{14}C	1	10^{-3}	750	760	800	1000	2000
^{14}C a/	100	$5\ 10^{-2}$	800	900	2000	2000	20000
^{60}Co	- b/	- b/					
^{63}Ni	- b/	- b/					
^{90}Sr	- b/	- b/					
^{99}Tc	0.7	$8\ 10^{-4}$	750	760	800	1000	2000
^{106}Ru	- b/	- b/					
^{129}I	100	$2\ 10^{-1}$	750	760	800	1000	2000
^{129}I a/	700	$3\ 10^{-3}$	800	900	1000	2000	10^{5}
^{137}Cs	- b/	- b/					
^{237}Np	2000	$2\ 10^{-2}$	$5\ 10^{4}$	$6\ 10^{4}$	10^{5}	$1.5\ 10^{5}$	$2\ 10^{5}$
^{238}U	600	$9\ 10^{-4}$	$3\ 10^{5}$	$4\ 10^{5}$	10^{6}	$1.2\ 10^{6}$	$1.6\ 10^{6}$
^{239}Pu	0.01	10^{-8}	$2\ 10^{5}$	$3\ 10^{5}$	10^{6}	$1.2\ 10^{6}$	$1.6\ 10^{6}$
^{241}Pu	0.01	10^{-7}	$5\ 10^{4}$	$6\ 10^{4}$	10^{5}	$1.5\ 10^{5}$	$2\ 10^{5}$
^{241}Am	0.4	$4\ 10^{-6}$	$5\ 10^{4}$	$6\ 10^{4}$	10^{5}	$1.2\ 10^{6}$	$1.6\ 10^{6}$

a/ Global circulation.
b/ Collective effective dose equivalent rate less than 10^{-12} man Sv a^{-1}, or collective effective dose equivalent commitment less than 10^{-10} man Sv.

Table 56

Normalized collective effective dose equivalent commitment from disposal of intermediate-level waste in a typical engineered trench

Radionuclide a/	Normalized activity concentration [TBq (GW a)$^{-1}$]	Normalized collective effective dose equivalent commitment [man Sv (GW a)$^{-1}$]
^{14}C	$5\ 10^{-3}$	0.5
^{99}Tc	$5\ 10^{-5}$	$3\ 10^{-5}$
^{129}I	$5\ 10^{-6}$	$4\ 10^{-3}$
^{238}U	$5\ 10^{-6}$	$3\ 10^{-3}$
^{239}Pu	$5\ 10^{-5}$	$5\ 10^{-7}$
^{241}Pu	$5\ 10^{-3}$	$5\ 10^{-5}$
^{241}Am	$5\ 10^{-5}$	$2\ 10^{-5}$

a/ Only those radionuclides are shown for which the collective effective dose equivalent commitment per unit activity exceeds 10^{-10} man Sv (TBq)$^{-1}$.

Table 57

Attitudes towards reprocessing in countries with nuclear power stations above 30 MW

Options for disposal of spent fuel

Geologic burial of unreprocessed spent fuel being considered by:	Reprocessing and burial of solidified high-level waste		
	Countries reprocessing	Countries contracted for reprocessing services	Uncommitted
Canada	France	Belgium	Argentina a/
Finland	Germany, Fed.Rep. c/	Germany, Fed.Rep.	Bulgaria b/
Sweden	India	Italy	China (Taiwan Prov.)
Switzerland	Japan	Japan	Czechoslovakia b/
United States	USSR	Netherlands	Finland b/
	United Kingdom	Sweden	German Dem.Rep. b/
		Switzerland	Hungary b/
			Pakistan a/
			Republic of Korea
			Spain
			Yugoslavia

a/ Pilot-scale reprocessing plants reported under construction.
b/ Spent fuel of Soviet origin ultimately to be returned to the USSR.
c/ Plant reported to be under maintenance.

Table 58

National programmes for reprocessing spent fuel
from commercial nuclear power generation

Country	Reprocessing capacity and plans
Belgium	The Eurochemic plant at Mol (annual capacity: 60 tonnes of uranium) was operated between 1966 and 1974; it was then closed on the grounds that it was uneconomic. A decision on recommencement of operations and the possibility of increasing capacity is expected to be made. Belgium has contracted with France for reprocessing of about 54 tonnes of uranium in fuel.
Canada	Research supporting vitrification development.
France	The UP1 plant at Marcoule (annual capacity: 1,200 tonnes of uranium) has been operated since 1958 and the UP2 plant at Cap de la Hague (annual capacity: 900 tonnes of uranium) since 1967 for natural uranium gas-graphite fuel. Following adaptation, the UP2 plant began in 1976 reprocessing LWR fuel at a nominal annual capacity of 100 tonnes of uranium. The capacity of the UP2 plant is being progressively expanded, and a new plant, UP2-800 (annual capacity: 800 tonnes of uranium) is scheduled to begin operation by 1989. A third plant, UP3A (annual capacity: 800 tonnes of uranium) began operation in 1987. A duplicate plant, UP3B, is also under consideration. France has international reprocessing contracts involving a total of about 6,000 tonnes of uranium LWR fuel.
Finland	Spent fuel of Soviet origin is to be returned to that country for reprocessing.
German Democratic Republic	Spent fuel is to be returned to the USSR for reprocessing.
Germany, Federal Republic of	The experimental WAK plant (annual capacity: 35 tonnes of uranium) at Karlsruhe, operational since 1971, was reported closed in May 1980 due to repairs. Construction of a plant at Wackersdorf in Bavaria (annual capacity: 350 tonnes of uranium) has been announced . A total of 1,700 tonnes of uranium of spent LWR fuel is contracted for reprocessing in France.
India	The plant at Trombay for reprocessing natural uranium metal fuel became operational in 1965 (annual capacity: 60 tonnes of uranium). The Tarapur plant for reprocessing HWR and LWR fuel became operational in 1977 (annual capacity: 100 tonnes of uranium). It is believed that a third plant for spent oxide fuel from HWRs will to be operational in the late 1980s at Kalpakkam (annual capacity: 100 tonnes of uranium).
Italy	20 tonnes of LWR fuel has been contracted for reprocessing in the United Kingdom. The Eurex pilot plant at Saluggia has an annual capacity of about 10-20 tonnes of uranium in LWR fuel and is used for research and development in reprocessing.
Japan	A small demonstration reprocessing plant at Tokai Mura has been reprocessing LWR fuel intermittently since 1977 (annual capacity: 210 tonnes of uranium). A commercial plant for reprocessing LWR fuel (annual capacity: 800 tonnes of uranium) is scheduled to begin operation in 1990. Japan has contracted for reprocessing 1,600 tonnes of uranium in LWR fuel in France and 160 tonnes of uranium in the United Kingdom. Japan has also renewed a contract for reprocessing 500 tonnes of uranium in gas-graphite fuel in the United Kingdom.
Netherlands	120 tonnes of uranium has been contracted for reprocessing in France.
Sweden	727 and 140 tonnes of uranium in LWR fuel have been contracted for reprocessing in France and the United Kingdom, respectively. The majority of Swedish spent fuel (6,000 tonnes) is to be stored in Sweden for up to 20 years pending a decision on its disposal.
Switzerland	470 tonnes of uranium has been contracted for reprocessing in France.
USSR	Spent fuel reprocessing is being carried out on a pilot scale; no data are available on the capacities or locations of Soviet reprocessing plants. It is understood, however, that spent fuel of Soviet origin produced in countries of the Council for Mutual Economic Assistance (e.g. Bulgaria, Czechoslovakia, German Democratic Republic, Hungary) is scheduled for return to the USSR. The USSR has also negotiated for the return of spent fuel of Soviet origin from Finland.

Table 58, continued

Country	Reprocessing capacity and plans
United Kingdom	The B204 plant (annual capacity: 1,000 tonnes of uranium) reprocessed natural uranium gas-graphite fuel in the 1950s and early 1960s. The B205 plant (annual capacity: 2,000 tonnes of uranium) has reprocessed this fuel since 1964 and is scheduled to undergo renovation. The B204 plant, after modification, reprocessed LWR fuel between 1968 and 1973. A thermal oxide reprocessing plant (THORP) (annual capacity: 1,200 tonnes of uranium) is under construction and expected to begin reprocessing LWR fuel by 1990. The United Kingdom has international contracts for reprocessing about 3,100 tonnes of uranium in spent fuel.
United States	The plant at West Valley, New York, (annual capacity: 300 tonnes of uranium) operated intermittently from 1966 until its closure in 1972. Due to maintenance problems, a novel plant at Morris, Illinois, (annual capacity: 300 tonnes of uranium) never began operation. Reprocessing of commercial nuclear power fuel was deferred indefinitely in 1977. Construction was halted on a plant at Barnwell, South Carolina, which could require an additional $ 800 million to complete (annual capacity: 1,500 tonnes of uranium). Its operation in the 1990s has been suggested.

Table 59

Radionuclides discharged in airborne effluents from fuel reprocessing plants, 1980-1985
[B1, B2, B3, B7, B8, B16, B29, F1, F4]

Year	Electric energy	Activity (TBq)				
		H-3	C-14	Kr-85	Particulate release	
	(GW a)				Total alpha	Total beta
		Sellafield, United Kingdom				
1980	2.21	252	8.2	31000	0.0005	0.538
1981	3.71	459	18.6	52000	0.001	0.403
1982	3.14	360	9.2	44000	0.0007	0.03
1983	2.96	268	6.8	41800	0.0005	0.022
1984	2.65	349	7.0	37100	0.0004	0.0171
1985	1.70	268	7.0	23800	0.0006	0.0129
Normalized activity, 1980-1985 [TBq (GW a)$^{-1}$]		120	3.5	14000	0.00023	0.063
		Cap de la Hague, France				
1980	2.65	9.1		30525	0.00011	0.00007
1981	3.11	9.9		35816	0.00003	0.00005
1982	4.50	6.3		51800	0.00001	0.00003
1983	4.50	8.1		51800	0.00001	0.00003
1984	2.35	8.5		27010	0.000004	0.00004
1985	6.11	31.8		70300	0.00001	0.0007
Normalized activity, 1980-1985 [TBq (GW a)$^{-1}$]		3.18		11500	0.0000075	0.00004
		Marcoule, France				
1980	1.41	80.4		19800		0.00041
Normalized activity, 1980 [TBq (GW a)$^{-1}$]		56.8		14000		0.00029

Table 59, continued

Year	Electric energy	Activity (TBq)					
		Isotopic composition of particulate activity (total beta)					
	(GW a)	Sr-90	Ru-106	I-129	I-131	Cs-134	Cs-137
		Sellafield, United Kingdom					
1980	2.21	0.018	0.013	0.015	0.001	0.036	0.45
1981	3.71	0.003	0.004	0.01	0.3	0.006	0.08
1982	3.14	0.0009	0.002	0.01	0.006	0.0006	0.01
1983	2.96	0.0007	0.0003	0.009	0.005	0.0004	0.007
1984	2.65	0.0004	0.0003	0.01	0.002	0.0004	0.004
1985	1.70	0.0004	0.00008	0.007	0.002	0.0004	0.002
Normalized activity, 1980-1985 [TBq (GW a)$^{-1}$]		0.0014	0.012	0.0037	0.019	0.0027	0.034
		Cap de la Hague, France					
1980	2.65			0.0178			
1981	3.11			0.0107			
1982	4.50			0.0159			
1983	4.50			0.0207			
1984	2.35			0.0270			
1985	6.11			0.0215			
Normalized activity, 1980-1985 [TBq (GW a)$^{-1}$]				0.0049			

Table 60

Radionuclides discharged in liquid effluents from fuel reprocessing plants, 1980-1985
[B1, B2, B3, B8, B16, B29, C5, F1, F4, G4]

Year	Activity (TBq)					
	Total alpha	Total beta (other than H-3)	H-3	Sr-90	Ru-106	Cs-137
Sellafield, United Kingdom						
1980	39	4300	1280	352	340	2970
1981	30	3800	1966	277	530	2360
1982	28	3500	1750	319	420	2000
1983	14	2489	1831	204	553	1200
1984	14	1190	1586	72	348	434
1985	6	587	1062	52	81	325
Normalized activity, 1980-1985 [TBq (GW a)$^{-1}$]	8.0 (±5.2)	969 (±550)	579 (±35)	77.9 (±49)	139 (±23)	567 (±441)
Cap de la Hague (France)						
1980	0.51	398	539	29.4	387	26.8
1981	0.54	836	708	27.1	331	38.6
1982	0.64	1260	810	86.3	470	50.5
1983	0.48	1180	1160	141.6	337	23.0
1984	0.71	1160	1460	109.6	351	29.8
1985	0.72	1200	2590	46.5	437	29.4
Normalized activity, 1980-1985 [TBq (GW a)$^{-1}$]]	0.16	256.9	285.6	18.97	99.1	8.53
Marcoule, France						
1980	0.089	38	414	4.6		4.6
1981						
1982						
1983						
1984						
Normalized activity, 1980 [TBq (GW a)$^{-1}$]]	0.063	27	294	3.3	-	3.3

Table 61

Isotopic composition of effluents from the Sellafield and Cap de la Hague reprocessing plants, 1980-1985
[B1, B2, B3, B8, B16, B29, F4]

Radionuclide	Activity (TBq)					
	1980	1981	1982	1983	1984	1985
	Sellafield, United Kingdom					
S-35	1.0	0.51	0.8	30.8	0.7	0.8
Mn-54	< 0.063	< 0.095	< 0.1	< 0.2	< 0.2	< 0.2
Fe-55	1.1	1.2	0.9	1.1	0.9	0.7
Co-60	0.78	0.74	1.1	1.7	1.3	2.3
Ni-63	0.41	0.53	0.5	1.1	1.5	0.4
Zn-65	0.033	< 0.034	< 0.04	< 0.04	< 0.03	< 0.06
Sr-89	12	11	< 13	< 8.5	< 3.0	< 1.8
Sr-90	352	280	319	204	72	52
Zr-95	60	130	212	211	162	18
Nb-95	100	200	304	385	312	28
Tc-99	57	5.8	3.6	4.4	4.3	1.9
Ru-103	4.6	11	17	19	8.4	1.6
Ru-106	340	530	419	553	348	81
Ag-110m	0.044	0.14	0.1	< 0.1	< 0.1	< 0.1
Sb-125	21	26	23	18	12	11
I-129	< 0.14	< 0.19	< 0.1	< 0.2	< 0.1	< 0.1
Cs-134	240	170	138	89	35	30
Cs-137	3000	2400	2000	1200	434	325
Ce-144	37	17	22	24	9	< 5
Pm-147	86	32	32	25	77	5.9
Eu-152	4.7	3.5	< 0.6	< 0.2	< 0.2	< 0.1
Eu-154	2.0	1.6	< 1.0	< 0.5	< 0.3	0.1
Eu-155	4.2	2.6	< 1.2	< 0.6	< 0.3	0.2
Uranium (kg)	4861	4499	6011	2602	2037	2447
Np-237	0.67	0.41	0.3	0.3	0.3	0.2
Pu-238	6.9	5.0	4.7	2.9	2.6	0.8
Pu-239/240	20	15	16	8.7	8.3	2.6
Pu-241	728	600	485	331	345	81
Am-241	8.3	8.8	6.4	2.2	2.3	1.6
Cm-242	0.33	0.19	0.28	0.4	0.1	< 0.1
Cm-243/244	0.19	0.11	0.14	0.1	0.1	< 0.1
Electric energy from fuel reprocessed (GW a)	2.21	3.71	3.14	2.96	2.65	1.70
	Cap de la Hague, France					
Co-60	2.7	4.0	3.1	13.5	24.6	15.3
Sr-90	29.4	27.1	86.2	141.7	109.1	47.0
Zr-95	0.07	0.09	0.5	1.1	0.08	0.01
Nb-95	0.05	0.03	0.05	0.1	0.02	-
Tc-99	-	-	-	11.7	12.9	25.9
Ru-106	387	331	468	336	353	437
Sb-125	50.9	43.8	74.5	149	132	109
I-129	-	-	-	0.1	0.1	0.2
Cs-134	3.7	6.0	8.4	4.9	4.8	8.2
Cs-137	26.8	38.6	50.5	23.0	29.8	29.4
Ce-144	2.7	4.1	3.1	2.4	3.2	2.2
Electric energy from fuel reprocessed (GW a)	2.65	3.11	4.50	4.50	2.35	6.11

Table 62

Total beta/gamma activity in fish from the Irish Sea and the North Sea, 1983
[H7]

Sampling area/ landing points	Sample	Number of sampling observations	Mean activity concentration (wet) Bq kg^{-1}		
			Total beta	134-Cs	137-Cs
Sellafield shoreline area	Cod	6	690	28	570
	Flounder	1	750	23	590
Sellafield offshore area	Plaice	4	430	15	340
	Dab	4	380	14	310
	Skate	1	500	16	380
	Whiting	1	570	18	400
	Cod	3	610	21	440
Ravenglass a/	Cod	8	420	14	310
	Plaice	9	370	14	310
Northern North Sea	Plaice	4	110	0.1	6.6
	Cod	6	130	0.2	7.1
	Haddock	4		0.06	5.1
	Saithe	1		n/d	2.8
Mid-North Sea	Plaice	8	110	n/d	4.0
	Cod	10	140	0.2	9.7
	Haddock	3		n/d	6.2
	Herring	1	84	n/d	8.9
	Whiting	1		0.5	21
Southern North Sea	Plaice	4	88	0.04	2.6
	Cod	3	200	0.08	6.2
	Whiting	1		0.4	8.2
	Herring	3	130	0.05	10
Iceland area	Cod	3	91	n/d	0.4
	Haddock	1	96	n/d	0.2
	Plaice	4	85	n/d	0.4

a/ Landing point.
n/d = not detected.

Table 63

Transuranic activity in fish and shellfish from the Irish Sea and North Sea, 1983
[H7]

Sampling area/ landing points	Sample	Number of sampling observations	Mean activity concentration (wet), Bq kg^{-1}					
			^{238}Pu	$^{239,240}Pu$	^{241}Pu	^{241}Am	^{242}Cm	$^{243,244}Cm$
Sellafield shoreline area	Cod	1	0.0047	0.025		0.020	0.00044	0.00011
	Crabs	3	0.71	2.9	80	7.3	0.097	0.051
	Lobsters	3	0.54	2.2	63	14	0.059	0.062
	Winkles	2	6.6	27	710	37	0.45	0.17
Sellafield offshore area	Plaice	1	0.0085	0.034		0.038	n/d	n/d
	Cod	1	0.0057	0.026		0.030	0.00081	n/d
	Skate	1	0.011	0.044		0.045	0.00054	0.00027
	Whelks	1	1.7	7.3		15	n/d	n/d
Ravenglass a/	Cockles	1	14	54		75	1.5	0.47
	Mussels	2	9.9	41	1000	55	0.45	0.25
	Plaice	1	0.011	0.043		0.048	0.0012	0.00014
	Cod	1	0.0040	0.016		0.015	0.00088	0.00026
Northern North Sea	Cod	1	0.00067	0.0038		0.0051	n/d	0.00002
	Nephrops	1	0.0019	0.0092		0.0074	0.00027	0.00008
Mid-North Sea	Nephrops	1	0.00075	0.0033		0.0025	n/d	n/d
	Mussels	1	0.0035	0.019		0.0045	n/d	n/d
Southern North Sea	Mussels	1	0.00077	0.0042		0.0013	n/d	n/d
	Cockles	1	0.0023	0.013		0.0054	n/d	n/d
Iceland area	Cod	1	0.000063	0.00027		0.00032	n/d	n/d

a/ Landing point.
n/d = Not detected.

Table 64

Normalized local and regional collective effective dose equivalent commitments from atmospheric releases from fuel reprocessing at Sellafield and Cap de la Hague

Pathway	Normalized activity 1980-1985 [TBq (GW a)$^{-1}$]		Normalized collective effective dose equivalent commitment [man Sv (GWa)$^{-1}$]	
	Sellafield	La Hague	Sellafield	La Hague
Cloud				
Kr-85	14000	11000	0.1	0.08
Deposited gamma				
Cs-137	0.034	0.0045	0.17	0.02
Inhalation				
H-3	120	3.5	0.05	0.001
Pu-239	0.00006	0.000002	0.01	0.0003
Pu-240	0.00006	0.000002	0.01	0.0003
Am-241	0.00005	0.000002	0.01	0.0003
Pu-238	0.00003	0.000002	0.004	0.0003
I-129	0.004	0.005	0.01	0.01
Cs-137	0.034	0.00004	0.001	-
		Total	0.095	0.01
Ingestion				
H-3	120	3.5	0.27	0.009
Cs-134	0.0027	-	0.02	-
Cs-137	0.034	0.0002	0.2	0.0002
C-14	3.5	0.66	1.4	0.3
Sr-90	0.0014	-	0.02	-
I-129	0.004	0.005	0.17	0.21
		Total	2.08	0.53
		Weighted total	1.3 man Sv (GW a)$^{-1}$	

Table 65

Normalized local and regional collective effective dose equivalent commitments from aquatic discharges from Sellafield and Cap de la Hague

Pathway	Normalized collective effective dose equivalent commitment [man Sv (GW a)$^{-1}$]	
	Sellafield	La Hague
Fish		
Cs-137	38	0.81
Mollusc and crustacea		
Ru-106	4.7	9.9
Sr-90	1	0.23
Pu-239-240	0.2	0.06
Total	44	11

Table 66

Occupational exposures at reprocessing plants in the United Kingdom and Japan
[A5, B12, B23, B28, H8]

Country	Year	Number of workers monitored	Annual collective effective dose equivalent (man Sv)	Annual average effective dose equivalent (mSv)	Collective effective dose equivalent commitment per unit energy generated [man Sv (GW a)$^{-1}$]
Japan	1980	740	0.60	0.8	1.0
	1981	940	0.64	0.7	1.1
	1982	1170	0.71	0.6	1.3
United Kingdom	1980	5200	43	8.2	19.4
	1981	5400	39	7.1	10.4
	1982	5600	38	6.7	12.1
	1983	5300	37	7.0	12.6
	1984	5600	36	6.7	13.4
	1985	5600	32	5.6	18.9

Table 67

Occupational exposures at Cap de la Hague and Marcoule, France, 1973-1985
[C6, H13, Z1]

Year	Number of workers monitored	Annual collective effective dose equivalent (man Sv)	Annual average effective dose equivalent (mSv)	Normalized collective effective dose equivalent [man Sv (GW a)-1]
		Cap de la Hague		
1973	1150	4.9	4.2	10.8
1974	1250	5.3	4.2	4.0
1975	1400	6.9	5.0	5.5
1976	1450	6.8	4.6	8.6
1977	1600	6.7	4.2	4.8
1978	1800	6.3	3.6	3.2
1979	1900	5.6	3.0	2.5
1980	2150	6.3	2.9	2.2
1981	2550	7.1	2.8	2.2
1982	2800	6.0	2.1	1.6
1983	3150	5.9	1.8	1.2
1984	3300	7.1	2.2	1.3
1985	3700	7.9	2.1	0.9
		Marcoule		
1973	1800	3.4	1.8	a/
1974	1950	3.3	1.7	a/
1975	2000	4.9	2.5	a/
1976	2150	5.0	2.3	a/
1977	2300	5.4	2.3	a/
1978	2550	6.5	2.6	a/
1979	2750	7.5	2.7	a/
1980	2850	9.6	3.3	6.8
1981	3050	8.3	2.7	a/
1982	3100	6.7	2.2	a/
1983	3300	5.8	1.8	a/
1984	3500	6.1	1.7	a/
1985	3550	7.0	2.0	a/

a/ No estimate available.

Table 68

Solid intermediate-level waste production at operating reprocessing plants
[T11, B32]

Plant	ILW production $[m^{-3}\ (GW\ a)^{-1}]$	Activity per unit energy generated $[TBq\ (GW\ a)^{-1}]$	
		Alpha	Beta/gamma
Sellafield Magnox	300	130	13 000
Sellafield AGR	50	30	25 000
Marcoule	1000	10	10 000

Table 69

Fraction of the fuel throughput of a reprocessing plant estimated to arise as low- or intermediate-level waste
[D4]

Waste category	Volume generated $[m^3\ (GWa)^{-1}]$	Fraction of fuel throughput for radionuclides				
		Sr/Cs	Ru/Ce	Pu	Am	Cm
Fuel residues, hulls and hardware	20	$5\ 10^{-4}$	$5\ 10^{-4}$	$5\ 10^{-4}$	$5\ 10^{-4}$	$5\ 10^{-4}$
Non-combustible waste	15	10^{-6}	10^{-6}	10^{-4}	10^{-6}	10^{-6}
Compressible and combustible wastes	70	10^{-6}	10^{-5}	10^{-3}	10^{-6}	10^{-6}
Concentrated liquids and particulate solidified wastes	6	10^{-5}	10^{-4}	10^{-3}	10^{-5}	10^{-5}

Table 70

Normalized collective effective dose equivalent commitment, truncated to different times for globally dispersed nuclides, weighted for the fraction of fuel reprocessed
[man Sv $(GW\ a)^{-1}$]

Radio-nuclide	Years				
	10	100	1000	10,000	1,000,000
Kr-85	0.07	0.12	0.12	0.12	0.12
H-3	0.003	0.004	0.004	0.004	0.004
C-14	1.7	6.3	12	63	63
I-129	-	0.0008	0.0016	0.0093	1.5

Table 71

Transport needs in the nuclear fuel cycle for generation of 1 GW a electrical energy by a LWR using plutonium recycle [11]

Material	Amount (tonnes)	From	To
Uranium ore	60000	Mine	Mill
Uranium yellow-cake	170	Mill	Refinery/enrichment/ fuel fabrication
Fuel elements	37	Fuel fabrication	Reactors
Spent fuel	37	Reactors	Storage/reprocessing
Recovered fissile materials	25	Reprocessing	Conversion/enrichment/ fuel fabrication
High-level waste	10	Reprocessing	Waste repository
Other solid wastes	1000	All facilities	Disposal sites

Table 72

Normalized exposure of members of the public from radionuclides in effluents from the nuclear fuel cycle, local and regional populations

Operation and main radionuclide	Normalized collective effective dose equivalent commitment [man Sv (GW a)$^{-1}$]	
Mining		
Radon		0.3
Milling		
Uranium, thorium, radium	0.02	
Radon	0.02	
		0.04
Mine and mill tailings piles (releases over five years)		
Radon		0.1
Fuel fabrication		
Uranium		0.003
Reactor operation		
Atmospheric		
Noble gases	0.20	
Activation gases	0.039	
Tritium	0.53	
Carbon-14	1.6	
Iodines	0.003	
Particulates (Cs, Ru, Co)	0.08	2.45
Aquatic		
Tritium	0.03	
Others (Cs, Ru, Co)	0.013	0.043
Reprocessing		
Atmospheric		
Tritium	0.007	
Krypton-85	0.005	
Carbon-14	0.04	
Caesium-137	0.01	
Iodine-129	0.01	
Alpha-emitters	0.001	0.07
Marine		
Caesium-134,137	0.8	
Ruthenium-106	0.4	
Strontium-90	0.03	
Alpha-emitters	0.005	1.2
Transportation		0.1
Total (rounded)		4

Table 73

Normalized exposures of members of the public from solid waste disposal and globally dispersed radionuclides in effluents from the nuclear fuel cycle

Source	Normalized collective effective dose equivalent commitment [man Sv (GW a)$^{-1}$]
Mine and mill tailings (releases over 10^4 years) and fuel fabrication	150
Reactor operation	
LLW disposal	0.00005
ILW disposal	0.5
Reprocessing solid waste disposal	0.05
Globally dispersed radionuclides	63
Total (rounded)	200

Table 74

Normalized occupational exposures from the nuclear fuel cycle

Operation	Normalized collective effective dose equivalent [man Sv (GW a)$^{-1}$]
Uranium mining and milling	0.7
Fuel fabrication	0.5
Reactor operation	10
Reprocessing	0.25
Transportation	0.2
Total (rounded)	12

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back to first page

ANNEX C

Exposures from medical uses of radiation

CONTENTS

Introduction

1. The Committee has previously reviewed data on exposures from medical uses of radiation in its Reports of 1958 [U1], 1962 [U2], 1972 [U3], 1977 [U4] and 1982 [U5]. Medical radiation may be incurred from (a) diagnostic and interventional x-ray examinations; (b) diagnostic nuclear medicine examinations; and (c) radiation therapy from either external or internal sources. In many countries, diagnostic medical examinations contribute the largest proportion of the collective effective dose equivalent from man-made sources received by the population.

2. The aim of this Annex is to assess the magnitude of radiation exposures delivered world-wide in the course of medical practice. Once this has been achieved, (a) sources of radiation exposure may be compared; (b) areas of concern can be identified; (c) possible detriment estimated; and (d) efforts channelled for an optimum global radiation dose reduction (if indicated). Thus far, the Committee has estimated that the

collective effective dose equivalent for the world from diagnostic medical radiation is about 400 man Sv per million population (i.e., about 0.4 per caput).

3. For diagnostic and interventional uses of radiation, there is a possibility of dose reduction, although one must be careful not to decrease at the same time the associated benefits. Medical radiation differs from other radiation sources in several ways. The first is that, with the exception of medical occupational radiation exposure, those receiving the doses delivered in the course of medical procedures are those who are expected to benefit directly from such procedures. The second difference is that the dose to patients during radiography is usually received over a short time and most often involves only a limited portion of the body. A third difference from other sources is that the exposed population is highly selected, insofar as many of the exposed individuals are suffering from some form of illness and insofar as their age distribution is quite different from the age distribution of the population at large.

4. One of the limitations of the previous Reports of the Committee, as well as of this Annex, is that good data on frequency of examinations and absorbed dose from medical examinations and occupational sources are available predominantly from developed countries, which account for less than 25% of the world's population. Fragmentary data on examination rates and numbers of machines and little or no data on absorbed doses are available for another 25% of the population, and no data are available at all for 50% of the world's population.

5. The present availability of radiodiagnosis is very uneven throughout the world: one x-ray machine is shared by fewer than 2,000 people in some countries and by 100,000-600,000 people in other countries. The frequency of procedures is also very uneven (15-20 procedures annually per 1,000 population in some countries and about 1,000 procedures annually per 1,000 population in others) [R1]. At present, there are approximately 5 10^9 people in the world and some authors indicate that more than three quarters of the world's population have no chance of receiving any radiological examination, regardless of what disease they may have. In many developing countries, between 30% and 70% of x-ray machines are out of order [M32, P2]. The lack of good data from areas that account for approximately three quarters of the world's population has led the Committee to adopt an extrapolation procedure for estimating world-wide medical use of radiation.

6. In the UNSCEAR 1958 Report [U1], the Committee was predominantly interested in exposures that might have hereditary effects, so it calculated a genetically significant dose (GSD). It became evident during the 1958 analysis that a major portion of dose was contributed by relatively few types of examinations. By 1977, and even more by 1982, the Committee became interested in estimating the mean doses to other tissues, particularly those tissues regarded as more susceptible to the induction of stochastic effects (e.g., the thyroid, active bone marrow, the lung and the female breast). For calculation of possible subsequent cancer induction, age at exposure was recognized to be important, but little data existed on this parameter.

7. Although it is of interest for the Committee's purposes to compare the risk from medical radiation with risks from other sources of man-made radiation or from natural background radiation, such comparison has always posed a difficult problem. The effects of radiation depend upon the energy of the radiation, instantaneous dose rate, the time over which the total dose is received, and the part of the body exposed. In this respect, diagnostic examinations are markedly different from radiotherapy procedures, in which substantially higher doses are given to a much smaller group of patients, in whom non-stochastic effects are present in the short term. The Committee has always felt that the potential stochastic risks to patients from diagnostic medical radiation and nuclear medicine should not be summed or compounded with the risks from radiotherapy. The reasons for this are that the risk coefficient for a given effect may vary with the magnitude of the absorbed dose and the dose rate. In addition, the radiation risk coefficient for cancer patients is unknown and their lifespan and age distribution are likely to be different from other populations. Radiation therapy is therefore assessed in this Annex only in terms of average absorbed doses in organs. It would be of interest to evaluate the absorbed doses to tissues outside the target volume in patients who have undergone radiation therapy for estimation of possible later stochastic effects. Unfortunately, the Committee has been unable to obtain data on the number and exact treatment regimes that have been utilized.

8. In 1977, the International Commission on Radiological Protection (ICRP) introduced a quantity called the "effective dose equivalent", defined as the sum of all the organ dose equivalents weighted for the relative radiation risk. The effective dose equivalent as defined for purposes of radiation protection [12] should not, in principle, be used to estimate the detriment in population groups with sex or age distributions that differ significantly from those of the working population, and it was not the original intention of the ICRP that the effective dose equivalent concept should be extended to patients. However, in the absence of good age distribution data on exposed patients, the Committee utilized the effective dose equivalent concept in the UNSCEAR 1982 Report [U5] as the best available estimate of medical exposure for the purpose of comparison with other sources of radiation exposure. This Annex examines the age distribution of populations undergoing different radiological examinations and points more specifically to the limitations to the use of collective effective dose equivalent for estimating detriment. A more detailed discussion of this problem is presented in section I.F.

9. In addition to examining the population structure itself, the Committee felt that it was important to examine trends in the utilization of various procedures used for a given diagnostic objective as well as trends in types of equipment. Over the past decade there have been many technological advances that may be

affecting medical exposure: old techniques are being replaced by new ones; additional examinations are being performed; and procedures are being carried out with different types of equipment, leading to increases or decreases in the mean absorbed doses in organs in the course of examinations. This Annex examines trends where sequential data are available. Although it is clear that trends vary markedly from country to country, it appears that, globally, the extent to which medical radiation is utilized is increasing. The World Health Organization published a report [W19] containing recommendations intended to alert the medical and governmental communities to the fact that, particularly in industrialized countries, many clinically unproductive radiological examinations are being performed. In contrast, there is probably substantial under-utilization in developing countries.

10. In an attempt to estimate the global use of medical radiation, the Committee has made use of the good correlations that exist between population per x-ray machine and population per physician (Figure I). A good correlation has been found between the number of x-ray examinations per unit of population and the number of physicians per population [M27]. Four levels of health care have been defined, based upon the number of population per physician in a given country in 1982. In countries with the highest level of health care (level I), more than one physician is available per 1,000 population. In countries of the next category (level II), one physician is available per 1,000-3,000 population. In countries with lower levels of health care, one physician serves 3,000-10,000 people (level III) and more than 10,000 people (level IV). By estimating the average number of medical radiation examinations in countries of the various health care levels and reported doses from representative countries, the doses to the world population can be determined. This approach is used in evaluating the doses from x-ray examinations, from diagnostic use of radiopharmaceuticals, as well as therapeutic uses of radiation.

11. This Annex also reviews doses to particular organs from various types of medical examinations. The individual and collective organ doses from various medical practices are computed to evaluate the contribution that medical practice makes to man's total radiation exposure. Since these data may also be used to determine whether special population groups are being highly exposed, they may be of epidemiological interest. There remain some difficulties, however, in comparison of absorbed doses, because the techniques presented in the radiologic physics literature sometimes measure exposure rather than absorbed doses. The determination of interest is the average absorbed dose in an organ. There is considerable variability from study to study in modelling, computational techniques and assumptions utilized. Because organ doses vary markedly from one procedure to another, it is useful to examine this variation within a given country as well as from country to country, in search of the underlying causes.

12. While absorbed dose data exist for many radiographic and nuclear medicine procedures, this Annex suggests that previous estimates of absorbed dose to the world's population may be somewhat low. The two most important reasons for this suspicion are the widespread use of fluoroscopy in developing countries and the large number of malfunctioning machines producing high absorbed doses (neither factor was widely appreciated in the past) [B16, D1]. For example, in the People's Republic of China, most radiographic examinations are performed with fluoroscopy machines that do not have image intensification systems [S32, Z4], resulting in higher dose equivalents per examination than in some other countries.

13. Finally, this Annex examines expected changes in the magnitude of medical exposure through the year 2000. The Committee recognizes that there is expected to be (a) a significant increase in total population of the world; (b) a marked aging of the population in many, mostly developed countries, with increased proportions of the population over the ages of 60 and 80; (c) an increase in the proportion of the world's population residing in cities; and, finally, (d) a shift in the spectrum of diseases [O4]. All of these factors are expected to play a significant role in the future use, availability and need for medical radiation.

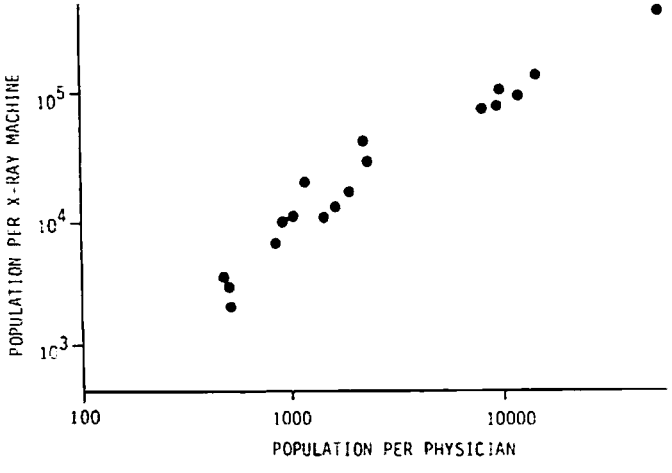

Figure I. Correlation between population per physician and population per x-ray machine in various countries. [M27, U6, U7, U8]

I. DIAGNOSTIC MEDICAL X-RAY EXAMINATIONS

A. FREQUENCY AND TRENDS

14. In the UNSCEAR 1977 Report [U4], data on the frequency of diagnostic x-ray examinations were available for only three countries: Japan, Sweden and the United States of America. The UNSCEAR 1982 Report [U5] reviewed the annual frequency of these examinations in several countries; however, it was difficult to discern world-wide trends since most countries had not conducted sequential surveys.

15. Information is now available from some other countries. The annual frequency of procedures per person varies significantly between countries [C7]. In many developing countries, radiology is used about

30 times less often per caput than in industrialized countries. The consumption of radiographic film per unit population is a poor parameter for assessing the global medical radiation exposure of the population because in many countries there is a preponderance of mass miniature radiography or fluoroscopy and these require higher values of dose. In its attempt to assess data, the Committee has therefore concentrated on numbers and types of machines as well as on the number of procedures.

16. Information on the annual frequency of diagnostic x-ray examinations in 13 countries of level I health care and one country of level II health care is collected in Table 1. The total frequencies in these countries range from 450 to 1,300 examinations per 1,000 population, with an average of 800 examinations annually per 1,000 population for level I countries. The number of diagnostic x-ray examinations is increasing, according to the results of sequential surveys in several countries.

17. From 1976 to 1980, the number of radiological examinations performed in hospitals in Canada increased by approximately 2.7 million. Most types of examinations increased in number. The examinations that decreased in frequency over that time period were those of the abdomen, breast and bronchus and those related to obstetrics and gynaecology [C1]. When the total examinations were considered for 1980-1981, there was an annual rate of approximately 1,000 medical x-ray examinations per 1,000 population (Table 1). Additional data have been reported from the province of Manitoba [M3], where the number of examinations reported per 1,000 population was 860 in 1974 and 840 in 1979.

18. In France in 1957, approximately 6.2 million radiographic examinations were performed. As of 1981, this number had risen to 45 million (835 examinations per 1,000 population). The number of various types of diagnostic radiologic examinations performed in France in 1982 is given in Table 1 [B9]. Le Gales et al. [L8] report that in France in 1980 approximately 9.8 million chest screening examinations were performed. About 60% of these were photofluorographic, 30% fluoroscopic, and the rest radiographic. In addition to screening for tuberculosis, there is also a well-defined radiologic screening programme in France for detection of congenital hip dysplasia. Bouvet et al. [B18] have reported that 3.4 million radiographies of the hip and pelvis were performed in 1982. Of these, 725,000 were carried out on children of less than one year of age. The annual birth rate in France is about 720,000.

19. In Italy in 1983, 744 medical x-ray examinations were carried out per 1,000 population [P1]. Indovina et al. [I9] indicate that mass screening in Italy resulted in 4.3 million chest photofluorographies in 1974 and 3.0 million in 1980.

20. The frequency of diagnostic x-ray examinations in Japan is made greater by the mass chest x-ray examination campaigns and by an emphasis on examinations of the abdomen and the gastro-intestinal tract (Table 1). Kumamoto [K22] reported that in 1980, 26.6 million (242 per 1,000 population) mass chest x-ray examinations were performed. This number is considerably lower than the 33 million photofluorographic examinations performed in 1975 [H4].

21. In the Netherlands in 1980, approximately 8.7 million examinations were performed [B6]. Approximately 40% of examinations were of the chest; half of these were mass miniature radiography. The total annual frequency was 648 per 1,000 population (Table 1).

22. A detailed report on the annual frequency and type of examinations performed in Norway in 1980 and 1983 has been published [S3, S4]. The total annual frequency for 1980 was 641 per 1,000 population (Table 1).

23. According to estimates by Kudritsky et al. [K19, K20], the number of x-ray procedures in the Russian Soviet Federative Socialist Republic (RSFSR) during 1970-1980 increased from 138.5 to 185.8 million and their annual frequency from 1,065 to 1,339 per 1,000 population. During that decade, the mean annual increase in the frequency of x-ray examinations remained constant at 2.3-2.4%. Somewhat lower annual frequency rates have been reported for the USSR by Vorobyev et al. [V7] and Nikitin [N9]. They indicated that between 1963 and 1981, the number of x-ray examinations increased by 21%. The ratio of photofluorographic to radiographic and fluoroscopic examinations increased substantially (Table 2). The annual frequency of various types of chest x-ray examinations was reported per 1,000 population as follows: photofluorography, 526; radiography, 118; and fluoroscopy, 149 [V7]. Similar figures have been reported by both Neamiro et al. [N6] and Nikitin [N9] (Table 1). About one half of all x-ray examinations in the USSR are chest photofluorography performed for prophylactic purposes.

24. An estimation of the annual frequency and type of examinations performed in Spain in 1986 has been made by Vano et al [V4]. The total annual frequency was 490 per 1,000 population (Table 1). The increment between 1985 and 1986 was 2.5%.

25. Wall et al. [W6] have indicated that the frequency of diagnostic examinations in the United Kingdom in 1983 was 488 per 1,000 population (Table 1). This is not significantly different from the value of 440 reported in the UNSCEAR 1977 Report [U5]. The annual increase in frequency for most types of examinations was 2-3%.

26. Growth of diagnostic radiological procedures in the United States appears to have been fairly rapid. There has been a general increase in almost all types of general radiographic examinations since 1964. The rate of hospital-based examinations per 1,000 population was 370 in 1964, 400 in 1970 and 570 in 1980, which is similar to the hospital x-ray examination rate of 650 per 1,000 estimated for Canada. The total frequency of medical x-ray examinations in the United States is 790 per 1,000 population [M28]. The increase

in frequency of examinations in Canada and the United States is probably due to a number of factors, including, among others, a change in the age distribution of the population. The number of medical diagnostic machines per 1,000 population increased from 0.53 in 1969 to 0.61 in 1981.

27. Zhang et al. [Z4] and Zhang [Z5] conducted a survey of radiological services in Shangdong Province, China, for the years 1976-1980, and this information has also been included in Table 1. The authors reported that during this time the annual frequency of examinations in rural areas increased by 77% (from 146 to 259 examinations per 1,000 population), while in urban areas the frequency was significantly higher but had increased much less as a percentage of the total (from 577 examinations per 1,000 population in 1976 to 710 in 1980, corresponding to a 23% increase). Chest fluoroscopy accounted for more than 70% of all examinations performed, chest radiography for only 2%, skeletal radiography for 6%, and special examinations for 4%. The authors also indicated that the majority of chest fluoroscopy was for screening purposes. Similar urban rates have been reported by Sun et al. [S32], who indicated that the total annual frequency of radiographic procedures in Beijing was 761 per 1,000 population, with chest fluoroscopy accounting for 65% of all procedures. Approximately 90% of all x-ray equipment in China in 1980 was fluoroscopic (i.e. about 70,000 fluoroscopic units). Somewhat lower utilization rates (about 120 per 1,000 population) have been reported by Zhang [Z7] for the Zhoukou region of Hunan Province, although the distribution of types of examinations is similar to that reported by other authors.

28. A discussion of diagnostic radiologic procedures in the Islamic Republic of Iran in 1980 [S19] indicates that the estimated overall rate of diagnostic x-ray procedures was 180 per 1,000 population. The authors also suggested that the frequency of x-ray examinations in urban areas was higher than in rural areas and small towns, with the urban population receiving approximately twice the national average.

29. Data regarding the number and frequency of x-ray examinations available from Turkey for 1977 [Y1] indicate that the annual frequency was about 80 examinations per 1,000 population.

30. The number of diagnostic radiological examinations performed in various Central and South American countries is difficult to ascertain, but some trends can be indicated by the growth in the number of machines (Table 3). There has been an increase in the number of radiodiagnostic machines in Argentina and Chile, while in Costa Rica, Ecuador and Mexico, the number of units per 1,000 population has not increased.

31. By applying the extrapolation procedure described in paragraph 10, the number of diagnostic x-ray examinations and machines on a world-wide basis may be estimated. The basic data used to make these estimates for various levels of health care are shown in Table 4. From these values, the average annual examination rate per 1,000 population and the population per x-ray machine for each level of health care are derived from the data in Table 4 and are shown in Table 5. To the extent that populations, numbers of x-ray machines and examinations have increased in direct proportion to one another since the surveys took place, it will be approximately valid to estimate current levels of practice from present world population. The number of annual x-ray examinations world-wide is thus estimated to be approximately 1,400 million, and the number of diagnostic medical x-ray units, approximately 440,000. The number of medical x-ray examinations for the four levels of health care are listed in Table 6. As might be expected, the one quarter of the world's population in countries of health care level I receives three quarters of the examinations. The average number of examinations performed per year and per x-ray machine ranges from 3,000 to 5,500 for all levels of health care.

32. Table 7 indicates that, of the diagnostic x-ray examinations performed in some Latin American countries, chest examinations account for 22-50% and examinations of the extremities for 22-36% of the total. This is fairly consistent with the data in Table 1, which showed that in level I countries 32% of all examinations were of the chest and 19% examinations of the extremities. The Committee has reviewed available data over the past decade on the percentage of the total of diagnostic x-ray examinations accounted for by each type of examination. This is shown in Table 8 for three levels of health care. The main difference appears to be that examinations of the abdomen and digestive tract represent 18% of the total in level I countries but decrease to 13% in level II countries and to 6% in level III countries. At the same time, there is an increase in the percentage of chest examinations from level I countries to level III countries. It is of interest that examinations of the head and neck and urogenital examinations account for a fairly uniform percentage regardless of the level of health care.

33. As might be expected, the urban population receives more x-ray examinations than the rural population (Table 9). Similar findings have also been reported by Cockshott [C7], who terms this the "capital city syndrome" even though the data are for urban areas in general and not just capital cities. In effect, a segment of the population often receives a disproportionately high number of examinations. This disproportion is also evidenced by the data available from China [S32, Z4] and the Islamic Republic of Iran [S19]. Urban populations may receive two to 10 times as many examinations per caput as rural populations.

34. Information on the historical trend in the annual frequency of diagnostic examinations in various countries is summarized in Table 10. With the exception of China and Turkey, the countries are level of health care I. For these level I countries the annual growth rate for examinations from 1955 through 1983 ranged from 0% to 10%, with an average of 3% over the decade 1970-1980.

35. Dental radiography is the most common type of diagnostic x-ray examination. In the UNSCEAR 1977

and 1982 Reports, the Committee reported data on the annual frequency of these examinations in several countries, but no data were available on trends. At present, data indicating trends are available from three countries.

36. The use of dental radiology in the United Kingdom has been reported by Wall and Kendall [W3]. It is apparent from these data that there was a marked increase in the use of dental radiology from 1963 to the end of 1981 (Figure II). The frequency of dental x rays more than doubled between 1970 and 1983. In 1983, there were 9 million dental x-ray examinations (165 per 1,000 population). In 1981 most were intra-oral (6.7 million), but 150,000 were extra-oral and 910,000 were pantomographic. The average number of films per examination in 1981 was 1.8.

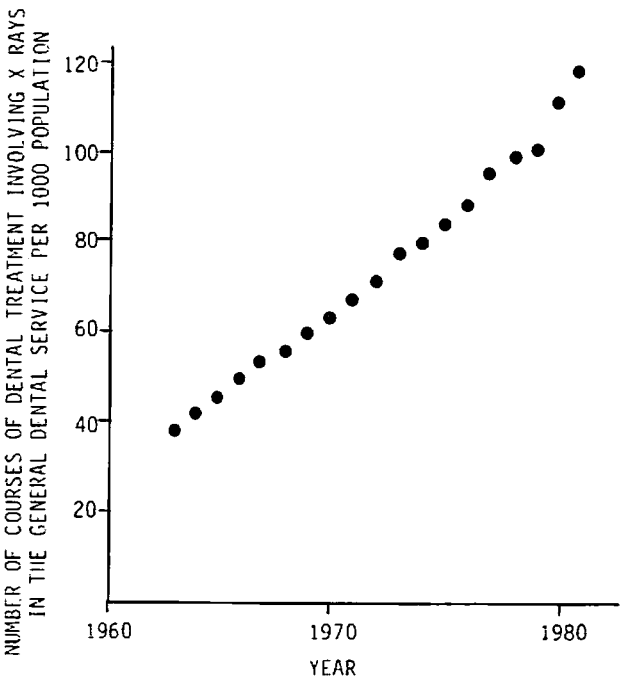

Figure II. Growth in the use of dental radiography in the United Kingdom since 1963.
[W3]

37. In France in 1984, 27.5 million intra-oral films and 1.76 million pantomographic films were taken [B11]. Between 600 and 1,200 examinations annually were performed per machine.

38. In Japan, a survey carried out in 1976 [K9] indicated that the annual number of oral radiographic films per caput in Hiroshima and Nagasaki ranged from 0.8 to 1.0. Exposure frequencies were approximately 1.5 times greater among the non-exposed individuals than among the atomic bomb survivors. According to Maruyama et al. [M11], who estimated the use of dental radiography in Japan in 1980, the annual number of exposures for intra-oral radiography was 90 million (769 exposures per 1,000 population), with an average of 1.7 exposures per examination (435 examinations per 1,000 population). Pantomographic examinations were estimated to be 9.6 million (82 per 1,000 population). This represents a total annual dental examination rate of 517 per 1,000 population. No increase in intra-oral examinations had occurred by 1985; however, there was a small increase in pantomographic examinations, to 11 million examinations.

39. According to the UNSCEAR 1977 Report [U5], the number of single dental exposures per 1,000 population in Sweden in 1974 was 1,500. Preliminary data from Sweden obtained for 1984-1985 [V2] indicate that about 1,800 dental films were obtained per 1,000 population. In the United States, there has also been a substantial increase in the frequency of dental x-ray examinations per 1,000 population [M10], although the increase was not as rapid as in the United Kingdom [W3]. The number of dental x-ray examinations in the United States increased from 67 million in 1970 to 105 million in 1982. Exposures increased from 280 million to 380 million during the same period. Between 1970 and 1982 in the United States the average annual compounded growth rate for examinations was 4.2%. The number of exposures (films) increased at an average annual compounded growth rate of 3.4%. The number of exposures per patient declined somewhat as pantomographic procedures gradually replaced full-mouth series. In 1970, approximately 6% of all dental x-ray examinations were pantomographic, and this number had risen to 18% by 1982. In 1982, the annual dental radiographic examination rate was 456 per 1,000 population. Dental x-ray machines increased from 98,000 in 1966 to 201,000 in 1981 [M10].

40. It appears that there has been relatively rapid growth in the number of dental x-ray examinations in countries of health care level I, ranging between 50 and 100% in the years 1970-1980. While there has been some increase in intra-oral dental x-ray examinations, the largest increase has occurred in pantomographic examinations. There has been growth not only in the number of examinations, but also in the number of dental x-ray machines.

41. By applying the extrapolation procedure described in paragraph 10, the total number of dental examinations performed world-wide for the various levels of health care can be estimated. The basic data required were taken from published information and have been collected in Table 11. Very limited data are available for level III countries and no data are available for level IV countries. The estimated total number of procedures for each level of health care is shown in Table 12. According to this estimate, 340 million dental radiographic procedures are conducted annually.

B. AGE AND SEX DISTRIBUTION OF PATIENTS

42. Knowledge of the age and sex distribution of those receiving examinations is important for evaluating the applicability of the collective effective dose equivalent as a measure of detriment for medical radiation, as well as for assessing the genetically significant dose.

43. Table 13 shows the percentage of the population of various countries, grouped by age and sex, receiving various examinations. Comparison for various examinations reveals some interesting differences between countries.

44. While only 31% of the population of the United States is 45 years or older, these individuals receive 51% of all the examinations; also, the 11% of the population older than 65 receive 25% of all the examinations. In the Islamic Republic of Iran, where only 16% of the population is over the age of 45, almost 50% of all examinations are performed on individuals under the age of 30 [S19]. In China, 44% of chest examinations are performed on persons under the age of 30, whereas in Norway, the United Kingdom and the United States the comparable figure is 20%. In general, the less developed a country, the younger the mean age of the population and the younger the population exposed in diagnostic radiology.

45. In the course of three surveys carried out in the USSR, Kudritsky et al. [K19, K20] analysed the distribution of x-ray examinations and identified some general regularities for various age groups in the population. The lowest frequency, observed in children under 15, was 20-50% of the mean value for the entire population. The frequency of x-ray examinations increased gradually with age, reaching a maximum for persons between 40 and 59 years, for whom it was 1.5 to two times higher than the average frequency. In the group aged 60 and above, the frequency of x-ray examinations was again somewhat lower, reaching levels 30% below the average.

46. In most of the countries of health care level I, the x-ray examinations are almost equally divided between males and females, the main exceptions being mammograms, cholecystograms and barium enemas. For these examinations there is in most countries a clear female predominance. In the Islamic Republic of Iran, 63% of all examinations are performed on males.

47. Although age and sex differences of populations receiving x-ray examinations are presented in this Annex, there has been no attempt to introduce any age-dependent correction factor in calculation of the effective dose equivalent commitment. A preponderance of old and ill persons in a population should theoretically reduce its risk of long-term effects compared to a population of workers. On the other hand, screening x-ray examinations involving children would, by a similar comparison, substantially increase the longer term risk to a population. At present there is very little information available on the impact of neoplastic and non-neoplastic diseases on radiation risk coefficients or upon reduction in lifespan.

C. IMPACT OF SPECIALIZED TYPES OF EXAMINATIONS

48. In the UNSCEAR 1977 and 1982 Reports, the Committee suggested that the increasing use of newer techniques might decrease the radiological exposure of the population. It was uncertain whether imaging modalities that do not utilize ionizing radiation (such as ultrasound) would replace existing radiologic procedures or simply add to the total number of procedures. Since 1982, magnetic resonance imaging has also become available; in this technique, images are generated by the induction of radiowaves in a magnetic field, and no ionizing radiation is utilized. At present, the main use of magnetic resonance appears to be for brain and spinal cord imaging, but no numerical data exist on the frequency or availability of this technique.

49. Hinz et al. [H14] and Schwarz et al. [S5, S6, S7] have examined the replacement of specific radiographic examinations by sonography. Their reports cover the years 1977-1982. The authors specifically considered procedures related to the stomach, abdomen, gallbladder, pancreas and urinary system. In those areas, there was a decrease of about 50% in radiographic examinations (Figure III) and an increase of about 150% in sonographic examinations (Figure IV). Decreases of 10%, 2% and 46% were found in contrast examinations of the small bowel, colon and gallbladder, respectively.

50. Pelvic imaging procedures were specifically considered, with the expectation that radiological examinations of the pelvis might have decreased as pelvic ultrasound examinations increased. The data are of limited value, however, because most surveys include x-ray examinations of the hip and pelvis in the same category. While ultrasound might be expected to have substantially reduced the number of oral cholangiograms, in the United States, at least, the marked increase in frequency of biliary ultrasound certainly had not decreased their number, although it may have reached a plateau as from 1980. There has been a substantial increase in radionuclide hepatobiliary imaging, while percutaneous cholangiography and intravenous cholangiography have markedly declined [E6].

51. Although there was a marked increase in echocardiography and nuclear medicine cardiac studies in the United States between 1972 and 1980 (Table 14), the number of invasive cardiac contrast procedures has substantially increased rather than decreased. It may be concluded that in developed countries ultrasound has replaced some radiographic procedures in imaging of the gallbladder, the kidneys and the foetus.

52. Data are available concerning the use of computerized tomography in several countries. In Japan, 14.4 million procedures (123 per 1,000 population) were performed in 1979 [N10, N11]. Of the total, about 75% were computerized tomographic scans of the head and the remainder were computerized tomographic body scans. Over 60% were performed on patients 45 years or older. Some related data are also available from the United States [E6]. Prior to 1970 computerized tomography and ultrasound procedures were hardly used at all. Table 15 shows that the increase in computerized tomography of the head coincides with a substantial reduction in the number of radionuclide brain scans being performed and

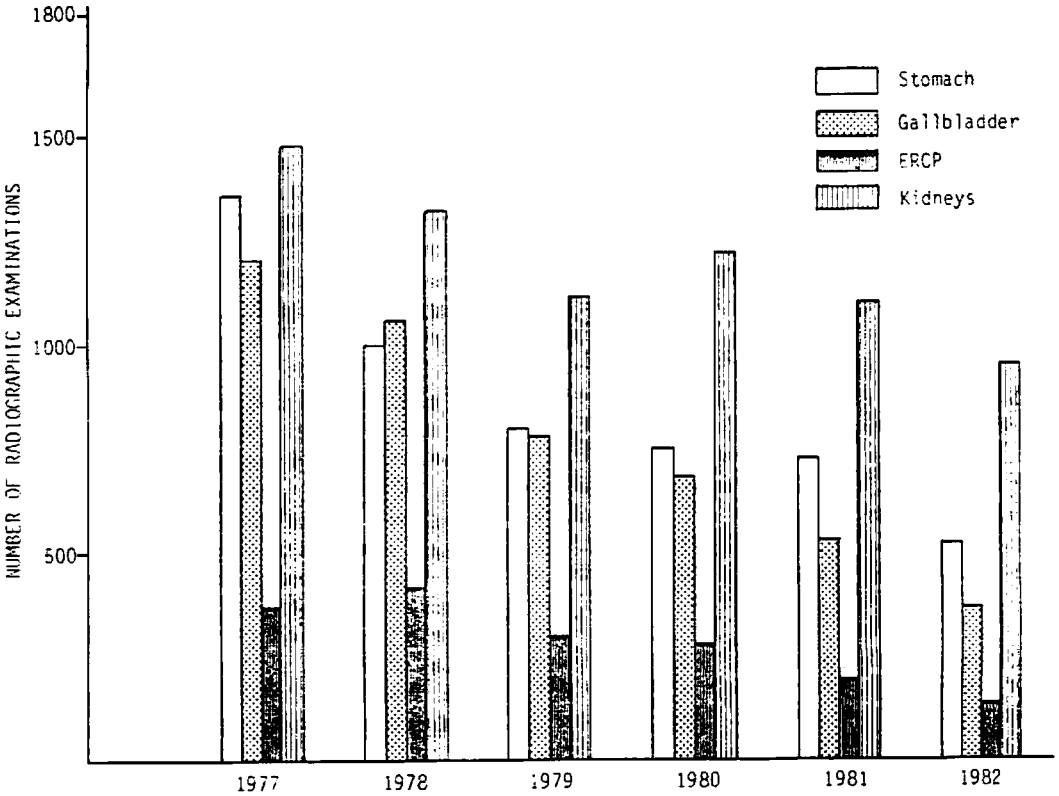

Figure III. Changes as a function of time (1977-1982) in the number of x-ray examinations of various organs in a University clinic in the Federal Republic of Germany. (ERCP = encoscopic retrograde cholangio pancreatography).
[H14]

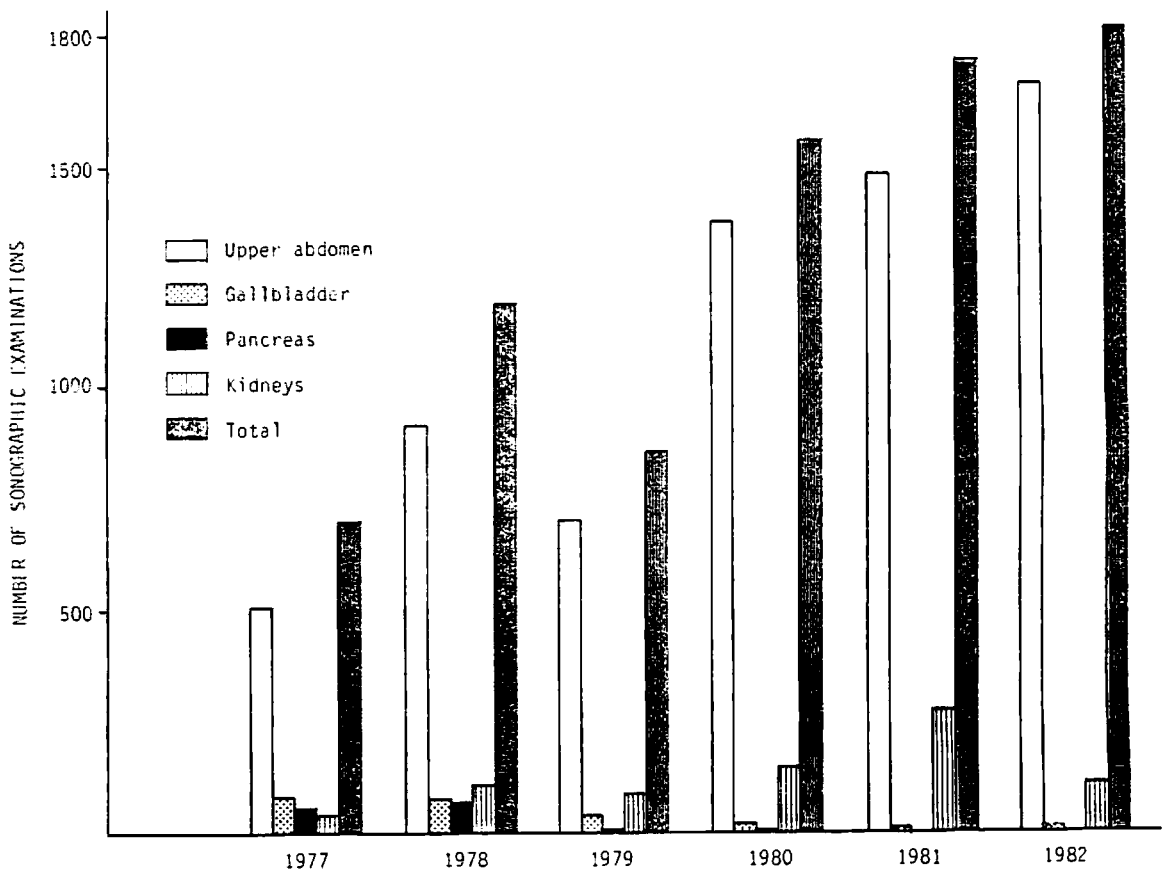

Figure IV. Changes as a function of time (1977-1982) in the number of sonographic examinations of various organs in a University clinic in the Federal Republic of Germany.
[H14]

that, during the same period, pneumo-encephalograms became extremely rare.

53. Evens et al. [E3, E4, E5, E6] and Hughes [H21] examined trends in the use of computerized tomography in the United States during 1981-1983. Total scans increased from 2,337,000 in 1981 to 4,303,000 in 1983. Cranial scans accounted for 75% of such procedures in 1981 but decreased to 63% in 1983. During the same period, computerized tomography scans of the spine increased from 3% to 10% and other body scans increased from 22% to 27%. However, the rate of increase in the use of computerized tomography slowed markedly with the percentage increase over the previous year being 53% from 1981 to 1982 and 21% from 1982 to 1983.

54. Imaging procedures of the abdomen are difficult to evaluate since many computerized tomographic examinations of the abdomen and ultrasound examinations are done for retroperitoneal pathology. The increasing use of abdominal ultrasound and computerized tomography may have decreased the number of ordinary x-ray examinations of the abdomen being performed in the United States between 1980 and 1982 [E6]. Whether this is a real finding or simply due to different reporting of the two surveys is unknown. Computerized tomography has decreased the need for some arteriograms [B2, K7, W12], but in general there has been a net increase in examinations utilizing relatively large amounts of ionizing radiation. Another interesting question is whether computerized tomography of the lumbar spine has partially replaced myelography. In the United States the frequency of myelography examinations has continued to increase in spite of increasing computerized tomographic examinations of the spine.

55. Another particularly important trend is that of mammography. Table 16 indicates the significant increase of mammography examinations per 1,000 female population. At present, the rate in the United States is slightly greater than 10 per 1,000 females annually.

56. In the past decade there has been a rapid expansion of both digital and interventional technologies. Digital technology in this context refers to the recording of transmitted photons on an image intensifier or other such receptor rather than on film. This process allows computer manipulation of the images. This technology has found widespread use in vascular radiology, but it can also be used in other examinations. Interventional technology refers to a number of techniques in which radiology is used to guide the radiologist or other physician in a semi-surgical diagnostic or therapeutic procedure. Examples of such procedures are placement of drainage catheters, needle biopsy of various lesions, catheter placement for infusion of pharmaceuticals, and balloon catheter placement for occlusion or dilatation of blood vessels. Most of these procedures require lengthy periods of fluoroscopy and may result in high absorbed doses to the patient as well as to the operator. Data on the frequency of such procedures are not available at this time.

D. EXPOSURE AND ABSORBED DOSE

57. The distribution of exposure or absorbed dose in a patient as a result of a diagnostic x-ray examination depends upon (a) the amount of incident radiation; (b) the location and direction of the incident beam; and (c) the quality and attenuation of the radiation in the body. The amount of incident radiation depends upon exposure at skin entrance and the size of the radiation field. Exposure for an examination is sometimes reported free-in-air (i.e., without the body present) and sometimes as skin surface exposure (i.e., with the body there). Alternatively, the absorbed dose in soft tissue at the surface may be reported. The ratio of the exposure on the body to exposure free-in-air for examinations that contribute significantly to the radiation dose is approximately 1.2 to 1.4. Some authors have reported results in terms of energy deposited in the total body or in a given organ rather than in terms of average energy absorbed per unit mass. It would be worthwhile to unify methods of expression of patient exposure and dosimetry.

58. While the average absorbed dose in a given organ of the body depends on all the factors listed above, some consolidations are possible when considering relative distribution of absorbed dose in the body. If the physical characteristics of the beam (tube potential, tube current, radiation field size, location and filtration) are the same for a series of exposures, the relative absorbed dose distribution is independent of the amount of incident radiation. Approximate but adequate constancy is also obtained for a small range of patient sizes for a particular type of examination. While the exposure at the body surface of adults for a given type of examination may range over a factor of up to 40 (Figure V) [U9], the relative absorbed dose is usually considered to be adequately constant so that the effective dose equivalent for a given type and projection of an examination is proportional to the exposure or absorbed dose of the incident radiation. It should be noted, however, that relative absorbed dose distributions change so that a different numerical proportionality is obtained for children and infants than for adults.

59. In the UNSCEAR 1977 Report [U4], typical skin doses in the primary beam for various examinations were given. More recently, data on trends and variability of exposures in the United States have become available from the Nationwide Evaluation of X-ray Trends (NEXT) programme [U9]. In this programme, exposure is measured for five projections using specified geometry and measured free-in-air. Histograms for composite data for the years 1973-1980, shown in Figure V, indicate a rather wide distribution of such exposures. Very similar data for 1975-1985 are available from the NEXT programme in Canada [C2] Italy [I1]. With the advent of rare-earth screens and faster film-screen combinations, one might expect that the mean exposure at skin entrance would be decreasing. However, data from the United States suggest that as of 1983, in spite of technological advances, there has been little reduction in average exposure [U9]. Therefore, one is led to conclude that world wide, skin doses have not

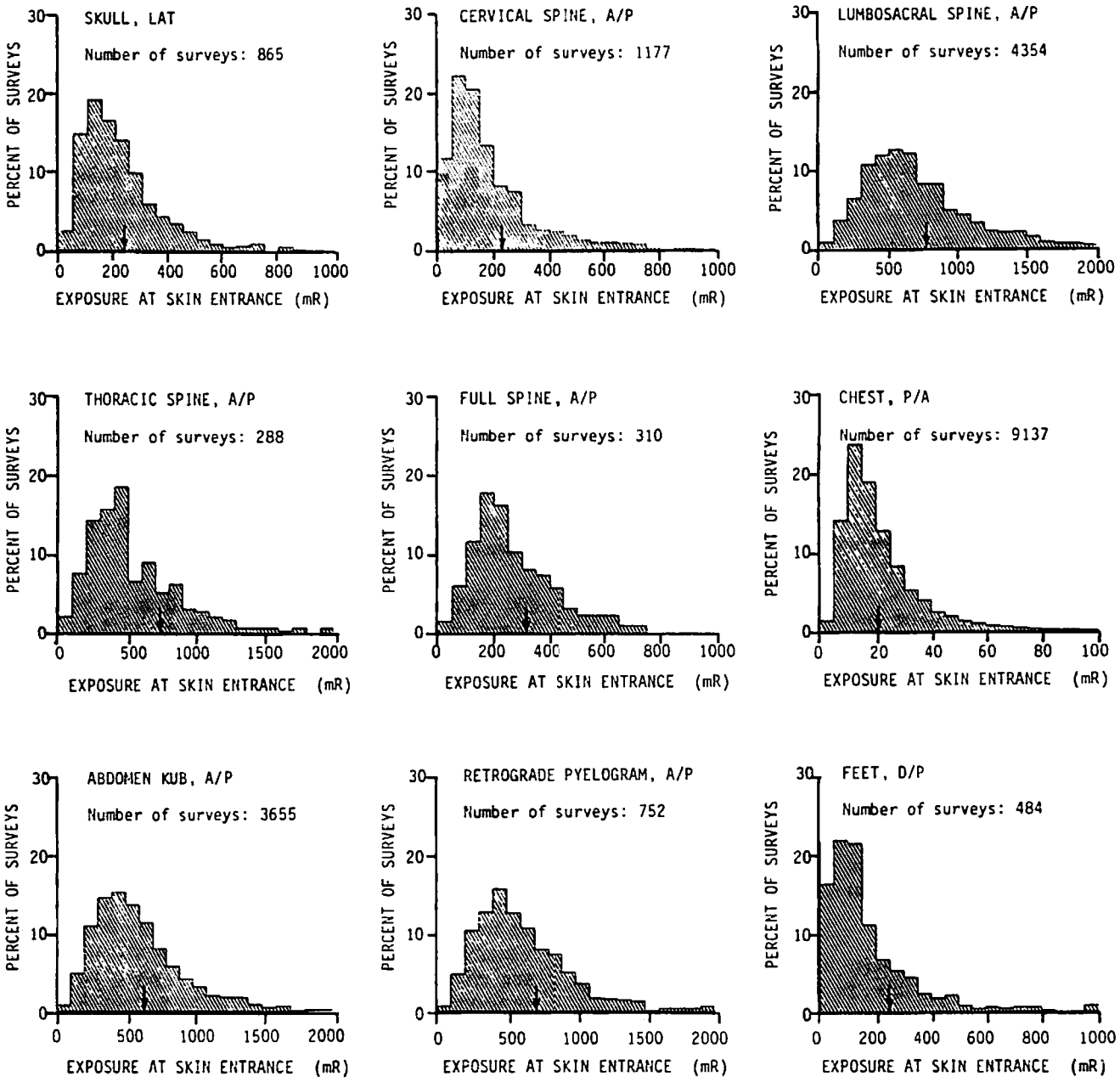

Figure V. Exposure at skin entrance (ESE) for various examinations (mR). (Exposure determined as "free-in-air"); (arrow refers to mean exposure value).
[U9]

changed significantly from those identified in the UNSCEAR 1977 Report.

60. For purposes of this Annex, in order to be able to compare results from different studies, exposures measured free-in-air have been converted to skin exposure using a backscatter correction factor of 1.3. The skin exposures were then converted to skin dose equivalent utilizing an absorbed dose to exposure conversion factor of 1 rad per roentgen or 1 centigray per roentgen. Mean skin doses in the primary beam for various diagnostic x-ray examinations have been measured in Canada, Italy, Poland, United Kingdom and the United States (Table 17). Skin doses were measured by placing thermoluminescent dosimeters (TLDs) on the skin surface of 1,340 patients during 22 types of diagnostic radiographic examinations in Beijing, China [J4]. These results are not significantly different from results in other countries. Mean skin doses for five common diagnostic examinations in the United Kingdom have been reported by Harrison [H2]. These data indicate an approximately tenfold range in dose within one country, with almost all of the distributions showing a long tail that extends into the upper range of doses. Absorbed doses reported for a given examination also differ significantly between countries.

61. In addition to actual measurements and surveys made of doses received from radiological examinations, simpler methods have been described involving the use of nomograms to estimate exposure or absorbed dose from machine parameters. Veitch et al. [V6] investigated the use of nomograms for estimating

the exposure to the skin of a patient from three-phase equipment. Substantial work had been done years ago on single-phase equipment [S22, M24]. More recently, Edmonds [E1] described a simple and rapid method for calculating patient skin doses based on peak voltage, current, source skin distance and filtration. In fact, the method provides a quick estimate of exposure, although it may overestimate the dose for three-phase x-ray equipment by a factor of nearly 4 [S12]. A nomogram for estimating skin doses in x-ray diagnostic examinations has also been published [W1].

62. Absorbed doses in various organs are needed in order to calculate the effective dose equivalent. The organs of interest include the thyroid, bone marrow, the lungs, the female breast and the gonads. A Monte Carlo computer technique and a mathematically describable anthropomorphic phantom have been developed and can be utilized to calculate tissue-air ratios for selected organs [K4, R8]. Drexler et al. [D5], Jones [J10] and Kramer [K16] calculated organ doses for x-ray diagnosis utilizing Monte Carlo methods for both male and female phantoms designed according to ICRP reference persons. By utilizing these techniques, one can derive mean absorbed doses in a number of organs, normalized to unit exposure measured free-in-air under different conditions of beam quality and field size. These provide information for thyroid, bone marrow, lung, female breast and gonads. Williams et al. [W15] constructed three-dimensional phantoms using computer tomographic data from patients, which allows very accurate calculations of absorbed dose in organs.

63. Monte Carlo calculations of organ doses take into account only the primary radiation and radiation scattered within the patient. The scattered radiation and leakage radiation from the diagnostic source assembly, as well as other stray radiation, is usually not included. When the organ of interest is within the useful beam, stray radiation is not likely to account for more than 1% of the organ dose. However, when the organ of interest is at least several centimetres outside the useful beam, neglecting the contribution from the stray radiation may result in underestimating organ dose by as much as 25-50% [B5]. To account for this, international [I3] and national [D4] standards required for equipment restrict the dose rate outside the beam to 1 per hour and require efficient collimation.

64. It is much more difficult to calculate organ doses when fluoroscopy is utilized. The reason for this is that automatic brightness controls are often used for fluoroscopic examinations, and the exposure rate and beam quality change as the beam is moved. Thus, even when exposure parameters are known and exposure times recorded, the confidence limits on the absorbed doses from fluoroscopy are larger than those from radiography. To overcome this difficulty (at least partially), area exposure product meters can be used [I4].

65. Fluoroscopic examinations also present other unique problems due to the continuous changes in beam direction, length of examination time, field size and positioning in the course of examination. During standard radiographic procedures, matters such as the incident exposure side of the patient are quite straightforward, but during fluoroscopic examinations the incident and exit sides of the patient are often changing. The length of time that a fluoroscopic procedure takes, and thus the resulting absorbed dose, varies widely depending on the complexity of the examination, the co-operation of the patient and the skill of the operator of the equipment. Harrison [H2] has reported total fluoroscopic screening times used in the United Kingdom for a barium meal examination. The average time was 146 seconds, but the range was 1-620 seconds. Rowley et al. [R9] have also reported the median exposure times for various fluoroscopic examinations in local areas of England. They report the following: barium swallow, 180 seconds; barium meal, 180 seconds; and barium enema, 150 seconds. Little difference in time was noted in relation to the sex of the patient, however, males consistently received higher absorbed doses due to larger body size. Longer screening times of 337 seconds for a barium enema and 240 seconds for a barium meal are reported by Pandovani [P1] in Italy. Maccia et al. [M1, M2] have reported on the use of fluoroscopy in France; their mean fluoroscopic times for various examinations are shown in Table 18. It is of interest that fluoroscopy is used to position or centre patients in 25-50% of examinations that are usually considered radiographic examinations.

66. In general, fluoroscopic procedures result in much higher doses to the individual patient than most other types of standard radiographic examinations. For this reason, the achievable dose reductions could in principle be larger. Several authors investigated the effect of variation in equipment design on patient dose. Tole [T4] reported that fluoroscopic machines with the tube placed over the table often give substantially higher organ doses (particularly to the male gonads) than machines with the tube under the table. This occurs because for most fluoroscopic examinations the patient is in the supine position or facing forward and the male gonads are relatively anterior in location and closer to the x-ray tube as well as being unshielded. Zeck and Young [Z3] pointed out the very high radiation levels that can be associated with C-arm fluoroscopes. In general, the minimum source-skin distance for a C-arm device is 30 cm. Spacers are usually used to maintain this distance but are sometimes removed and not replaced. If the patient is then positioned close to the tube, the entrance skin exposure rates will be much higher than usually calculated.

67. Fluoroscopy times accompanying coronary angiograms are usually about 10-20 minutes [A3]. Cascade et al. [C5] have recently reported exposures and fluoroscopy times for the relatively new technique of percutaneous transluminal coronary angioplasty. In this technique, fluoroscopy is utilized to monitor the progress of a balloon catheter introduced in order to dilate one or more stenotic coronary arteries. When only one stenosis was dilated, the fluoroscopy time was 36 minutes and skin dose was 0.5 Sv. When two

stenoses were dilated, the fluoroscopy time was 51 minutes and patient skin dose was 1.0 Sv. Similar data have been reported by Faulkner [F2].

68. While technical parameters affecting absorbed dose are relatively well known in some countries, the technical factors used for specific examinations are often unknown. Large portions of the population receive uncertain but possibly large doses. For example, Hussain [H22] surveyed x-ray installations in Bangladesh and reported that the current and voltage indicators on many machines did not work at all and that over 40% of machines either had no collimator or that it was not functional. Thus the exposure at skin surface, field size and location of the centre of the field are generally not known. Under these conditions, calculations of absorbed doses to various organs or determination of effective dose equivalent are virtually impossible.

69. Similar conditions were described for India by Bhargava [B16]. In a survey of diagnostic x-ray installations, 20% of the machines produced excessive exposure during fluoroscopy and showed excessive leakage from the tube housing. In 20% of the machines neither cones nor collimators were used to control the beam size. Das et al. [D1], reporting on exposure to the patient's skin for various examinations in India, indicated that a fluoroscopic examination of the chest results in a skin dose of approximately 120 mSv.

70. In Beijing, China, Sun [S32] has measured skin exposure during 2,395 fluoroscopic chest examinations at 44 hospitals, and he reports mean skin doses of about 10 mSv. Wu et al. [W23] have reported on skin exposures in 370 patients who had various upper gastro-intestinal examinations in China. For most examinations, the skin doses were 50-180 mSv. Weng and Wu [W11] measured skin exposures for 30 patients in China having cardiac catheterization. The skin dose in the field was 100 mSv for the fluoroscopy alone and an additional 260 mSv for the radiographic portion. All these measurements were made on the skin surface utilizing thermoluminescent dosimeters. Because the use of fluoroscopy is still widespread in developing countries where data are scanty, the absorbed dose to the population may be estimated only very roughly.

71. Absorbed dose from mass screening examinations continues to be of interest to the Committee. The average skin dose equivalent in the field for a mass chest x-ray examination in Japan in 1980 was 1.5 mSv for adults and about 0.8 mSv for children [K22]. In many countries mass chest screening is often performed with photofluorography. Bengtsson et al. [B13] have indicated that the average dose to the breast from such examinations is about 2.0 mSv, almost as high as the absorbed dose from a mammogram. The effective dose equivalent for chest x-ray mass screening in France has been reported to be 0.07 mSv for radiography, 0.32 mSv for photofluorography and 0.98 mSv for fluoroscopy [L8]. The collective effective dose equivalent for this practice in France in 1980 was about 4,500 man Sv. In Japan, mass screening of the stomach is often performed utilizing photofluorography. Maruyama et al. [M13] have indicated that 4.1 million such examinations were performed in Japan in 1980; this represents about one examination per 30 people. The collective effective dose equivalent from this practice was estimated to be about 16,000 man Sv.

72. Knowledge of absorbed doses to the uterus, embryo and foetus is useful in situations where a pregnant woman has been exposed to diagnostic x rays. Glaze [G4] and Drexler [D5] described a computer-assisted procedure for estimating both patient exposure and foetal dose from radiographic examinations. The dose incurred in paediatric x-ray examinations is also of interest since a large portion of the child's body is often included in the primary beam [N2]. Morris [M36] calculated doses in Australia for patients in the age group 2-4 years and for those under the age of two. Similar Monte Carlo calculations are also available to assess the doses from paediatric x-ray examinations to the total body, bone marrow, thyroid, lungs, ovaries and testes [G5]. For typical examinations in paediatric x-ray diagnosis, Williams et al. [W16] and Zankl et al. [Z2] used a Monte Carlo code to calculate the doses to a baby and child phantom constructed from tomographic data.

73. Radiation doses to neonates requiring intensive care were examined in the United Kingdom by Robinson et al. [R5]. These babies are of particular concern since they may receive larger numbers of radiographs than adults, and the treatments often include barium examinations and computerized tomography scans. The marrow dose from all examinations was found to vary approximately inversely with birth weight. In addition, children with lower birth weight received more examinations (Table 19). Gustafsson et al. [G8, G9] have examined the relationship between body weight and energy imparted for children of various ages and body sizes. The energy imparted was less per kilogram for the older and larger children. There was, however, substantial variation in energy absorbed for children of the same weight. This variation was ascribed to technical factors, such as beam collimation. Gustafsson also discussed the relationship of beam direction to dose. Performing a chest radiograph in the posterior/anterior projection causes relatively larger dose to the bone marrow than when it is done in the anterior/posterior projection. The latter projection, however, delivers a larger dose to the breast and thyroid. Leibovic et al. [L4] reported on paediatric angiocardiography procedures, which provide the highest exposure per examination of any diagnostic paediatric procedure. The authors noted that as much as 25% of the exposure from such examinations was contributed by manual test exposures to adjust the technique. The average dose rate to the skin in the posterior/anterior projection for cine filming was 0.7 mSv per second and in the lateral projection 2.1 mSv per second. Fluoroscopy exposure rates were approximately 5% of this.

74. Of special interest is the dose received by the breast in mammography. Bates [B3] has reported on skin exposures in 27 screening centres in the United States. The results showed that a substantial reduction

in exposures and tissue dose was achieved during the course of this project. Rimondi [R3] has reported doses from mammography in Italy. He indicated that, even with the same type of x-ray apparatus and film-screen combination, very different exposure values were obtained, ranging over two orders of magnitude. Skin doses in this survey ranged from 2 to 220 mSv. The mid-plane doses were from 0.18 to 11.1 mSv.

75. Hammerstein [H1] and Stanton et al. [S26] reported doses measured in a breast phantom designed to simulate a breast with a uniform mixture of equal amounts of adipose and glandular tissue. Similar results have been reported by Panzer [P5] from a study of 170 facilities in the Federal Republic of Germany. Average glandular doses for non-screen films ranged from 5 to 35 mSv with a median value of 16 mSv, and for screen-film systems from 0.8 to 19 mSv with a median value of 6.6 mSv. Zuur [Z12] reported similar dose values from 12 institutions in the Netherlands, ranging from 1.0 to 8.8 mSv.

76. Sato [S2] carried out a survey on the radiographic technique and frequency of mammography in Japan. Of the 75 institutions surveyed, 45 utilized intensifying screens and film for mammography, 30 used a non-screen system and 20 did not have any special apparatus for mammography. There were approximately 2.9 exposures per examination, or 1.5 exposures per breast.

77. Gannon [G2] reviewed the equipment performance at 28 mammography centres in the United States. In this study the actual peak voltage was measured and compared to the dial reading on the xerox-mammography-type machines. In most cases the desired peak voltage was between 36 and 50 kVp. In only one case was the measured peak voltage that which was actually desired; in some instances the peak voltage differed by as much as 7 kV from that set on the machine. Such differences significantly affect image quality. The two-view (mediolateral and cranio-caudal) mid-line (3 cm depth) dose measured in a phantom ranged from 2.5 to 11.6 mSv.

78. The use of grids in mammography has been advocated to improve image quality and to reduce scattered radiation incident on the image receptor. Kirkpatrick [K11] measured the effect of such grids on patient dose. He showed that, although there was a gain in image quality, absorbed doses were approximately three times higher, unless there was a significant change in exposure parameters. Whether the improvement in image quality was worth the increased dose was not indicated, although the use of grids is usually restricted to circumstances where the thickness of the compressed breast exceeds 5 cm.

79. Measurement of absorbed dose from the newer technologies has also been a matter of concern. In the UNSCEAR 1982 Report, the large amount of literature reviewed indicated that the dose to the skin from a computerized tomography scan could be as high as 560 mSv, although in clinical practice the absorbed doses were mostly around 60 mSv. The dose distribution within the body from computerized tomography is markedly different from that from conventional radiography. In most radiography, the dose is highest on the incident side and lowest on the exit point; in most computerized tomography, the dose is lowest at the centre of the body section studied. The effective dose equivalent and absorbed dose from various computerized tomography procedures have been derived by Stieve [S31].

80. The x-ray beam of a computerized tomography unit is usually highly collimated, but the eye may be in or near the primary beam on scans of the brain or face, and the dose to the eye is of particular interest for radiation protection purposes. Lund et al. [L6] and Kronholz [K18] indicated, for brain computerized tomography, that although slice thickness and patient position have some effect on the absorbed dose in the lens of the eye, the greatest doses are those received when the scan is done with the gantry angled downwards in relation to the orbito-meatal (from the outer corner of the eye to the external ear canal) line. In this circumstance the eye is included in the primary beam. This positioning factor caused the dose to the lens of the eye to increase by a factor of 2-4 compared to standard orbito-meatal scans. Doses to the lens of the eye from cranial computerized tomography are in the range of the absorbed dose from other neuro-radiological procedures. Isherwood et al. [I5] indicated absorbed doses to the lens as follows: orbital hypocycloidal tomography, 120 mSv; petrous bone tomography, 100 mSv; cerebral angiography, 50-100 mSv; pneumo-encephalography, 20 mSv; and skull examination, 15 mSv. Panzer [P4] collected dose values from 120 facilities in the Federal Republic of Germany. Preliminary results show a large variation in the dose values (free-in-air) on the axis of rotation, from 10 to 200 mSv per slice, with a median value of 30 mSv. By calculations using Monte Carlo methods, the dose (free-in-air) can be converted into organ doses [D5].

81. The radiation doses to various organs for the types of computerized tomography scanners used in Japan have been published by Nishizawa et al. [N10, N11] and are given in Table 20. While the exact values depend upon the technique and the type of scanner, the values presented are in general consistent with those reported by other authors [B1, C8, E7, E8, G3, I5, M20, M25, S10, S11, S24, S35, W12]. McCrohan et al. [M16] surveyed 250 computerized tomography systems in the United States to determine the radiation dose from a head scan. For the typical adult scan the absorbed dose was 22-68 mSv; doses varied by a factor of two for the same manufacturer and model of machine. Beck et al. [B4] have devised a Monte Carlo model for absorbed dose calculations in computerized tomography.

82. There is a trade-off between image noise and radiation dose [T5]. All calculations used by the computer to construct the image are limited by the statistical distribution of the detected photons. Several attempts have been made to reduce the dose through various technical modifications. Dose reduction can be achieved by radiating and collecting data only during a portion of the scan cycle. Oppenheim [O5] found that artifacts caused the method to be of limited usefulness. More recently, Stanton et al. [S27] devised

a method that collects data over the entire scan but exposes the region of interest to a higher dose than other anatomical structures within the scan volume. This is accomplished through the use of a variable thickness filter; dose peak reductions of up to 80% are claimed for head scans. Moseley et al. [M37] discussed various methods of reducing radiation dose in the management of intracranial lesions that are clinically followed by use of computerized tomography, but they did not report any quantitative dose reduction factor. McCullough [M23] suggested that the performance of each computerized tomography scanner be specified and checked in order to ensure a typical level of performance and to provide a baseline value for a programme of quality assurance. Parameters tested usually include slice geometry, patient dosage, artifactual behaviour and contrast detail performance.

83. Digital medical radiographic systems are now becoming available in most developed countries. The contrast resolution of such instrumentation is limited primarily by quantum mottle. Rimkus et al. [R4] indicated that the number of meaningful levels of grey that are imaged will significantly affect the radiation dose. For example, if 128 meaningful visual shades of grey on an image require a dose of 17 mSv to the patient, simply raising the level of contrast resolution to 256 shades of grey will increase the dose by a factor of 5-10. However, since such contrast resolution is rarely needed for diagnosis, this is an area where unnecessary dose can be avoided.

84. The frequency of dental examinations was discussed in section I.A. Data on dental exposures are available from the Nationwide Evaluation of X-ray Trends (NEXT) survey in Canada [C2]. For a dental bite-wing posterior examination, a dose at skin entrance was recorded with a minimum of 0.56 mSv, a maximum of 43 mSv and a mean of 4.7 mSv. For periapical examinations, the maximum was 2.6 mSv, the minimum was 0.57 mSv and the mean was 2.2 mSv (standard error 1.1 mSv). Results from 200 dental facilities in the Federal Republic of Germany were reported by Panzer [P6]. Entrance doses for examinations of a molar tooth ranged from 2.5 to 45 mSv with a median value of 8.5 mSv. Comparison with earlier results (1970) showed a remarkable decrease in entrance doses.

85. In Japan [M11], the annual per caput doses for dental radiography were estimated to be 0.09 Sv (genetically significant dose) and 13 Sv (mean bone marrow dose). Iwai [I6] compared absorbed doses to the gonads and to bone marrow for both intra-oral and panoramic dental examinations and reported the total risk for intra-oral examinations to be lower than the dose for the less frequent panoramic examinations.

86. Radiation doses from dental x-ray examinations were discussed in detail in the UNSCEAR 1982 Report [U5]. The radiation exposure for dental films may be decreasing somewhat. The Nationwide Evaluation of X-ray Trends (NEXT) programme in the United States [U9] indicated that the mean dose at skin entrance from dental bite-wing posterior films was 9.1 mSv in 1973 and 4.3 mSv in 1981. There was an increase in the dose from pantomographic examinations, from 0.3 mSv in 1973 to 0.8 mSv in 1981.

87. Weighted dose equivalents in the United Kingdom have been calculated by Wall et al. [W3]. These values are 20 μSv for intra-oral examinations consisting of two films, 30 μSv for extra-oral examinations consisting of two films and 80 μSv for one pantomographic film. They estimated the collective weighted dose equivalent to the population of the United Kingdom to be 212 man Sv. The mean dose equivalent to various organs per dental examination is shown in Table 21.

88. Pellerin et al. [P10] reported on exposures in both phantoms and patients for various types of dental examinations in France. The intra-oral exposure was the most commonly used and delivered a maximum dose to the skin of about 15 mGy. The pantomographic view gives a picture of the entire dentition but delivers a dose of approximately 10 mSv to three intracranial "hot spots". Tingey [T2] has re-emphasized the need for quality control procedures to reduce the exposure factors and also to avert repeat examinations.

89. Dosimetry in panoramic examinations has also been studied in the Soviet Union by Trunov et al. [T8] and Kirko [K10] utilizing thermoluminescent dosimetry and anthropomorphic phantoms. The radiation dose was 15-20 μSv per film for examinations of the upper jaw and 25-30 μSv per film for examinations of the lower jaw. The thyroid doses were 40-180 μSv and gonadal doses were 13-150 μSv.

90. Hayami [H12] recently devised a Monte Carlo computer programme to estimate exposure to the head and thyroid for panoramic intra-oral x-ray tube radiography. With 55 kV (kilovolts) and 0.5 mAs (milliampere seconds), the energy imparted for a routine examination was 2.1 mJ to the head from each exposure of the mandible and maxilla, about 8.5 μJ to the thyroid from a mandibular radiograph and 1.7 μJ to the thyroid from a maxillary radiograph.

E. CAUSES OF DOSE VARIATION AND POSSIBILITIES FOR DOSE REDUCTION

91. Some possibilities for dose reduction are found by examining the causes of variation in dose for a given examination. Dose reduction cannot be taken as an ultimate goal in medical radiation since the images generated must have sufficient informational content to be of diagnostic value. An underexposed radiograph that cannot be interpreted is of no value to the patient even though the absorbed dose is low. Many aspects of image quality and its assurance were discussed at a seminar organized by the Commission of the European Communities [C10]. The actual assessment of priorities and analysis of cost versus benefit in this regard is beyond the scope of this Annex. Such analyses would be highly dependent on the availability of operating equipment and the knowledge of health care practitioners of a given country. There certainly are, however, some simple and low cost methods that can be used to substantially reduce absorbed dose. Russell [R10] has

presented one form of methodology that could be utilized for such assessments.

92. Wall et al. [W4] have discussed a number of the factors involved in dose reduction. There have been many changes in diagnostic radiology techniques over the past 20 years, many of which might be expected to have had a significant effect on patient doses. The trend towards faster films and the advent of highly sensitive rare-earth screens should have resulted in lower exposures per radiograph. However, the adoption of rare-earth screens has been very slow; for example, only five out of 21 hospitals surveyed in the United Kingdom [W4] used them at all, and then only for obstetric examination or casualty work. High cost and poor spatial resolution due to quantum mottle are the most common reasons for their poor acceptance. Use of such rare earth screens appears to be higher in Italy [C11]. The use of new materials (such as carbon fibre) for construction of table tops, grids and film cassettes has the potential to reduce patient dose by 30-50% [H19]. Dose reductions in pelvimetry have been marked in the United Kingdom and the United States, as a result of fewer examinations, fewer projections and the increased use of ultrasound.

93. An appropriate combination of radiography and fluoroscopy can result in dose reduction, particularly for examinations of the gastro-intestinal tract [S28]. Fluoroscopic screening times have not decreased [W4], and therefore the hoped for dose reduction owing to the increasing use of image intensifiers has not materialized in the United Kingdom. In some departments automatic brightness controls are allowing examinations to be conducted in ambient light rather than in a darkened room. This increases the absorbed dose to the patient. Maccia et al. [M1] have reported the percentage of the collective effective dose equivalent in France that is contributed by fluoroscopy (Table 22). It appears that about 5,000 man Sv are contributed by the use of fluoroscopy to position patients prior to routine film radiography.

94. The effect of gonadal shielding upon gonadal dose has been discussed by Poretti [P14]. Such shielding is particularly effective if the gonads are in the useful beam. Although gonadal shields are relatively inexpensive and easy to use, their use is not widespread. Wall et al. [W4] have reported that in the United Kingdom gonadal shielding was used for males only 35% of the time for hip and upper femur examinations, 26% of the time for lumbar spine examinations and 15% of the time for pelvis examinations.

95. One area that has received some attention, with resultant dose reduction, concerns the tailoring of the size and shape of the beam to the area of interest and to the film size [C11]. Many older medical x-ray machines have a circular beam, while the film is generally rectangular. Johnson [J7] point out that collimation of the primary beam has been an evolutionary process, whose stages are, at first, circular cones, then, variable rectangular collimators and, finally, positive beam limitation. In general, rectangular collimators are almost as good as positive beam limitation, but with circular cones there is almost twice as much radiation given as needed (Figure VI). The shift from circular to rectangular collimation for chest radiographs in the United States is shown in Figure VII and the resultant reduction in the amount of x rays utilized is shown in Figure VIII. The situation is somewhat different with fluoroscopy. In this case the collimators are usually rectangular while the image intensifier is circular. If the operator wishes to use the whole of the circular image, there is about 25% additional and unnecessary radiation.

96. Use of lower voltage for a given study will require higher entrance surface doses. Contento et al. [C11] have reported that in France softer x-ray spectra are used for a given examination than in Great Britain and Italy. The voltage and radiation output variation of x-ray machines have been studied by Henshaw [H13] and Pauly [P9]. They observed variations from the desired voltage, ranging from 5% to 20% or more and averaging approximately 10%. Belletti et al. [B7]

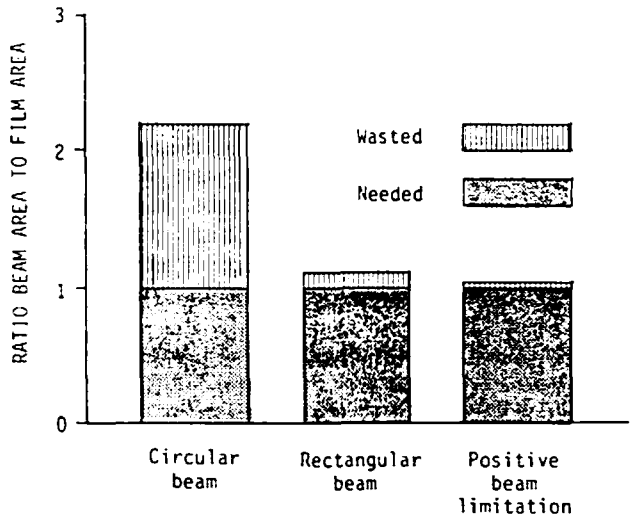

Figure VI. Mean ratio of beam area to film area for chest x rays in the United States, 1977-1983. [J7]

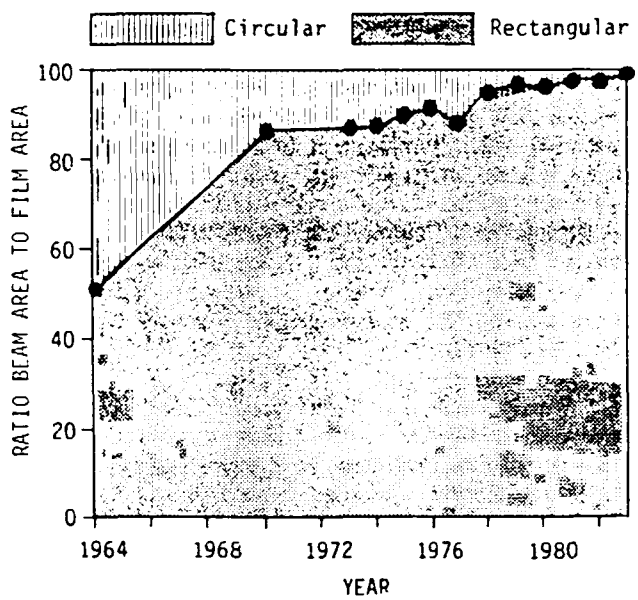

Figure VII. Trend in beam shape for chest x rays in the United States, 1964-1983. [J7]

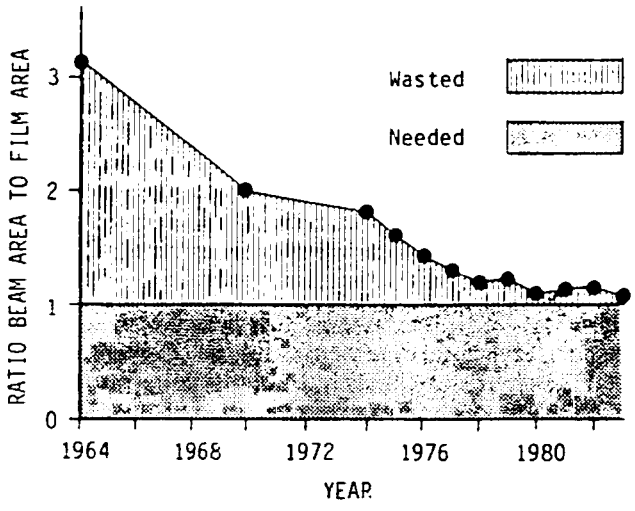

Figure VIII. Mean ratio of beam area to film area in the United States, 1964-1983.
[J7]

analysed the causes of repeat films. One of the more significant results was again the discrepancy between measured and selected peak voltage. Ten to 15 per cent of the machines examined were found to have a 10% difference between the set and measured values of peak voltage. Of all films that were spoiled, over 50% had been under- or over-exposed. Most spoilage was due to variations in the voltage produced by unstable generators, incorrect choice of either voltage or current, or malfunctions in film processing.

97. Practical recommendations to improve radiation protection in clinical mammography have been published [N3]. Breast compression is particularly important, not only to improve contrast and diminish motion unsharpness but also to reduce absorbed dose. Firm compression of the breast can reduce the absorbed dose by 25-50% while resulting in images of equal clinical usefulness. From an analysis of data from some 60 mammography installations throughout the United States [S17], it was concluded that the choice between xerographic and film-screen receptors is the most critical factor affecting breast exposure, followed by the choice of half-value layer and target material. Film-screen receptors without grids result in two to five times less absorbed dose than xeromammography. Panzer et al. [P5] have indicated that even a distinct increase in image detector sensitivity by switching to film-screen combinations did not always correspond to a comparable decrease in dose to the patient. Part of the reason for this may be that doses to obtain optimal images for various film-screen combinations for mammography vary by up to a factor of 2 [K12].

98. Breast phantoms have been utilized to assess absorbed dose from various imaging systems. Computation of absorbed dose is of course dependent on breast size, adiposity etc., but for analysis of detriment one needs to know the average size and composition of the breast in the population of interest. At the present time, research continues into alternative methods for breast imaging, such as thermography, ultrasonography, computerized tomography, magnetic resonance imaging and digital x-ray mammography. With the possible exception of ultrasonography, none of the other techniques has had any impact on the use of mammography.

99. The effect of patient size on dose variation received in diagnostic radiology has been studied by Maillie [M4]. The thickness of the patient is more important at low potential (voltage), and the average absorbed dose from radiographs taken on individuals of various thicknesses may differ by as much as a factor of 4 or 5. As the peak voltage is raised, the variation due to body thickness is reduced to a factor of 2. Of course the thickness of the irradiated part is not the only factor that influences the doses to various organs: for example, a taller person will have some organs farther displaced from the useful x-ray beam than a shorter person. Increasing radiation quality (voltage) reduces entrance skin dose but increases absorbed dose to organs at depth. The percentage reduction in effective dose equivalent is much less than the corresponding reduction in entrance skin dose. This potential method of dose reduction should be weighed against possible disadvantages such as reduced image contrast.

100. Dosimetric methodology can be a significant cause of reported absorbed dose variation. Padovani [P1] has recently shown some limitations of the Monte Carlo methods when they are used to determine absorbed doses to various organs as a result of medical practice. Monte Carlo methods assume good practice (e.g., excellent collimation). In his survey in north-east Italy he found major differences between the absorbed dose to organs calculated by Monte Carlo methods and that measured by thermoluminescent dosimeters. Actual testicular doses for specific examinations were higher, by factors of 4-50, than Monte Carlo calculations would suggest. Similar findings were reported for absorbed doses to the breast and thyroid. These findings are probably due to the organs being near the field of interest and poor collimation being utilized.

101. Stieve et al. [S30] and others have repeatedly emphasized that training in the use, calibration and quality assurance of x-ray equipment is an essential part of any dose reduction programme. In many, if not most, countries, over one half of x-ray examinations are performed by persons with little or no formal training. Even in well-developed countries, many non-radiologist physicians perform x-ray examinations though they have little or no formal training in uses of x rays or of x-ray protection. Proper theoretical and practical training of all persons involved in the medical uses of radiation is one of the most important ways to achieve dose reduction [S30, V5]. Cohen [C9] attempted to generalize and assess the benefits of quality assurance programmes. He estimated that in a developed country a quality assurance programme would lead to a reduction of 50% in the per caput whole-body dose equivalent from diagnostic radiology, from approximately 1.0 mSv per year to 0.5 mSv per year.

102. Some of the possible methods of dose reduction and their quoted dose reduction factors are summarized

in Table 23. The largest dose reduction factors occur as a result of switching from chest fluoroscopy and photofluorography to chest radiography, with dose reduction factors of about 20 and 5, respectively. It should be mentioned that economic and other factors often dictate what equipment is available to be used. Certainly, in the correct clinical setting, chest fluoroscopy is preferable to no chest x-ray at all. The simplest and least expensive methods that do work and that do offer modest dose reductions are (a) installation of collimation on machines; (b) added beam filtration; (c) the use of gonadal and thyroidal shielding; and (d) proper film processing. The judicious use of radiographic examinations and the elimination of non-productive examinations, which are another area for potential dose reduction, have been the topic of several recent WHO reports [W19, W22]. Discussions have centred on the efficacy of screening or pre-operative chest x-rays, skull films after minimal head trauma, pre-employment examinations of the lumbar spine or the chest, and examinations of the genito-urinary system and sinuses in children [G8, G9].

F. MEASURES OF RISK

103. The genetically significant dose (GSD) for a population has been used as a measure of the genetic detriment to be expected from a practice. It is defined as "the dose which, if given to every member of the population, would produce the same genetic detriment as the actual doses received by the various individuals." In some countries, such as China, gonadal doses and GSD may be greater than had been previously estimated. Apparently fluoroscopy is used in some provinces of China to check for the presence and location of intra-uterine contraceptive devices. Zheng et al. [Z10] have reported that mean skin doses for such examinations measured with TLDs was 8 mSv. In other countries there are problems in determining gonadal doses because of the lack of good data regarding the presence of collimation. As was mentioned earlier, in India and Bangladesh 20-40% of the machines have no functional collimation. Many genetically significant dose surveys have been performed and were summarized in both the UNSCEAR 1977 Report [U4] and the UNSCEAR 1982 Report [U5]. Since then, Sohrabpour et al. [S19] reported that in the Islamic Republic of Iran the genetically significant dose in 1980 was estimated to be 93 μSv, with the male and female contributions being 57% and 43%, respectively. In the province of Manitoba, Canada, the genetically significant dose was calculated in 1979 by MacEwan [M3] to be 260 μSv.

104. Kumamoto [K22] indicated that the genetically significant dose from mass chest x-ray examinations in Japan in 1980 was 0.17 μSv. The genetically significant dose due to computerized tomographic examinations in Japan in 1979 was estimated to be 1.1 μSv.

105. In France in 1982 the annual genetically significant dose was estimated to be 0.29 mSv [B10]. This represents a 64% increase from 1957. Table 24 also shows that the genetically significant dose for females is more than twice that of males (0.20 mSv versus 0.09 mSv). Examinations of the pelvis/hip and intravenous urography contributed almost 60% of the genetically significant dose. Fluoroscopy accounted for only 10%, while x-ray examinations contributed 90%.

106. Figure IX indicates a very high gonadal dose in French children under one year, which is apparently due to mandatory screening for hip dysplasia. The average dose equivalent to the female and male gonads from various examinations is shown in Tables 25 and 26, respectively.

107. Genetically significant doses in various countries are shown in Figure X and Table 27. It is clear from these data that the average gonadal dose (as well as skin dose) often varies between countries by a factor of 3 or more. Poretti [P14] has reported on both gonadal dose and genetically significant dose in Switzerland, where the GSD rose from 0.19 mSv in 1971 to 0.23 mSv in 1978. He also calculated the dose to the gonads with and without gonadal shielding for

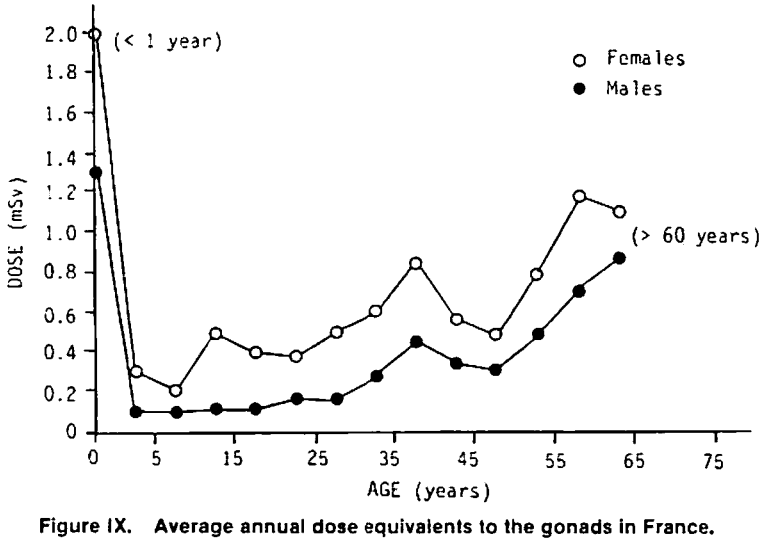

Figure IX. Average annual dose equivalents to the gonads in France. [B10]

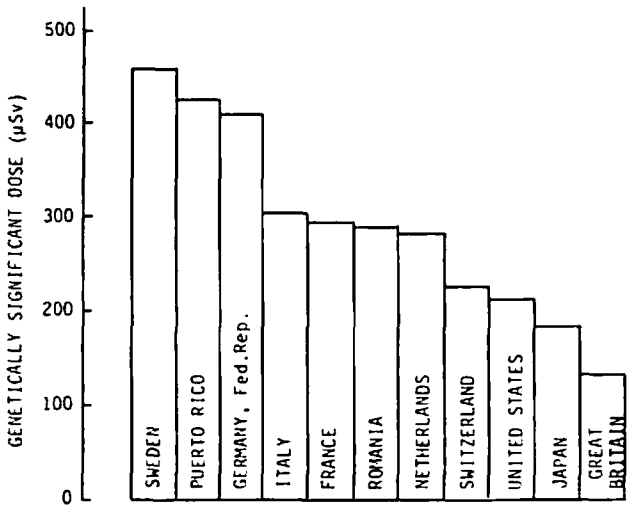

Figure X. Annual genetically significant dose in various countries.
[B10]

four common examinations. Gonadal shielding reduced gonadal doses by a factor of 2-10, depending upon the examination and on the distance of the gonads from the area of interest being radiographed.

108. According to estimates made by Kudritsky et al. [K19, K20] for the Russian Soviet Federative Socialist Republic, the trend is towards a gradual increase in the mean per caput gonadal dose. During 1970-1980, this increased by nearly 50%, probably owing to an increase in the number of special examinations and in examinations of the digestive organs and the osteo-articular system. The value of the genetically significant dose also changed. It increased by 0.06 mSv during the decade, mainly as a consequence of examinations involving radiation of the pelvic region (Table 28).

109. The genetically significant dose includes only absorbed dose that can be expected to affect the progeny; it does not take into account the somatic effects in the exposed population. Examples of the limitations of the genetically significant dose concept in practice have been given by Kaul et al. [K4]. In cases of a simultaneous increase in both the rate of examinations involving ionizing radiation in a given population and the number of alternative procedures applied in paediatric examinations in the same population, the genetically significant dose may lead to a misinterpretation of population exposure. Kaul et al. [K4] have therefore recommended that when sources of radiation exposure are being compared, the genetically significant dose should be indicated with estimates of somatic radiation exposure. The authors also pointed out significant limitations in the use of the genetically significant dose, particularly in countries where the age distribution changes with time. Under such circumstances the genetically significant dose alone is an unreliable indicator of the state and trend of medical radiation exposure; comparative evaluations of mean radiation exposure in different populations will also be of limited value. The latter point will become more important over the next several decades, when the world's population is expected to age markedly.

110. Stochastic risk estimates of various types for Japan have been published by Hashizume [H3, H4]. Annual per caput bone marrow dose in Switzerland was reported by Poretti [P14] as 0.63 mSv in both 1971 and 1978. The average active mean bone marrow dose to the United States population from radiological procedures in 1980 was about 1.3 mSv if the method of Shleien et al. [S9] is used for calculation. This compares with 0.83 mSv in 1964 and 1.0 mSv in 1970. Estimates of mean per caput marrow doses are very dependent upon the modelling parameters utilized. While the relative contribution by examination type does not change appreciably according to the method, the numerical quantity obtained is significantly different [R8, S9].

111. Beentjes et al. [B6] reported that the annual per caput "somatic effective dose" in 1980 in the Netherlands from diagnostic radiology was about 0.5 mSv and that the average somatic dose per examination was approximately 0.8 mSv. The somatic effective dose was defined as the uniform whole-body dose that would cause the same somatic risk as the actual non-uniform dose from the x-ray examinations.

112. A somatic dose index has also been proposed [L7] that utilizes individual organ doses weighted according to sex-dependent factors for the relative radiation risk and not weighted for the gonads. Kaul et al. [K4] compared the ICRP weighting factors and the modifications occurring when the genetic risk is neglected: their conclusion is that excluding the genetic risk has an effect less than the uncertainty involved in the calculation of the absorbed dose to an organ.

113. Calculations of effective dose equivalent from diagnostic procedures must include an analysis of the dose distribution within the body. The dose equivalent in an organ, T, for a given radiographic examination must be obtained by the formula:

$$H_T = \Sigma_k n_k D_{T,k} Q \qquad (1)$$

where k is the type of view involved in the examination, n_k is the number of films for the view k; $D_{T,k}$ is the average absorbed dose in the organ for view k, and Q is the quality factor. Q is 1.0 for x rays used in diagnostic radiology.

114. The effective dose equivalent, H_E, for an examination of type I is obtained from the following equation:

$$H_{E,I} = \Sigma w_T H_{T,I} \qquad (2)$$

where w_T is the weighting factor for each organ given in ICRP Publication 26 [I2]. One main difficulty encountered by most authors has been in selection of the "remainder" organs as required by the ICRP definition, which may change from one examination to the next. The selections, however, are not consistent. Some potential solutions to this problem have been suggested by many groups, e.g., Stavitsky et al. [S29]. In many published articles in which effective dose equivalents have been reported, the methods for choosing remainder organs are not given.

115. Calculations of the effective dose equivalent for different types of examinations in Poland in 1976 and in Japan in 1979 were included in the UNSCEAR 1982 Report [U5]. Since that time, there has been one additional publication from Poland [J1], in which the effective dose equivalent per adult in 1976 was estimated to be about 1.7 mSv. Vano et al. [V4] reported an annual per caput dose equivalent of 0.8 mSv for Spain and a collective effective dose equivalent of 32,500 man Sv.

116. Reported annual effective dose equivalents for different examinations are shown in Table 29. Values generally range from 0.1 to 10 mSv.

117. From diagnostic x-ray examinations of the population in France in 1982, the collective effective dose equivalent was 86,000 man Sv, or an annual per caput effective dose equivalent of 1.6 mSv [B2]. In France the examination with the largest percentage contribution to the collective effective dose equivalent is intravenous urography, whereas in other countries, such as Japan and the United States, barium enemas and upper gastro-intestinal examinations play a larger role. Even from one highly developed country to another, the per caput effective dose equivalent may vary by up to a factor of 5. Some of these differences are certainly due to the number of examinations and to technical differences (beam quality, collimation etc.). In addition, however, Benedittini et al. [B10] indicate that there have been significant differences in the calculations of effective dose equivalents in specific organs by various authors. Benedittini et al. [B11] reported on the absorbed doses to patients from dental radiology in France in 1984. The collective effective dose equivalent was estimated as 2,000 man Sv and the per caput effective dose equivalent as 0.037 mSv. Although pantomographic examinations accounted for only 6% of the total number of examinations, their higher absorbed dose caused them to contribute 29% of the collective effective dose equivalent. Nikitin et al. [N8, N9] and Vorobyev et al. [V7] reported a per caput effective dose equivalent of about 1.5 mSv for the USSR. In 1981 the collective effective dose equivalent was estimated at about 400,000 man Sv.

118. It should be noted parenthetically that it is uncommon for a person to receive the "average" per caput effective dose equivalent calculated for the country in which he is living. Some authors have reported their findings as collective effective dose equivalents. Once this quantity has been derived for a given country, it can be divided by the population to obtain the per caput effective dose equivalent. Although in highly developed countries the frequency of diagnostic medical examinations may approach one examination per person per year, it is unlikely that more than 25-50% of people will actually have one examination in a given year. In less developed countries (health care levels II-IV), the situation is even more extreme, with perhaps only 1 person in 1,000 actually receiving an examination in a given year. Under such circumstances the person undergoing the examination would receive 1,000 times the average per caput effective dose equivalent or genetically significant dose, while 999 persons would receive no dose.

119. The energy imparted during a radiographic procedure has been suggested as an approach to estimating radiation risk. This method ignores the different sensitivities of individual body organs and calculates the energy imparted during a given procedure. It is attractive since it avoids the problem of calculating mean radiation doses to large organs [P8]. Bengtsson [B12] and Shrimpton [S13, S14] have already reported a reasonable correlation between the mean energy imparted and radiological risk. Over a range of two orders of magnitude in dose, mean energy imparted correlates with the quantity effective dose equivalent within factors of 2 or 3 [S14]. There is also a reasonable correlation between the energy imparted and the somatic effective dose. However, Huda [H15] has examined this approach with respect to computerized tomography scanning and concluded that it can lead to large errors in patient risk estimates.

120. Various other weighting factors can be utilized in an attempt to represent the impact of medical radiology in a more accurate fashion than can be done with the effective dose equivalent. As a first approximation, one could take the effective dose equivalent for the mean age of the population having a certain examination, multiply this by the specific rate for that examination and by absorbed dose. A second approximation would use the total age-specific weighted dose equivalent but would not use organ-specific risk factors. A third approximation could take sex into account as well by assuming a standard ratio of males to females having a specific examination. The greatest precision would be obtained by a fourth approximation which would apply age-specific and sex-specific weighting factors for each organ. Such weighting factors would be multiplied by the known dose to each organ for each examination type as well as each examination rate.

121. For a population of both sexes and a certain age distribution, the ICRP risk coefficient is normally utilized. To calculate the expected number of deleterious effects, n, after irradiation, Bengtsson et al. [B14] use the formula

$$n = RH_E N = RS \qquad (3)$$

where R is the risk coefficient, N is the number of individuals in the group and S is the collective effective dose equivalent. If age and sex are to be taken into consideration, one can have groups $i_{1-\infty}$. Additionally for each organ or tissue T, the risk in a given tissue per unit dose equivalent can be defined. One can then apply a series of risk factors for each tissue as a function of that tissue and age and sex of the individual. This would be expressed as r_{iT}. The probability of deleterious health effects in a given tissue would be expressed as the product of r_{iT} and H_E in that tissue. The probability of a deleterious effect in all tissues of a given individual in an age group would be given by

$$\bar{n}_i = \sum_T r_{iT} H_{Ti} \qquad (4)$$

Similarly, the expected number of deleterious effects for all persons in group i would be given as

$$\bar{n} = \sum_T r_{iT} H_{Ti} N_i \qquad (5)$$

where r_{iT} is the risk coefficient in tissue T, H_{Ti} is the dose equivalent in tissue T and N_i is the number of individuals in group i. In a similar fashion, the expected number of deleterious effects produced by examinations of type J in the total population would be given by

$$\bar{n}_J = \sum_i \sum_T r_{iT} H_{Ti} N_{iJ} \quad (6)$$

This could also be written as

$$n_{iJ} = S^*_J R \quad (7)$$

where S^*_J is the collective reference population dose for examination J. r_{iT}/R could also be termed "f factor" rather than weighting factor.

122. This appears to be quite precise in theory although, as was pointed out earlier, there are many uncertainties in the exposure factors, the absorbed dose to various organs and, particularly, the tissue-specific risk factors in the presence of disease. The risk factors used for this model were derived for continuous exposure of a working population and not for exposures received over a short time. Therefore, the risk derived is at best semi-quantitative. In a population with a very skewed age distribution, the use of collective effective dose equivalents may lead to an overestimation of detriment by a factor of between 1.5 and 3 [J8, M29]. The applicability of the concepts of effective dose equivalent and risk-weighted dose equivalent quantities to medical radiation have been examined by Drexler [D6], Ivanov [I8] and Kramer [K14].

G. WORLD-WIDE ESTIMATES OF DOSES FROM DIAGNOSTIC X-RAY EXAMINATIONS

123. In spite of the difficulties mentioned in the preceding section, an attempt is made here to derive per caput and collective effective dose equivalents from diagnostic x-ray examinations. The reported annual per caput doses for countries having various levels of health care are shown in Table 30. For health care level I countries, the effective dose equivalent and genetically significant dose agree reasonably well. In these countries the average per caput effective dose equivalent is approximately 1 mSv and the genetically significant dose is approximately 0.3 mSv. An analysis of diagnostic x-ray examination frequencies and contributions to absorbed doses in countries of health care level I is shown in Table 31. In previous UNSCEAR Reports it was been assumed that in less developed countries the collective effective dose equivalent would be lower, perhaps by an order of magnitude, due to the lesser frequency of radiological examinations. This would appear to be true according to literature on genetically significant dose in countries of health care levels II and III. However, most of these reports have not included fluoroscopy.

124. If the frequency of examinations is one tenth of that reported for countries of health care level I, and if fluoroscopy accounts for 30-70% of the total examinations, then the effective dose equivalent and genetically significant dose for countries of health care levels II, III and IV may in fact be comparable to those of level I countries. As was pointed out earlier, Zhang [Z4] estimated that in China chest fluoroscopy accounts for 70% of all examinations performed and that the associated absorbed dose is at least 15 times higher than that for chest radiography. It must be remembered in this connection that China accounts for approximately 20% of the world population.

125. Faced with these difficulties, the Committee has decided to calculate upper and lower limits for the effective dose equivalent and genetically significant dose for medical diagnostic radiography world-wide (Table 32). Calculations were performed by two methods. Method 1 is based on the frequency of examinations at various levels of development, and it assumes that the average doses for a given examination are comparable in countries of differing levels of health care. This method leads to a lower limit of approximately $1.8\ 10^6$ man Sv for the effective dose equivalent and of $0.5\ 10^6$ man Sv for the collective genetically significant dose equivalent. Method 2 assumes that although examinations are less frequent in countries of health care levels II, III and IV, the absorbed doses are 10 to 20 times higher than in level I countries primarily because of the extensive use of fluoroscopy and poorly calibrated machines. This method yields upper limits of $5\ 10^6$ man Sv for the collective effective dose equivalent and $1.5\ 10^6$ man Sv for the genetically significant collective dose.

126. Data on the effective dose equivalent and genetically significant dose from dental radiography are uneven and come only from countries of level of health care I. Since it appears that fluoroscopy is not widely used for dental purposes, one might assume that the per caput and collective doses for countries of health care levels II, III and IV are related predominantly to the frequency of examinations. The genetically significant dose from dental radiography in level I countries appears to be about 1/10,000 of that from medical diagnostic radiography. Estimations of the per caput and world wide effective dose equivalent and genetically significant dose for dental radiography are shown in Table 33. The annual collective effective dose equivalent world-wide from dental radiography is estimated to be about 17,000 man Sv with an annual genetically significant dose of 0.04 μSv.

H. OCCUPATIONAL EXPOSURE FROM DIAGNOSTIC RADIOGRAPHY

127. In the UNSCEAR 1982 Report [U5], occupational exposure was considered in a separate Annex. For this report, however, occupational exposures are considered along with the associated sources or practices. Evaluation of occupational exposures from medical radiation usage is complicated by the fact that the radiation usually comes from point sources close to the workers. Thus, the exposures are significantly non-uniform over the body because of the inverse square law as well as attenuation in the body. The effective dose equivalent cannot be easily inferred from one personal dosimeter on an individual, and this is especially true if the dosimeter is not in the

primary radiation fields striking the body. To make matters more complicated, the dosimeters are not always worn in the same position, although generally they are worn at the waist or neck. Often the recorded data do not indicate whether the worker wore the dosimeter inside or outside a protective lead apron. For these reasons it is very difficult to utilize average dose as measured by a dosimeter and to correct it to effective dose equivalent. Other difficulties are that minimum detectable levels vary as a function of dosimeter type and that the administrative decisions on whether to record the minimum detectable dose as zero or some other value are often arbitrary. Such decisions can have a major impact on estimation of the collective occupational dose, since in occupational exposure from medical radiation, a large percentage of workers receive doses at or near the minimum detectable level [D7]. The best that can be said is that for radiation qualities used for diagnostic x-ray procedures, the dosimeter usually measures a value that is 2-4 times higher than the effective dose equivalent [J12, M39], if a protective apron is not worn and if the exposure is relatively uniform. If a protective apron is worn and the personal dosimeter is placed on the outside (as is practice in the United States), then reported doses could be as much as 10 to 20 times higher than the effective dose equivalent.

128. As was discussed in the UNSCEAR 1982 Report [U5], another major complicating factor is accurate job classification of workers. While there is not usually a problem in differentiating between diagnostic radiologists and radiographers (technicians), the total number of workers in the field is sometimes expanded to include nurses, porters, aides, dark-room technicians etc., which can lead to erroneous calculations when determining mean annual individual dose. Many of these latter workers are not usually monitored since they usually receive very low doses. The number of unmonitored persons who occasionally perform x-ray examinations is unknown but is probably quite large, even in developed countries. The exact number of monitored workers engaged in performing diagnostic x-ray examinations varies widely from country to country. The range appears to be one monitored worker per 150-750 examinations annually [U4, U5, W17].

129. Data on occupational doses received from medical x-ray diagnosis are given in Table 34. In general, the average annual effective dose equivalents range from 0.1 to 3 mSv annually above natural background for radiologists and technologists. The dose distribution among the population of workers is markedly skewed, with a long tail of higher doses received by very few individuals. The highest exposures to radiologists, technologists and nurses occur during fluoroscopic procedures. Ameil et al. [A2, A3] and Tryhus et al. [T7] have reviewed the literature on absorbed dose to the radiologist during angiographic examinations. Doses were reported as follows: eyes, 0.01-0.5 mSv; thyroid, 0.03-0.5 mSv; waist (inside lead apron), 0.02 mSv; and hands, 0.05-1 mSv. Absorbed doses can be higher by a factor of 10 or more if the radiologist makes a manual injection (staying in the room during filming) or if there is an over-the-table x-ray tube. Gustafsson et al. [G10] have estimated that the effective dose equivalent to the radiologist performing angiography is about 0.03 mSv per examination.

130. It had been previously assumed by the Committee that lower estimates for occupational exposures would be appropriate for countries that had a lower frequency of examinations. This may not be the case, however, as is indicated by the recent data published by Wang [W10] and Zhang [Z9] for China, where average annual occupational doses for diagnostic medical workers are reported to be between 2.2 and 4.3 mSv. This figure is two to 10 times higher than the comparable figure in some countries of health care level I. There has, however, been remarkable progress in China (health care level II) in reducing the occupational doses over the past several decades. Wang [W10] reports that the average annual dose to diagnostic x-ray workers was 55.5 mSv before 1957, 8.7 mSv from 1957 to 1966 and 2.2 mSv from 1967 to 1980. The reason the doses still remain higher than in countries of health care level I is probably the extreme use of fluoroscopy and the lack of image intensification systems. The situation may be significantly worse in countries of levels of health care III and IV. Hussain [H22] reported on 311 x-ray installations in Bangladesh (level IV) and found that a majority of the installations had no shielded control booth, lead aprons or gloves. As was mentioned previously, almost one half of the machines had no functional collimation. In this Annex it will be conservatively assumed that the collective effective dose equivalent per million population is the same in countries of various levels of health care.

131. Information on occupational doses incurred as a result of dental radiography is very limited; however, the average annual doses are relatively low (Table 34), ranging from 0.02 mSv to 0.4 mSv annually in countries of health care level I. Dental practice generally contributes less than 1% to the collective effective dose equivalent from all occupational sources.

I. FUTURE TRENDS IN DIAGNOSTIC RADIOGRAPHY

132. It is instructive to postulate the future medical uses of radiation and the extent of their application and to examine potential areas of concern over the next 15 years. There is little doubt that, world-wide, the frequency and total number of procedures involving medical radiation will increase substantially [O4, U6]. There are three main reasons for this. First, there is the aging of the population, particularly in Europe. The Federal Republic of Germany, Switzerland, Italy and Greece are expected to have more than 20% of their populations over the age of 60 by the year 2000. The USSR and several other countries are expected to experience the same phenomenon, but to a lesser degree. As was indicated earlier, the older population accounts for a disproportionate number of medical diagnostic and radionuclide examinations as well as radiotherapy procedures.

133. The world's population is experiencing a marked contraction in the percentage of population between 0 and 30 years and a marked expansion in the percentage above the age of 30 [U6]. From 1950 to 1980, changes in the age distributions varied among the regions of the world. For example, the median age of the populations of Europe and the Soviet Union increased by about four years between 1950 and 1980 and that of Africa decreased by about 1.5 years. The median age for other regions decreased by about 2 years until the early 1970s and then began increasing, and it is now at the same level as it was in 1950. In contrast with past trends, the future is expected to be characterized by an aging of populations in all regions: the median age of the world population is expected to increase from 22.6 years in 1980 to 26.1 years in the year 2000 and to 30.8 years in the year 2025. The oldest populations in 2025 will be in Europe, East Asia, and Northern America. The youngest populations will be in Africa and Latin America, with median ages of 22.8 and 27.4 years, respectively.

134. Second, the total number of examinations will also undoubtedly increase simply as a result of population increase. The world's population was 2.5 billion in 1950 and 4.4 billion in 1980; it is projected to be 6.1 billion in 2000 and 8.2 billion in 2025. Even if the annual per caput effective dose equivalent and genetically significant dose remained the same, the collective doses would increase by over 60% from 1988 to 2025.

135. The population of the world had an annual growth rate of 1.7% in 1985. Throughout the nineteenth century and the first half of the twentieth century its annual growth rate was 0.5-0.8%. In the late 1960s, the world's population was growing at about 2% annually, and projections are that the growth rate will fall to 1.5% by the year 2000 and to 1% by 2025. It is important to note that while the rate of growth is declining, the annual increment to the world's population is increasing. The annual increment to the world's population in 1950 was 46 million, and in 1980 it was about 75 million. The annual increment is expected to peak at approximately 88 million near the end of the century and then decline somewhat by 2025. Although the global growth rate appears to be on the decline, there is marked difference between the more developed and less developed regions of the world. In the developed regions population is growing at an annual rate of 0.6%; in the less developed countries, it is growing at approximately 2%. As a consequence, the proportion of the world's population living in the less developed countries is expected to increase steadily.

136. Demographic trends also vary substantially from one part of the world to another. There is a rapid growth of the population in Africa, which is currently increasing at 3% per year and is expected to continue to increase at this rate until the end of the century. In 1950, the population of Africa accounted for about 8.7% of the total, but in 2025 it is expected to account for 19% of the total. Another rapidly growing area is Latin America, which has a growth rate of 2.5% per year or higher. Latin America's share of the total world population grew from 6.5% to 8.2% between 1950 and 1980, and by 2025 its share is expected to be 10.6%.

137. Third, the number and frequency of examinations will increase as a result of growing urbanization. At present, 41% of the population is classed as urban; in the year 2025, this percentage is expected to rise to 65%. As already discussed, urban populations have a much higher frequency of x-ray or radionuclide examinations than rural populations, the difference being an order of magnitude or more. If 50% of the world's population were urbanized by the year 2000, if the population aged as predicted and if the total population were 6 billion, the per caput doses and collective doses could be 50-100% higher than at present.

138. There are some countering factors in these projections. As the population ages, the assumed detriment would have less time to be expressed, and the use of an age-weighted dose equivalent would assume more importance. In simpler terms, although the number and frequency of examinations would increase, an older population would have less time to be at risk for the induction of stochastic effects. Moreover, in addition to depending on age, the genetically significant dose also depends on the reproduction rate. The gross reproduction rate in most developed countries is expected to remain fairly steady in the period 1980-2000, but it is decreasing in developing countries. This will be an additional factor to take into account when calculating the genetically significant dose. Overall, it can be assumed that the genetically significant dose will increase, but not as rapidly as the per caput or collective effective dose equivalent.

II. DIAGNOSTIC USE OF RADIOPHARMACEUTICALS

A. FREQUENCY AND TRENDS

139. Since the UNSCEAR 1982 Report, the Committee has obtained information from various countries on the number of in vivo diagnostic nuclear medicine examinations performed. This information is collected in Table 35. The frequency of all nuclear medicine examinations for countries of health care level I is in the range of two to 49 examinations per 1,000 population and for China (level II) it is 0.6 examinations per 1,000 population. Only in vivo diagnostic nuclear procedures are being considered in this chapter.

140. Malmstrom [M6, M7, M8, M9] reported statistics concerning nuclear medicine examinations in Sweden for the years 1979 through 1982. The total number ranged between 125,000 and 130,000 examinations annually (15 per 1,000 population).

141. The number of diagnostic nuclear medicine studies in the United Kingdom in 1982 was reported to be about 380,000, 84% of which were imaging

examinations. Bone scans were the most often performed procedure, although cardiac studies had increased 150-fold since 1973. Technetium-99m was the radionuclide used in 75% of the administrations, while iodine-131 was used in only 5%.

142. The number of diagnostic nuclear medicine procedures performed in the United States in different years is shown in Table 36. This Table documents the progressive increase in the frequency of diagnostic nuclear medicine procedures, both in absolute terms and per unit population. There was a rather sharp increase between 1970 and 1976, a plateau between 1976 and 1980 and another increase until 1982, with a sharp rise in cardiovascular and hepatobiliary imaging procedures. The only category in which a decline is evident is radionuclide brain scans. Similar trends have been reported in Denmark by Ennow [E2].

143. The percentage of each type of diagnostic nuclear medicine procedure may differ substantially from country to country. Table 37 shows that while thyroid imaging constitutes a large percentage of procedures in many Latin American countries, it constitutes only 9% of diagnostic radionuclide procedures in the United States, a variation that was also noticed and commented on in the UNSCEAR 1977 Report [U4]. A survey of radionuclide thyroid studies in the United States was reported by Parker et al. [P7], who identified substantial intra-country variation in methodology and radionuclide use. Technetium-99m pertechnetate was used for 54% of all thyroid scans and iodine-131 was used for only 9% of them. The rest of the thyroid scans were done with iodine-123. In summary, administered activity for a given examination varies by as much as a factor of 4 not only between countries but also within countries. The reasons for such variation are not known, but they may include training of the staff and, possibly, sensitivity of equipment.

144. The extrapolation procedure described in paragraph 10, which has been used to estimate world-wide medical diagnostic x-ray activity, can also be used to estimate nuclear medicine activity. A broad correlation exists between population per physician and annual nuclear medicine examinations per 1,000 population. There is also a strong relationship between population per physician and the population per scanner or gamma camera (Figure XI). The source terms and trends utilized to obtain averages for various levels of health care are shown in Tables 38 and 39. Estimates of annual examinations per 1,000 population for various levels of health care are shown in Table 40. It is estimated that there are approximately 24,000 gamma cameras or scanners world-wide and that approximately 24 million in vivo diagnostic radionuclide examinations are performed annually (Table 41). The number and type of nuclear medicine imaging devices in the United Kingdom have been reported by Wall [W8, W9]. The number of gamma cameras has markedly increased since 1974, while the number of rectilinear scanners has decreased.

B. AGE AND SEX DISTRIBUTION OF PATIENTS

145. For calculations of the genetically significant dose and related quantities it is necessary to know or assume the age and sex distribution of patients undergoing nuclear medicine procedures. Results of

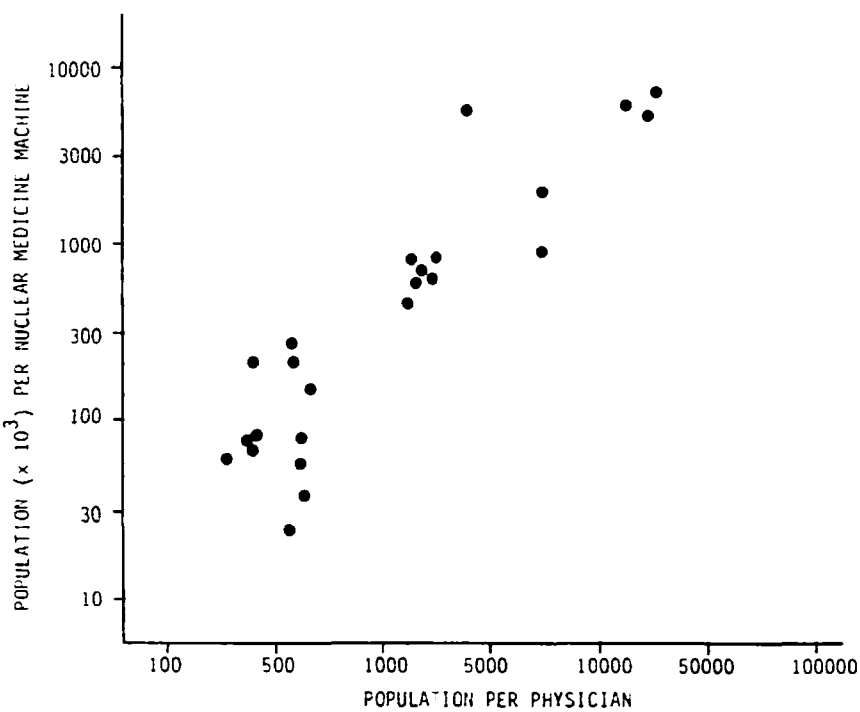

Figure XI. Correlation between population per physician and population per nuclear medicine machine in various countries.
[M28]

surveys performed in Poland in 1981 [S25] and the United States in 1980 [U10] are given in Table 42. About one third of the procedures in the United States are performed in persons over the age of 64 and approximately 70% of the procedures are performed in persons over the age of 45. This is true for most procedures, with the exception of thyroid and renal imaging procedures. In Poland the population receiving nuclear medicine examinations of all types is much younger.

C. IMPACT OF NEW TECHNOLOGIES

146. The impact of the new techniques has already been discussed briefly in section I.C, particularly the impact of computerized tomography on radionuclide brain scans and the possible impact of cardiovascular nuclear medicine procedures on invasive contrast studies. In view of the relatively high absorbed dose to the thyroid delivered in the course of examinations with iodine-131, many countries have begun to utilize either iodine-123 or technetium-99m pertechnetate. Unfortunately, iodine-123 is both difficult to obtain and expensive. The number of thyroid imaging procedures was rather stable in the United States over the period 1978-1982, and the number of thyroid ultrasound procedures performed is so far relatively small and does not appear to have significantly reduced the number of thyroid nuclear medicine procedures [M31]. Another area in which some replacement might be expected is radionuclide liver scans, which could be replaced by either hepatic ultrasound or hepatic computerized tomography. No data are available on the frequency of labelled monoclonal antibodies used predominantly for tumour detection.

D. ABSORBED DOSE

147. The range of administered activities for some types of examinations in different countries is shown in Table 43. As with absorbed dose in diagnostic radiology, the administered activity follows a skewed distribution. There are some differences between countries in the average activity used for certain examinations. For example, in the United States administered activity for a technetium-99m pertechnetate thyroid scan is about four times higher than in other developed countries. Kaul et al. [K2] and Johansson et al. [J5] published data concerning the dosimetry of unsealed incorporated radionuclides and discussed the mathematical-physical and metabolic dose models.

148. The effective dose equivalent in the USSR in 1980 from diagnostic radionuclide examinations has been reported by Knizhnikov et al. [K13]. He has indicated that the per caput value is 0.04 mSv per year. In the following years, in spite of the increasing number of radionuclide examinations, the average dose decreased to a per caput value of 0.03 mSv due to expanded use of short-lived radionuclides [V7]. The collective effective dose equivalent from all radionuclide examinations was estimated to be about 8,700 man Sv for 1981 [V7]. Table 44 shows that the collective effective dose equivalent for all diagnostic nuclear medicine procedures in the United States in 1982 has been estimated at 32,000 man Sv (0.14 mSv per caput) [M30]. A more in-depth analysis of the effect of age- and sex-specific weighting factors has been performed by Johansson [J8, J9], who concluded that detriment was about 40% of that calculated from the effective dose equivalent.

149. The annual per caput effective dose equivalent for most developed countries ranges between 0.03 and 0.14 mSv (Table 44). This is due mainly to the use of iodine-131 and technetium-99m. The percentages of collective effective dose equivalent attributable to different radionuclides are shown in Table 45. There are large differences between countries.

150. Radiation dose estimates for orally administered radionuclides used in upper gastro-intestinal disease have been calculated by Siegel et al. [S18]. Patient exposure and radiation risk in Bulgarian diagnostic nuclear medicine has been reported by Poppitz [P13]. One of the main sources of exposure in this particular case was the iodine-131 used in thyroid studies.

151. As a result of the widespread use of radiopharmaceuticals labelled with iodine-131 and technetium-99m there has been an increasing interest in assessing the radiation dose from breast milk following administration of such compounds to nursing mothers. Many authors have discussed the subject [B13, B17, C4, O1, O2, T6, V1, W24]. The nature of the radiopharmaceutical significantly affects breast secretion, with technetium pertechnetate having as much as 10% of the activity in breast secretions [A1]. In almost all instances, the secretion rate in milk 24 hours after injection is insignificant. An important exception to this arises in the case of iodine-125 fibrinogen. Ahlgren et al. [A1] recommend that when nursing mothers have received this radiopharmaceutical, breast feeding should be stopped for three weeks.

152. The method of estimating average practice in countries of various levels of health care can be used to estimate the world-wide annual per caput doses and collective dose from nuclear medicine. Reported effective dose equivalents and genetically significant doses for countries of health care level I are shown in Table 46 and are used as source terms. Since data are limited or lacking altogether for countries of health care levels II, III and IV, the values for those levels have been estimated according to the frequency of examinations. This may result in a slight underestimate, however, because it may be that longer-lived radionuclides are being used in less developed countries. For example, technetium-99m has a short physical half-life, making the dose of the pharmaceutical lower than that of a similar pharmaceutical labelled with iodine-131. At the same time, the short half-life makes it impractical to use technetium-99m in some less developed areas. The annual per caput doses and collective effective dose equivalent for health care levels I-IV are shown in Table 47. The annual collective effective dose equivalent is estimated to be 74,000 man Sv; the genetically significant collective dose is estimated world-wide to be approximately 15,000 man Sv.

E. OCCUPATIONAL EXPOSURE FROM DIAGNOSTIC NUCLEAR MEDICINE

153. Over the past decade there has been rapid expansion in the use of nuclear medicine and particularly in the use of the many technetium-99m labelled radiopharmaceuticals. Since these are administered predominantly by injection, there is a potential for relatively high doses to the hands of the workers. Generally, lead-shielded syringes are recommended; however, they are not always used. Direct handling of thin-walled plastic syringes can result in skin doses of 0.012-0.25 mSv per hour per MBq. Following injection, the patient represents another source of exposure to the technologist.

154. The limited data concerning occupational doses incurred in the practice of diagnostic nuclear medicine are presented in Table 48. Mean annual individual doses are 0.3-2.0 mSv. Nuclear medicine contributes approximately 2% to the collective dose from all occupational sources. The number of monitored workers in the field of nuclear medicine varies widely among countries. On the average, there are 100-300 examinations carried out annually for each monitored worker in developed countries [U5, W17]. Certainly, one nuclear medicine technologist can perform as many as 1,000 in vivo studies annually; however, the monitored workers also include physicians, chemists, physicists, pharmacists and, in some instances, clerks.

III. THERAPEUTIC USES OF RADIATION

A. FREQUENCY AND TRENDS

155. Data on the use of radiotherapy are often confusing because of imprecise definitions. With teletherapy, a treatment course may extend over several weeks and include many irradiations or treatments. By contrast, brachytherapy and the use of radiopharmaceuticals for therapy usually entail only one or two applications. For the purposes of this Annex, a teletherapy course or a brachytherapy application will be referred to as a procedure. Some authors refer only to the number of patients treated; the use of their data may cause the frequency of radiotherapy to be underestimated, since some patients are re-treated for recurrent tumours. Additional confusion arises in the matter of patient numbers, since some patients may receive treatment for more than one body area. Since the UNSCEAR 1982 Report [U5], some data have become available on the number of therapeutic radiology treatments in various countries. Table 49 shows there was a slight annual increase in therapeutic radiology treatments in Canada from 1978 to 1981. The annual frequency of treatments is approximately 26 per 1,000 of population. The estimated number of different types of cases treated by radiotherapy in some western hemisphere countries is shown in Table 50.

156. Hashizume et al. [H5, H6, H7] have reported on the status of external beam radiotherapy in Japan, where 77,000 patients were treated in 1978 with 1.78 10^6 irradiations (treatments). The average number of irradiations per treatment course was 21, with an average of 2.4 fields per patient. About 55% of the treatments were done with cobalt-60 units, 38% with high-energy x rays, 6% with high-energy electrons and 1% with conventional x-ray units. More than 50% of the patients were over the age of 45, about 4% were under the age of 14 years, and less than 1% of patients were treated for non-malignant disease. Marayuma et al. [M13] reported that in Japan in 1983 a total of 38,900 brachytherapy procedures were performed, 36,300 (93%) of which were in females.

157. In United States hospitals [K15] the number of new patients per radiotherapy unit was about 300 annually from 1973 through 1979 and the number of new patients per 1,000 population rose from 1.46 to 1.73 during the same period. Trends in equipment have been discussed in both the UNSCEAR 1977 Report [U4] and the UNSCEAR 1982 Report [U5]. Although orthovoltage units are still common in some Latin American countries and in parts of Europe, they have been almost completely replaced in the United States by cobalt-60 units and high-energy accelerators. Table 51 shows that in the United States from 1975 to 1980, there has been an increasing use of high-energy accelerators while the number of cobalt machines has remained approximately stable.

158. By estimating average practice in countries at various levels of health care, it is possible to obtain a rough estimate of radiation therapy activity world wide. The known radiation therapy experience by level of health care is shown in Table 52. For most countries of health care level I, approximately 2,400 brachytherapy and teletherapy procedures are performed annually per million population. In most countries approximately 200 new patients are treated annually per machine. Using these source terms, it is possible to estimate radiation therapy activity by level of health care (Table 53). The estimated number of procedures and machines by level of health care is shown in Table 54. By this estimation method it appears that there are approximately 5 million patients treated by radiotherapy annually and approximately 18,000 machines in use world-wide. The annual genetically significant dose from radiation therapy in countries of health care level I is approximately 0.015 mSv (Table 55); estimates for countries of health care levels II, III and IV are also shown in the Table.

159. The future of radiotherapy is somewhat difficult to predict. Certainly as the population ages, expands and becomes more urban, both the need for and the availability of radiation therapy will increase. In addition, the spectrum of diseases will change with time. One of the diseases in which there has already been such a change (and which often is treated with radiation therapy) is lung cancer. Since 1950, the lung cancer death rate has doubled, and in some instances tripled, in many European countries [O4].

160. At present, the Committee has no information on the age distribution of the population receiving radiotherapy in various countries nor does it have

information on the percentage of patients who may be long-term survivors. There are few new data since the UNSCEAR 1982 Report on the uses of radiation therapy for benign diseases. Probably the most common use is administration of sodium iodide-131 for hyperthyroidism. The effective dose equivalent depends on the percentage of iodine accumulated by the thyroid, but in cases of hyperthyroidism the effective dose equivalent usually exceeds 15 mSv per MBq [J4]. Wall [W9] has indicated that in the United Kingdom in 1982, treatments for thyrotoxicosis constituted 2.0% (7,600) of all nuclear medicine procedures and had a mean administered activity of 367 MBq and a range of 120 to 1,550 MBq. Similar experience has been reported from Denmark in 1985 [E2]. Therapy with unsealed radionuclides represented 1.4% of all nuclear medicine procedures. Therapy for thyrotoxicosis accounted for 88% of therapeutic procedures and thyroid cancer, 11%. The remaining 1% was for therapy with other radionuclides (such as ^{89}Sr for prostatic metastases and ^{131}I metaiodobenzylguanidine, ^{32}P and ^{90}Y for other tumour types). The number of therapeutic procedures for thyrotoxicosis in Denmark doubled between 1977 and 1985 [E2]. Whether this is also happening in other countries is unknown. In Sweden between 1979 and 1982 about 3,300 therapeutic nuclear medicine procedures were performed annually [M4, M5, M6, M7]. This accounted for about 2.5% of all nuclear medicine procedures. Due to the high absorbed doses, particularly to the thyroid, where non-stochastic effects predominate, therapeutic procedures are not usually included in assessment of annual collective dose from nuclear medicine. Patients with either thyrotoxicosis or thyroid carcinoma are predominantly young and female and have long survivals compared to other patients undergoing radiotherapy. Some recent data from Sudan [S36] indicate that 10% of all radiotherapy treatments are for benign diseases, with the majority of these (8.5% of the total) being for thyroid disease.

161. Extensive literature exists on endometrial carcinomas occurring 10 or more years after pelvic irradiation for squamous cell carcinoma of the cervix or carcinoma of the ovary, and after radiation-induced menopause [C13, F3, M5, R6, U4, U5]. The relative importance of such delayed effects depends not only on the availability of radiotherapy in various countries but also on the incidence of these tumours in the various countries. Because the incidence rates of cancers of various types vary from country to country, the relative percentage of secondary tumour types and the number of long-term survivors could also vary even if radiotherapy were equally available.

162. As the prospects for long-term survival improve following therapy and the possibility of secondary radiogenic tumours increases, there has been renewed interest in dose levels outside the useful radiotherapy beam. This was briefly discussed in the UNSCEAR 1982 Report [U5]. In patients treated for Hodgkin's disease, the relative risk of a second malignancy is 5.2 times that of the normal population. The mean actuarial 15-year risk reported recently by Tucker [T9] was 17.6%, of which 13.2% was due to solid tumours. The risk of leukaemia, although elevated after radiation therapy alone (relative risk 11 compared to the normal population), was much higher after either adjuvant chemotherapy (relative risk 117) or chemotherapy alone (relative risk 130). Such risks will continue to confound long-term follow-up studies to assess radiation risk in these patients.

B. ABSORBED DOSE

163. The dose delivered outside the useful radiation beam is determined mainly by scattered radiation in the patient and to a lesser extent by radiation scattered in air. For x-ray therapy units and linear accelerators levels of leakage radiation through the housing of the source contribute only 0.1-0.2% of the dose rate inside the useful beam. For neutron generators, however, the value may be 10 times as high [G6]. Results obtained by Kase et al. [K1] suggest that the machine collimators contribute 20-40% of the dose to patients outside the treatment field and that local shielding of organs from scattered radiation generated in the machine collimators could reduce the risk of carcinogenesis by as much as a factor of 2. Hudson et al. [H18] have also examined dose levels outside the beam, with particular emphasis on the provision of radiation therapy to a pregnant patient. They observed that the shielding blocks themselves may contribute to scattered radiation and that this is most likely to occur if the block is positioned immediately adjacent to the main beam. If the shielding block is moved away from the main beam, a dose reduction of some 30% is possible. Williams et al. [W14] and Petoussi et al. [P12] have published tables that include Monte Carlo calculations of dose to various organs for different fields in radiotherapy. These results are extremely useful since doses to organs and tissues outside the irradiated volumes are not often quoted in the literature. Vasilev et al. [V4] have reported that when patients are being treated for benign diseases, appropriate selection of x-ray potential can result in improved precision of dose delivered as well as reduction of dose to areas not being treated.

164. The annual genetically significant dose from brachytherapy in Japan in 1983 is estimated to have been 13 mSv and the per caput mean bone marrow dose, 0.31 mSv [M13]. The genetically significant dose from all radiotherapy in Japan in 1978 was 0.7 μSv and the per caput bone marrow dose was 1.5 mSv. This amounted to decreases of 93% and 26%, respectively, compared to 1971 [H6].

165. Of course, the fields or body areas treated by radiotherapy vary widely from country to country, so a world-wide assessment of risk from this practice would require data not only on the number of patients and treatments but also on the tissues or fields irradiated. As an example, cancer of the lung and breast are very common in the United States, although overall, cancer is a less significant cause of death than heart disease. In contrast, Olivares [O3] pointed out that cancer is the leading cause of death in Lima, Peru, with stomach cancer being most common in

males and cancer of the cervix being most common in females. While the Committee recognizes such major regional differences, it feels that a complete discussion of them is beyond the scope of this Annex. For this reason and others, discussed earlier, the Committee has not attempted to calculate an effective dose equivalent from the practice of radiotherapy.

166. The optimization of radiotherapy is intimately connected with the quality assurance and optimization of cancer control programmes. Zaharia [Z1] has indicated that in Latin America the most serious obstacle to cancer control is very late diagnosis and referral for treatment. This is predominantly due to lack of awareness of the early signs and symptoms of cancer. For example, in Peru, 92% of the patients presenting for treatment of cancer were in stages II to IV. This is in contrast to Sweden, where more than 40% of the patients presenting were in stage I and more than 80% were in stages I or II [W20]. The World Health Organization has examined most of the aspects related to optimizing radiotherapy. It indicates that the need for radiotherapy may not be uniform in all countries because the cancer sites in patients referred to radiotherapy institutes may have different rates of occurrence. In most industrialized countries, approximately one third of all cancer patients need radiotherapy either alone or combined with surgery. Approximately one half need surgery either alone or combined with other therapies. About one quarter of all patients either do not obtain, or are too advanced for, specific therapy. In less developed countries, the distribution of treatment needs will be different if the distribution of cancer sites is different. For example, when comparing North America with Latin America, researchers have found that the death rates from cancers of the breast (highest in North America), cervix, uterus and larynx (higher in Central and South America) often differ by a factor of 3 or more [P3].

167. The World Health Organization has also indicated that there is a difference in the age distribution of cancer patients from developed countries to developing countries, and the genetically significant dose will vary accordingly. For example, the average age of patients diagnosed with cancer was 55.7 years for Europeans, 45.9 years for Asians, and 35.9 years for Africans. Of the age group 10-40 years, Africans constituted 40%, Asians 31%, and Europeans only 12% [W20].

168. The World Health Organization maintains a quality control and dose comparison programme for clinical dosimetry [W20]. In an IAEA/WHO dose intercomparison programme, radiotherapy institutes received thermoluminescent capsules by mail and were requested to radiate them under varying circumstances. Similar co-operative programmes exist in Europe and in the United States. It is interesting to note that, even in highly industrialized countries, 15% of the institutions made dosimetry errors of more than 10%. Such errors may significantly affect the number of cases cured as well as the complication rates of the radiation therapy. Similar dosimetry intercomparison programmes have been reported on by Greene et al. [G7].

C. OCCUPATIONAL EXPOSURE FROM RADIATION THERAPY

169. Occupational exposures during the practice of radiotherapy come from several sources. In general, with the use of external beam radiotherapy the rooms are very well shielded and the attendant staff receive little exposure. An exception to this is doses incurred when using either neutron beams or electron accelerators operating above 10 MeV. The neutrons cause the activation of nearby materials, which then constitute a source of radioactivity and exposure to the staff even after the primary beam has been turned off. LaRiviere [L1] and Hoffman [H16] have examined this problem, and it appears that 75% of the staff dose is due to photoactivation products in the treatment head. The remainder is due to other activation products in the room; however, induced activity in the patient is not a significant source. The exact occupational dose equivalent received by a worker in this manner is a function of the workload. This is measured by personal dosimeters and appears to be 0.3-2.0 mSv, annually. Tatcher et al. [T1] have examined patients treated in a fast neutron therapy facility to determine how much the (n, 2n) reaction and production of carbon-11 and oxygen-15 in the patient added to the technologists' exposure. They concluded that patients were the source of less than 10% of the occupational exposure of the technologist.

170. A main source of occupational exposure from radiotherapy is brachytherapy. This often involves the insertion or surgical implantation of radioactive wires, needles or seeds. Pre-loaded surface applicators are also sometimes used. There has been a trend towards utilizing after-loading devices whenever possible to reduce occupational exposure. This involves the pre-positioning of an applicator or holder on or in the patient and then inserting the radioactive material at a later time. The occupational dose received from brachytherapy is also very dependent on whether the source insertion is manual or automated in some manner. Once the sources have been inserted the radiation exposure of persons around the patient must be considered. Since such exposure may be non-uniform, a comparison with doses incurred from other more uniform sources may be difficult. Annual occupational absorbed doses from brachytherapy usually range from 2 to 5 mSv [U4, U5].

171. Table 56 presents the limited data that are available concerning occupational doses from the entire practice of radiotherapy. Average annual individual exposures are 1-3 mSv, but, as pointed out, they can be higher in those individuals intimately involved with brachytherapy [H16]. The reported personal dosimeter values for radiation therapy workers are undoubtedly closer approximations of the effective dose equivalent than for diagnostic radiology workers. This is because in radiation therapy the energy of the incident radiation is higher and because protective aprons are not worn. The number of monitored workers in radiotherapy is difficult to assess. At present, data are available only from the United States, where it appears that there is one monitored person for each 25-50 procedures annually.

IV. SUMMARY

172. The present state of knowledge regarding the frequency of use of medical radiation and the associated absorbed dose is good for approximately 25% of the world's population. Data are fragmentary for another 25% of the population, and essentially no data exist for 50% of the population. For this reason, the Committee has developed an estimation procedure based on the good correlation that exists in most countries between population per physician and medical uses of radiation.

173. The main sources of uncertainty in the effective dose equivalent from medical diagnostic radiology are (a) the frequency of examinations and absorbed dose per examination, especially in the case of fluoroscopy; and (b) poorly calibrated or malfunctioning equipment. The effective dose equivalent from diagnostic medical examinations is far greater than that from dental or diagnostic nuclear medicine examinations.

174. The estimated world-wide per caput and collective effective dose equivalent and genetically significant dose from medical radiation are shown in Table 57. It would appear that the per caput annual effective dose equivalent is likely to be no lower than 0.4 mSv, but may be as high as 1.0 mSv. Similarly, the annual genetically significant dose may range from 0.1 to 0.3 mSv. The potential risk from medical radiation, if calculated from the effective dose equivalent for medical radiation, is probably an overestimate. This is particularly true in countries where the older portion of the population receives most of the medical irradiation.

175. The world-wide collective effective dose equivalent from medical radiation is estimated to be between $1.8\ 10^6$ and $5\ 10^6$ man Sv, and the genetically significant collective dose to be between $0.5\ 10^6$ and $1.5\ 10^6$ man Sv. Between 90% and 95% of both these values are attributable to medical diagnostic radiology. Dental radiography and nuclear medicine together contribute only 5-10% of the collective dose. In developed countries the contribution of diagnostic medical radiation to the collective effective dose equivalent is about 0.001 man Sv per examination.

176. There are many possibilities for dose reduction. In developed countries it may be possible to reduce the per caput effective dose equivalent to half its present value. In less developed countries the use of radiography rather than fluoroscopy, as well as the calibration and maintenance of equipment, would reduce the dose per examination, but the feasibility, cost and magnitude of these measures are unknown. One of the simplest and least expensive methods of dose reduction is appropriate collimation of the beam to conform only to the area of clinical interest. The genetically significant dose can be substantially reduced through the use of gonadal shielding, a practical, low-cost method. In spite of such measures, the collective effective dose equivalent may increase as x-ray examinations become more available in some countries, but this increase may in fact be medically appropriate. There have already been positive trends in dose reduction (including decreasing absorbed dose per examination as well as decreasing absorbed dose per patient without jeopardizing the desired clinical objective), particularly in well developed countries.

177. Occupational exposure from medical practices includes contributions from medical diagnostic radiology, dental radiography, nuclear medicine and radiation therapy. The sum of these for various countries is shown in Table 58. The average annual collective dose equivalent from medical occupational exposure is about 1 man Sv per million population. In both Canada and the United Kingdom, occupational exposure from medical practice represents about 10% of the collective dose equivalent from all occupational sources [U5]. In spite of the fact that the medical uses of radiation are increasing in most countries, limited trend data indicate that both annual individual doses and collective occupational doses are decreasing by 10-20% per decade. In the United States, for example, the number of occupationally exposed medical workers rose as follows: 300,000 in 1960; 400,000 in 1970; and 584,000 in 1980. During this time the annual collective dose equivalent decreased from 580 to 410 man Sv (Table 58). For developed countries the average occupational exposure is about 1 μSv per examination. The data also indicate that on average 150-750 examinations are carried out annually for each medical radiation worker.

178. The frequency and total usage of medical radiation is expected to increase over the next several decades as a result of (a) a general aging of the world's population; (b) an increase in the total number of people; and (c) a trend toward urbanization in the developing countries. By the year 2000, the collective dose will probably increase by 50% and by the year 2025 it may more than double.

179. Consideration of the following points would improve future assessments of exposures from the medical uses of radiation:

(a) collection of better data on the use of, and effective dose equivalents from, both mass miniature radiography and fluoroscopy in developing countries;

(b) continuing analysis of the aging and urbanization of population groups and its effect upon use of medical radiation;

(c) continued examination of the data for determining age- and sex-weighted dose equivalent values; and

(d) collection of data on the number of patients treated with radiotherapy and the proportion of long-term survivors in various countries.

Table 1

Annual frequency of common diagnostic x-ray examinations per 1000 population

Numbers in parentheses indicate per cent

Examination	Canada 1980 [C1]	China 1980 [Z5]	France 1981-1982 [B9, F1, M1]	Germany, Federal Rep. 1978 [U5]	Italy a/ 1983 [P1]
Skull and face	-	0.4 (0.1)	74.0 (8.9)	108.2 (12.4)	41.5 (5.6)
Cervical spine		1.0 (0.3)	23.5 (2.8)	-	26.7 (3.6)
Dorsal spine	113 a/ (11.1)	0.4 (0.1)	18.5 (2.2)	35.7 (4.1)	12.6 (1.7)
Dorsal lumbar sp.		-	33.6 (4.0)	21.9 (2.5)	-
Lumbosacral spine		3.5 (1.4)	13.3 (1.7)	-	36.4 (4.9)
Chest					
Radiographic	329.6 (32.4)	4.9 (1.9)	285.0 (34.1)	333.9 (39.6)	242.6 (32.6)
Photofluorogr.	-	-	-	-	80.9 (10.8)
Fluoroscopic	-	188.1 (72.6)	-	-	-
Mammography	2.7 (0.3)	-	4.8 (0.6)	27.9 (3.2)	6.7 (0.9)
Abdomen		0.4 (0.1)	29.6 (3.5)	4.1 (0.5)	22.3 (3.0)
GI tract and					
barium enema	132.3 (13.0)	9.2 (3.6)	35.3 (4.2)	67.8 (7.9)	46.0 (6.2)
Cholecystography		0.2 (0.1)	12.4 (1.6)	-	12.6 (1.7)
Urography	32.2 (3.2)	0.1 (0.1)	37.4 (4.4)	42.0 (4.9)	12.6 (1.7)
Hysterography		-	3.4 (0.4)	-	-
Pelvis and hip	-	1.5 (0.6)	62.2 (7.4)	49.0 (5.2)	40.1 (5.4)
Extremities	254.9 (25.1)	5.9 (2.3)	182.6 (21.9)	172.9 (20.2)	138.8 (18.6)
Computer tomography					
Head	-	-	-	-	5.2 (0.7)
Body	-	-	-	-	5.2 (0.7)
Others	151.3 (14.9)	43.4 (16.7)	19.4 (2.3)	-	14.1 (1.9)
Total (medical)	1016 (100)	259 (100)	835 (100) c/	863 (100) c/	744 (100)

Examination	Japan 1986 [M18]	Netherlands 1980 [B6]	Norway 1980-1983 [S3,S4]	Spain 1986 [V4]	Sweden 1979 [U5]
Skull and face	56.5 (4.8)	42.9 (6.6)	6.3 (1.0)	15 (3.1)	43.3 (8.8)
Cervical spine	41.2 (3.5)			)	
Dorsal spine	10.8 (0.9)	9.6 (1.5)	10.0 (1.6)	)	9.6 (1.9)
Dorsal lumbar sp.	52.5 (4.5)	-	0.6 (0.1)	) 97 (19.8)	17.8 (3.6)
Lumbosacral spine	14.4 (1.2)	30.8 (4.8)	27.0 (4.2)	)	2.6 (0.5)
Chest					
Radiographic	445.0 (38.1)	135.0 (20.8)	123.3 (19.2)	128 (26.0)	176.8 (35.8)
Photofluorogr.	-	123.1 (19.0)	84.4 (13.2)	-	-
Fluoroscopic	-	10.9 (1.7)	5.2 (0.8)	-	-
Mammography	1.3 (0.1)	8.4 (1.3)	2.5 (0.4)	14 (2.9)	6.4 (1.3)
Abdomen	82.9 (7.1)	12.7 (2.0)	8.0 (1.2)	45 (9.2)	11.7 (2.4)
GI tract and					
barium enema	174.9 (14.9)	19.7 (3.0)	33.1 (5.2)	40 (8.2)	32.6 (6.6)
Cholecystography	10.6 (0.9)	13.7 (2.1)	3.0 (0.1)	-	11.8 (2.4)
Urography	13.0 (1.1)	15.6 (2.4)	20.2 (3.2)	13 (2.6)	22.8 (4.6)
Hysterography	0.7 (0.1)	0.9 (0.1)	-	-	0.6 (0.1)
Pelvis and hip	12.7 (1.1)	33.6 (5.2)	46.4 (7.2)	15 (3.1)	36.4 (7.4)
Extremities	101.5 (8.7)	173.3 (26.7)	146.3 (22.8)	25 (5.1)	112.0 (22.7)
Computer tomography					
Head	-	-	7.4 (1.2)	7 (1.4)	1.2 (0.2)
Body	-	-	2.8 (0.4)		0.2 (0.1)
Others	154.0 (13.1)	18 (2.8)	115 (17.9)	91 (18.6)	8.0 (1.6)
Total (medical)	1172 (100) c/	648 (100)	641 (100)	490 (100) c/	494 (100) c/

Table 1, continued

Examination	Russian Federation 1980 [N9]	United Kingdom d/ 1983 [W6]	United States 1981 [M28]	Level I countries e/ (average)
Skull and face	52.2 (4.0)	39 (7.8)	36.1 (4.6)	50 (6)
Cervical spine	11.5 (0.9)	13 (2.7)	22.4 (2.8)	20 (2)
Dorsal spine	6.9 (0.5)	6 (1.2)	7.9 (1.0)	13 (2)
Dorsal lumbar sp.	17.4 (1.3)	-	-	25 (3)
Lumbosacral spine	4.6 (0.4)	24 (4.5)	56.8 (7.2)	25 (3)
Chest				
Radiographic	118.0 (9.0)	163 (32.9)	282.0 (35.7)	240 (30)
Photofluorogr.	525.0 (40.1)		-	25 (3)
Fluoroscopic	149.0 (11.4)		-	2 (1)
Mammography	-	5 (0.9)	5.7 (0.7)	7 (1)
Abdomen	-	21 (4.2)	34.8 (4.4)	55 (7)
GI tract and barium enema	181.0 (13.8)	20 (4.0)	55.1 (7.0)	70 (9)
Cholecystography	9.7 (0.7)	6 (1.3)	15.0 (1.9)	13 (2)
Urography	42.0 (3.2)	8 (1.7)	18.5 (2.3)	24 (3)
Hysterography	-	1	-	2 (1)
Pelvis and hip	10.0 (0.8)	22 (4.3)	20.7 (2.6)	38 (5)
Extremities	123.2 (9.4)	67 (13.4)	198.2 (25.1)	150 (19)
Computer tomography				
- Head	-	4 (0.8)	11.8 (1.5)	7 (1)
- Body	-	1	2.6 (0.3)	2 (1)
Others	58.0 (4.4)	89 (20.3)	22.5 (2.8)	32 (4)
Total (medical)	1308 (100)	488 (100)	790 (100)	800 (100)

a/ Northeast Italy only.
b/ Includes pelvis.
c/ Does not include mass screening.
d/ Great Britain only.
e/ Excluding China.

Table 2

Diagnostic x-ray examinations in the USSR [V7]

Examination	Number per 1000 population		Change
	1964	1981	
Fluoroscopy	439	220	-50%
Radiography	171	235	+37%
Photofluorography	183	503	+175%
Total	793	958	+21%

Table 3

Diagnostic x-ray machines in some western hemisphere countries

Country	1973 [P16]			1980 [I7]		
	Units	Population (thousands)	Units per 1000 population	Units	Population (thousands)	Units per 1000 population
Argentina	5170	24290	0.21	10000	27862	0.36
Chile	720	10309	0.07	1320	11104	0.12
Costa Rica a/	300	1896	0.16	124	2245	0.06
Ecuador	300	6726	0.04	345	8354	0.04
Mexico	3500	54300	0.06	3800	71910	0.05
United States	117151	209851	0.56	137000	227158	0.60

a/ Number of units reported in 1973 may include dental x-ray units.

Table 4

Diagnostic x-ray examinations by level of health care

Level of health care	Country	Annual examinations per 1000 population	Population per x-ray machine	Year	Reference
I	Argentina		2800	1978-1982	[I7]
	Canada	1016	3200	1980	[C1]
	Finland	958	-	1984	[T3]
	France	835	2700	1981-1982	[B9,M1,P11]
	Germany, Fed. Rep.	863	-	1978	[U5]
	Italy	744	3290	1983	[C11]
	Japan	1380	-	1986	[M18]
	Libyan Arab Jamal.	-	8000	1977	[C7]
	Netherlands	648	-	1980	[B6]
	Norway	641	-	1983	[S3, S4]
	Spain	490	4400	1986	[V4]
	Sweden	494	-	1979	[U5]
	United Kingdom	488	5000	1983	[W6, C11]
	United States	790	1800	1980	[M28]
	USSR	958	-	1981	[V7]
II	Bolivia	-	27000	1978-1982	[I7]
	Brazil	179	13400	1982	[C13]
	Chile	166	13000	1982	[C13]
	China	259	16400	1980	[Z4, Z5, S32]
	Colombia	211	14300	1978-1982	[I7]
	Costa Rica	270	19200	1981	[C13]
	Cuba	139	11000	1978-1982	[I7]
	Dominican Republic	20	80000	1981	[C13]
	Equador	36	-	1981	[C13]
	Islamic Rep.of Iran	180	-	1981	[S19]
	Mexico	70	15000	1980	[C13]
	Nicaragua	57	-	1981	[C13]
	Paraguay	-	41000	1978-1982	[I7]
	Peru	-	12000	1978-1982	[I7]
	Turkey	80	-	1978	[Y1]
	Uruguay	-	8800	1978-1982	[I7]
	Venezuela	-	10000	1978-1982	[I7]
III	Kenya	36	100000	1970	[C7]
	India	23	65000	1977	[C7]
	Liberia	80	70000	1977	[C7]
	Singapore	-	60000	1977	[C7]
	Sri Lanka	21	-	1979	[U5]
	Sudan	-	150000	1984	[S36]
	Thailand	34	-	1977	[C7]
IV	Ethiopia	-	300000	1977	[C7]
	Ghana	22	100000	1977	[C7]
	Côte d'Ivoire	40	190000	1977	[C7]
	Nigeria	25	90000	1977	[C7]

Table 5

Average diagnostic x-ray examinations by level of health care

Level of health care	Annual examinations per 1000 population	Population per x-ray machine
I	800	4000
II	150	20000
III	50	80000
IV	< 30	170000

Table 6

Estimated world-wide diagnostic x-ray examinations and machines in 1987

Numbers in parentheses indicate per cent of total

Level of health care	Population (millions)	Diagnostic x-ray machines (thousands)	Diagnostic examinations (millions)	Approximate examinations per machine
I	1300 (26)	330 (76)	1040 (75)	3000
II	1750 (35)	88 (20)	260 (19)	3000
III	1220 (24)	15 (3)	61 (4)	4000
IV	730 (15)	4 (1)	22 (2)	5500
Total	5000 (100)	440 (100)	1380 (100)	

Table 7

Diagnostic x-ray examinations in some Latin American countries in 1981
(per cent)
[17]

Country a/	Nervous system	Chest	Neck	Digestive tract	Uro-genital	Extremities	Other
Chile	5	40	2	18	5	30	-
Costa Rica	6	22	1	9	8	36	18
Dominican Republic	10	33	2	19	5	30	1
Ecuador	3	26	4	8	5	35	19
El Salvador	10	38	3	6	6	26	11
Mexico	-	40	6	12	5	28	9
St. Lucia	-	50	5	7	5	22	11

a/ All countries are of health care level II.

Table 8

Diagnostic x-ray examinations by level of health care
(per cent)

Examination	Level of health care a/			
	I	II	III	IV
Head and neck	8	9	8	
Chest	33	36	50	
Abdomen and digestive tract and gallbladder	18	13	6	
Urogenital	4	6	4	
Extremities	19	27	23	
Other	18	9	9	
Sample size (number of countries)	(12)	(7)	(2)	(0)

a/ Information for health care level IV not available.

Table 9

Estimated percent of urban and rural populations receiving diagnostic x-ray examinations in some Latin American countries in 1981 [I7]

Country	Urban	Rural
Chile	15	2
Costa Rica	5	2
Dominican Republic	15	10
El Salvador	9	-
Mexico	20	2
St. Lucia	6	3

Table 10

Annual frequency of diagnostic x-ray examinations per 1000 population a/

Country	1955-59	1964	1969-70	1974-76	1977-79	1980-83	Reference
Canada					987	1016	C1
China b/				146	208	259	S22,Z4,Z5
France	150					835	B9, B10
Germany, Fed.Rep.					863		U5
Japan	259		641	729	1328		U4, U5
Netherlands			810			648	U4, B6
Norway	609					641	S4, S5
Sweden	430	650			494 c/		U4
Turkey					80 d/		Y1
United Kingdom	300	310	400		440	496	U4,K5,W6
USSR		793	911	1094		1272	V7, N9
USSR e/			1065	1192		1339	K13, K19
United States		637	717			790	M28

a/ Includes mass screening and fluoroscopy unless otherwise indicated.
b/ Refers to Shangdong Province; frequency for Beijing (1983): 671 [Z11].
c/ Does not include mass screening.
d/ Does not include fluoroscopy.
e/ Russian Federation.

Table 11

Dental radiography, 1975-1987
[B8, B11, C11, H20, M10, M14, P11, U4, U5, W6]

Level of health care	Country	Films per 1000 population	Procedures per 1000 population	Population per machine a/
I	Argentina			3900
	France	540	-	1800
	Italy	118	70	
	Japan		800	1700
	Poland		44	
	Sweden	1800		
	United Kingdom	255	165	
	United States	1650	456	1000
	Rounded average		250	2500
II b/	Chile		3.9	21000
	Costa Rica			6500
	Ecuador			9400
	Mexico			80000
III	Sri Lanka		0.8	

a/ Data provided are difficult to evaluate for number of dental machines since standard radiographic machines are often used.
b/ Data for health care level II countries from [I7].

Table 12

Estimated world-wide dental radiography, 1980

Level of health care	Films per 1000 population	Procedures per 1000 population	Population per machine	Estimated total procedures (millions)
I	1000	250	2500	330
II	-	4 (50) a/	20000	7 (87)
III	-	0.8 (16)	-	1 (19)
IV	-	(8)	-	0.3 (6)
Total				340 (440)

a/ Numbers in parentheses refer to estimates for levels II-IV based on diagnostic radiologic activity. These estimates may be high by an order of magnitude.

Table 13

Age and sex distribution of diagnostic x-ray examinations
(per cent)

Examination	Age and sex	Norway 1983 [S3, S4]	United Kingdom 1983 [W6]	United States 1980 [U10]
Skull and face	< 15 male	10.0	10.9	11.2
	female	7.2	6.8	7.1
	both	17.2	17.7	18.3
	15-29 male	12.2	13.1	20.1
	female	8.6	8.6	14.8
	both	20.8	21.7	34.9
	30-44 male	10.0	9.8	8.7
	female	9.4	9.3	8.9
	both	19.4	19.1	17.6
	45-64 male	13.6	11.5	7.3
	female	12.6	13.0	9.0
	both	26.2	24.5	16.3
	> 64 male	7.4	6.8	5.1
	female	8.8	10.2	7.8
	both	16.2	17.0	12.9
	All ages male	53.2	52.1	52.4
	female	46.8	47.9	47.6
	both	100.0	100.0	100.0

Examination	Age and sex	China a/ 1980 [Z4, Z5]	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Chest	< 15 male	10.4	5.8	7.3	3.7	4.6
	female	6.5	4.0	6.0	2.4	3.4
	both	16.9	9.8	13.3	6.1	8.0
	15-29 male	14.1	4.7	9.2	6.9	7.4
	female	13.3	4.3	7.7	7.0	7.5
	both	27.4	9.0	16.9	13.9	14.9
	30-44 male	12.4	7.1	9.6	8.5	6.7
	female	12.4	7.5	9.1	8.1	8.1
	both	24.8	14.6	18.7	16.6	14.8
	45-64 male	14.1	18.7		16.4	14.6
	female	9.9	17.1		13.4	14.4
	both	24.0	35.8	26.0	29.8	29.0
	> 64 male	4.0	15.9	25.1	16.4	15.6
	female	2.9	14.9	51.1	16.5	17.7
	both	6.9	30.8		32.9	33.3
	All ages male	55.0	52.2	52.1	52.6	49.9
	female	45.0	47.8	47.9	47.4	51.1
	both	100.0	100.0	100.0	100.0	100.0

a/ Chest fluoroscopy, which constitutes 95% of all chest examinations in China.

Table 13, continued

Examination	Age and sex	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Abdomen	< 15 male	4.5	4.0	4.7	4.5
	female	2.9	2.9	4.6	3.8
	both	4.7	6.9	9.3	8.3
	15-29 male	5.4	9.3	8.4	8.2
	female	6.2	13.0	10.9	10.2
	both	11.6	22.3	19.3	18.4
	30-44 male	7.1	10.9	6.3	7.9
	female	7.0	13.4	8.8	8.3
	both	14.1	24.3	15.1	16.2
	45-64 male	17.2		12.0	14.2
	female	11.9		12.0	14.2
	both	29.1	24.3	24.0	26.5
	> 64 male	19.6	22.2	14.2	13.6
	female	21.2	46.5	20.1	17.0
	both	40.8		34.3	30.6
	All ages male	50.8	48.5	45.6	48.4
	female	49.2	51.5	56.4	51.6
	both	100.0	100.0	100.0	100.0

Examination	Age and sex	China 1980 [Z4, Z5]	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Upper GI (barium meal)	< 15 male	2.1	3.2	0.9	0.9	1.4
	female	0.9	2.4	0.7	0.4	1.5
	both	3.0	5.6	1.6	1.3	2.9
	15-29 male	13.7	8.4	9.8	6.6	6.0
	female	9.2	5.4	7.5	4.9	9.2
	both	22.9	13.8	17.3	11.5	15.2
	30-44 male	15.8	10.2	14.0	13.4	8.4
	female	9.9	8.6	12.4	12.5	11.9
	both	25.7	18.8	26.4	25.9	20.3
	45-64 male	21.7	18.8		18.2	14.3
	female	15.8	16.2		15.0	18.7
	both	37.5	35.0	29.3	23.2	33.0
	> 64 male	6.7	12.2	25.4	9.8	11.3
	female	4.2	14.4	54.7	18.3	17.2
	both	10.9	26.6		28.1	28.6
	All ages male	60.0	52.8	54.0	48.9	41.5
	female	40.0	47.2	46.0	51.1	58.5
	both	100.0	100.0	100.0	100.0	100.0

Examination	Age and sex	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Barium enema	< 15 male	0.2	4.5	0.4	1.1
	female	0.2	5.9	< 0.1	1.1
	both	0.4	10.4	0.4	2.2
	15-29 male	5.0	4.0	3.5	3.5
	female	6.6	6.4	5.8	6.9
	both	11.6	10.4	9.3	10.4
	30-44 male	8.2	9.5	4.9	5.5
	female	13.2	7.3	11.3	10.8
	both	21.4	16.8	16.2	16.3
	45-64 male	15.2		14.8	13.6
	female	23.6		17.2	21.2
	both	38.8	28.3	32.0	34.8
	> 64 male	10.7	34.1	15.2	14.1
	female	17.1	62.4	26.9	22.2
	both	27.8		32.1	36.3
	All ages male	39.3	46.3	38.8	37.8
	female	60.7	53.7	61.2	62.2
	both	100.0	100.0	100.0	100.0

Table 13, continued

Examination	Age and sex	Norway 1983 [S3, S4]	United Kingdom 1983 [W6]	United States 1980 [U10]
Biliary tract	< 15 male	< 0.1	< 0.1	0.3
	female	0.2	1.1	0.6
	both	0.2	1.1	0.9
	15-29 male	2.8	1.5	4.5
	female	7.0	9.1	12.1
	both	9.8	10.6	16.6
	30-44 male	7.2	8.2	8.1
	female	05.9	10.0	15.9
	both	23.1	18.2	24.0
	45-64 male	13.8	11.8	14.0
	female	27.0	33.2	20.1
	both	40.8	45.0	34.2
	> 64 male	8.3	8.5	9.6
	female	17.8	12.6	14.7
	both	26.1	21.1	24.3
	All ages male	32.1	30.0	36.6
	female	67.9	70.0	63.4
	both	100.0	100.0	100.0

Examination	Age and sex	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Urogram	< 15 male	4.4	6.6	6.5	1.8
	female	9.9	10.4	3.9	2.7
	both	14.3	17.0	10.4	4.5
	15-29 male	5.4	7.9	8.1	7.1
	female	7.5	9.9	8.5	9.7
	both	12.9	17.8	16.6	16.8
	30-44 male	8.0	11.0	9.9	9.0
	female	9.3	10.5	5.3	11.6
	both	17.3	21.5	15.2	20.6
	45-64 male	16.6		24.2	15.6
	female	13.5		7.7	15.5
	both	29.1	25.8	31.9	31.1
	> 64 male	17.4	17.9	19.1	15.0
	female	7.8	43.7	6.8	12.0
	both	25.2		25.9	27.0
	All ages male	52.0	51.3	67.8	48.5
	female	48.0	48.7	32.2	51.5
	both	100.0	100.0	100.0	100.0

Examination	Age and sex	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Lumbosacral spine	< 15 male	2.0	2.4	3.6	1.1
	female	2.3	2.7	1.9	1.1
	both	4.3	5.1	5.5	2.2
	15-29 male	13.0	6.0	5.8	16.2
	female	8.6	5.1	8.3	9.5
	both	21.6	11.1	14.1	25.7
	30-44 male	13.0	11.5	13.2	13.6
	female	12.2	14.4	13.7	11.1
	both	25.2	25.9	26.9	24.7
	45-64 male	15.2		12.3	12.5
	female	16.9		14.4	15.1
	both	32.1	25.4	26.7	27.6
	> 64 male	6.6	32.5	11.0	6.6
	female	10.2	57.9	15.8	13.2
	both	16.8		26.8	19.8
	All ages male	49.8	45.3	54.1	50.0
	female	50.2	54.7	54.1	50.0
	both	100.0	100.0	100.0	100.0

Table 13, continued

Examination	Age and sex	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
Pelvis and hip	< 15 male	9.2	18.6	5.6	4.3
	female	10.8	27.1	7.5	3.7
	both	20.0	45.7	13.1	8.0
	15-29 male	4.2	4.7	6.1	8.2
	female	4.2	3.9	6.6	5.6
	both	8.4	8.6	12.7	13.8
	30-44 male	4.4	5.3	8.2	5.6
	female	8.0	6.0	6.5	5.1
	both	12.4	11.3	14.7	10.7
	45-64 male	10.8		10.4	10.2
	female	20.0		13.6	13.1
	both	30.8	14.1	24.0	23.3
	> 64 male	8.4	20.3	9.9	12.6
	female	20.0	34.4	25.6	31.6
	both	28.4		35.5	44.2
	All ages male	37.0	42.7	40.2	40.9
	female	63.0	57.3	59.8	59.1
	both	100.0	100.0	100.0	100.0

Examination	Age and sex	Isl.Rep. of Iran 1980 [S19]	Norway 1983 [S3, S4]	Poland 1978 [N5]	United Kingdom 1983 [W6]	United States 1980 [U10]
All diagnostic examinations	< 15 male	9.0	-		6.7	5.5
	female	6.7	-		4.6	4.2
	both	15.7	-		11.3	9.7
	15-29 male	21.6	-		10.7	12.1
	female	10.7	-		8.3	9.8
	both	32.3	-		19.0	21.9
	30-44 male	16.0	-		9.9	8.1
	female	11.0	-		8.5	9.1
	both	27.0	-		18.4	17.2
	45-64 male	12.4	-		13.9	12.1
	female	7.5	-		13.2	14.1
	both	19.9	-		27.1	26.2
	> 64 male	3.2	-		7.6	10.5
	female	1.4	-		15.6	14.5
	both	4.6	-		23.2	25.0
	All ages male	63.0	50.9	48.8	48.3	
	female	37.0	49.1	51.2	51.7	
	both	100.0	100.0	100.0	100.0	

Table 14

Cardiac imaging procedures in the United States
(thousands)
[N1]

Examination	1972	1973	1980
Angiography		200	504
Coronary and left ventriculography		200	504
Echocardiography	0		1400
Radionuclide blood pool	11	25	580
Radionuclide infarct scan	2		580
Radionuclide scan perfusion/ischemia thallium	0		580

Table 15

Head x-ray and radionuclide examinations in the United States
(thousands)
[E6]

Examination	1964	1970	1972	1973	1978	1980
Head CT			0	<10		1600
Skull	2500	3600				3700
Pneumo-encephalogram		48				2
Arteriogram	121					315
Radionuclide brain scan			1250		1546	867
Radionuclide cisternogram			12			16

Table 16

Mammography examinations in the United States
(thousands)
[N1]

	1964	1970	1980
Number in hospitals	53	199	1000
Number in surgeries	13	47	260
Total	66	246	1260
Per 1000 female population	0.6	2.4	11

Table 17

Skin dose in the primary beam per film a/
(mSv)

Examination (projection) b/	Canada [C2]	Italy [I1]	Poland [J1]	United Kingdom [H2,S16]	United States [U9]
Skull (LAT)	2.1 (0.8- 8.9)	4.3 (2.6-19.6)	- -	2.3 -	2.3 (0.1-36.1)
Chest (P/A)					
Radiographic	0.17 (0.04-3.15)	0.69 (0.07-16.8)	2.0	0.22 (0.10-0.90)	0.21 (0.03-7.1)
Photofluorographic	-		7.7	1.2	-
Abdomen (A/P)	6.2 (0.6-39.9)	-	27.0	8.4	6.2 (0.4-86.4)
Retrograde pyelogram (A/P)	6.6 (3.7-74.5)	13.1 (2.9-39.8)	-	-	6.8 (0.8-61.9)
Cervical spine (A/P)	1.8 (0.5-4.4)	-	16.8	-	2.2 (0.1-33.6)
Thoracic spine (A/P)	3.2 (1.9-20.3)	-	-	6.2	6.8 (0.8-42.6)
Lumbar spine					
(A/P)	5.3 (0.7-36.1)	12.3 (12.4-36.5)	27.7	9.2	7.5 (0.4-98.0)
(LAT)	-	-	-	22.8	-

a/ Values expressed as median, numbers in parentheses refer to range when available.

b/ A/P and P/A and LAT refer to beam entrance and exit on the body. For example, on a P/A chest radiograph the beam is incident upon the posterior thorax and exits on the anterior thorax.

Table 18

Mean number of radiographs and fluoroscopy screening time by examination in France, 1982
[M1]

Examination	Mean number of films	Fluoroscopy screening time (s) a/	Examinations involving fluoroscopy (%)
Cervical spine	3.7	53	37
Thoracic spine	43.	34	33
Lumbar spine	4.8	47	41
Sacro-lumbar spine	4.8	82	56
Pelvis, hip	2.2	26	25
Abdomen	2.4	34	29
IV urography	10.7	83	52
Hysterography	4.9	96	73
Cholecystography	5.7	73	67
Skull	3.2	29	24
Barium enema	9.5	187	76
Barium meal	9.5	267	81
Thorax	1.5	17	10
Cerebral angiography	46	482	40
Thoracic angiography	24.2	455	86
Abdominal angiography	37.7	302	78
Inferior limbs angiography	14.3	78	60
Phlebography	10.1	182	15
Obstetrical abdomen	3.4	53	57
Pyelography	5.2	75	54

a/ For static examinations, such as lumbar spine, cervical spine, abdomen etc., fluoroscopy is mostly used for centring the patient.

Table 19

Radiation doses to neonates receiving diagnostic examinations in the United Kingdom
(sample size 85 infants)
[R5]

Gestation (weeks)	Mean birth weight (kg)	Mean number of films per infant	Mean number of CT scans per infant	Mean marrow dose (mSv)
26-27	0.83	6.7	0.5	2.47
28-29	1.15	10.5	0.3	1.65
30-31	1.49	11.3	0.6	2.17
32-33	1.80	3.6	0.3	1.05
34-35	2.23	2.8	0	0.05
36-37	2.60	4.1	0	0.08
38-39	2.38	3.3	0	0.06
40-41	3.39	2.4	0	0.04
42	3.42	1.5	0	0.03

Table 20

Organ doses from computerized tomography scans in Japan [N10]

Organ	Mean absorbed dose (mSv)		
	Cranial	Upper abdominal	Lower abdominal
Ovary	0.0043	0.18	9.50
Testes	0.004	0.17	0.175
Bone marrow	1.41	1.74	2.60
Brain	25.0	0.06	0.06
Sublingual gland	1.45	0.45	0.04
Thyroid	9.60	0.54	0.043
Breast	0.15	14.8	0.21
Stomach	0.04	7.60	0.43
Lung	0.18	6.80	1.13
Liver	0.04	5.80	0.38
Upper large intestine	0.006	0.26	12.0
Lower large intestine	0.006	0.26	12.0
Rectum	0.005	0.16	9.20
Eye (right)	28.0	0.15	0.03

Table 21

Organ doses from dental radiography in the United Kingdom [W3]

Organ	Mean dose equivalent per examination (mSv)		
	Intra-oral (2 films)	Extra-oral (2 films)	Pantomography (1 film)
Gonads	0.002	0.001	< 0.005
Breast	0.01	0.005	0.01
Bone marrow	0.025	0.02	0.05
Lungs	0.002	0.001	0.01
Thyroid	0.01	0.01	0.07
Bone surface	0.12	0.10	0.20
Brain	0.10	0.03	0.50
Salivary glands	0.03	0.70	1.1
Sinuses	0.50	0.05	0.20

Table 22

Collective effective dose equivalent from diagnostic x-ray examinations in France, 1982
[M2]

Examination	Collective effective dose equivalent (man Sv)	Accounted for by fluoroscopy (%)
Cervical spine	1680	18 a/
Thoracic spine	2100	16.5 a/
Lumbar spine	8580	13 a/
Sacro-lumbar spine	3400	7 a/
Pelvis, hip	5350	3 a/
Abdomen	4120	6.5 a/
IV urography	20580	11.5 a/
Hysterography	810	17
Cholecystography	4860	34.5
Skull	4990	10 a/
Barium enema	8210	21.5
Barium meal	7460	31.5
Thorax	4110	3 a/
Cerebral angiography	1780	15
Thoracic angiography	680	70.5
Abdominal angiography	5590	34
Inferior limbs angiography	280	15
Phlebography	940	37
Obstetrical abdomen	930	8 a/
Pyelography	370	24

a/ Examinations in which fluoroscopy is only used for positioning the patient prior to film radiography.

Table 23

Procedures to reduce collective dose equivalent in diagnostic x-ray examinations

Area	Procedure	Entrance dose reduction factor	Reference
All types	Elimination of medically unnecessary procedures	1.2	[C9]
	Introduction of quality assurance programme (general) a/	2.0	[C9]
Radiography	Decrease in rejected films through QA programme	1.1	[G1, P15]
	Increase of peak kilovoltage	1.5	[W13]
	Beam collimation	1.0-3.0	[J7, M35]
	Use of rare earth screens	2-4	[K21, N7, S8, W2]
	Increase of filtration	1.7	[K21,M34,W13]
	Rare earth filtration	2-4	[T10]
	Change from photofluorography to chest radiography	4-10	[J1, M38, N6]
	Use of carbon fibre materials	2	[H17]
	Replacement of CaWO4 screens with spot film technique	4	[K21]
	Entrance exposure guidelines	1.5	[L2]
	Gonadal shielding	2-10 b/	[P14]
Pelvimetry	Use of CT topogran	5-10	[S27]
Fluoroscopy	Acoustic signal related to dose rate	1.3	[A4]
	Use of 105 mm camera	4-5	[R9]
	Radiologist technique	2-10	[R9]
	Variable aperature iris on TV camera	3	[L3]
	Change from chest fluoroscopy to radiography	20	[S32]
	High and low dose switching	1.5	[L3]
Digital radiography	Decrease in contrast resolution	2-3	[R4]
	Use of pulsed system	2	[R4]
Computed tomography (head)	Gantry angulation to exclude eye from primary beam	2-4 c/	[I5]
Mammography	Intensifying screens	2-5	[N3, S17]
	Optimal compression	1.3-1.5	[N3]
	Filtration	3	[H1]

a/ The role of proper training in radiation protection is extremely important. Dose reduction factors in this regard may be large, however they are difficult to quantify.
b/ To gonads.
c/ To eye.

Table 24

Genetically significant dose in France in 1982
(mSv)
[B10]

Age (years)	Fluoroscopy		Radiography		Total		
	Males	Females	Males	Females	Males	Females	Total
< 1	0.002	0.001	0.017	0.028	0.019	0.029	0.048
1- 4	0.001	0.000	0.006	0.019	0.007	0.020	0.027
5- 9	0.001	0.002	0.008	0.016	0.009	0.019	0.028
10-14	0.002	0.001	0.009	0.040	0.011	0.041	0.052
15-19	0.003	0.002	0.008	0.032	0.011	0.034	0.045
20-24	0.003	0.003	0.010	0.027	0.013	0.030	0.043
25-29	0.002	0.002	0.007	0.019	0.009	0.022	0.031
30-34	0.002	0.001	0.004	0.007	0.006	0.008	0.014
35-39	0.001	0.000	0.002	0.002	0.003	0.003	0.006
40-44	0.000	0.000	0.001	0.000	0.001	0.000	0.001
> 45	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Total	0.017	0.013	0.072	0.190	0.089	0.202	0.295

Table 25

Average dose equivalent in the female gonads per examination
(mSv)

Examination	France a/ [M2]	Germany Fed.Rep. [H15]	Great Britain [W4]	Isl.Rep. of Iran [S19]	Italy [P1]
Cervical spine	0.01				
Dorsal spine	0.36				
Dorsolumbar spine	1.07	2.0	3.8		
Lumbosacral spine	1.28		8.39		3.85
Pelvis, hip		0.15 b/	0.75 b/	1.92	0.51 b/
	1.40	0.9	1.64 c/	1.31	23.8 c/
Abdomen, without preparation	1.36	1.5	0.84	1.56	2.33
IV urography	6.71	5.7	3.58	5.28	5.50
Hysterography	3.83			4.27	
Cholecystography	1.40			3.17	
Skull	0.05				
Barium enema	6.25		16.0	11.7	15.40
GI tract	1.70		3.60	0.73	1.51
Thorax	0.04			0.02	

Examination	Japan [H4]	Poland [J1]	Switzerland a/ [P14]	Turkey [Y1]	USSR [V7]	United States [R8]
Cervical spine					0.02	
Dorsal spine					0.13	
Dorsolumbar spine	1.62	1.6				4.05
Lumbosacral spine					2.8	6.4
Pelvis, hip	1.12 b/		1.2	11.0 b/		
	1.44 c/	1.0		1.58	15.0 c/	0.78
Abdomen, without preparation	0.72		0.6	1.40		2.72
IV urography	1.98	6.6		4.22	3.0	6.36
Hysterography	1.1			6.95		
Cholecystography	0.3	2.5		0.90		0.06
Skull	0.0007	0.01			0.0003	0.0001
Barium enema	9.7		10.20	38.0 d/	7.87	
GI tract	0.81	4.6		3.14	0.17 d/	0.45
Thorax	0.0005	0.01			0.02	0.0006

a/ Per film.
b/ Hip and upper femur.
c/ Pelvis.
d/ Includes fluoroscopy.

Table 26

Average dose equivalent in the male gonads per examination
(mSv)

Examination	France a/ [M2]	Germany Fed.Rep. [H15]	Great Britain [W4]	Isl.Rep. of Iran [S19]	Italy [P1]
Cervical spine	0.02			0.01	
Dorsal spine	0.15				
Dorsolumbar spine	0.60				
Lumbosacral spine	0.86	0.05		1.28	0.06
Pelvis, hip	1.48	0.1 b/	8.4 b/	1.02	
		2.5			
		1.7 c/	3.53		
Abdomen, without preparation	0.61	0.12	1.64	0.34	0.43
IV urography	2.46	0.5	4.3	1.10	2.27
Hysterography					
Cholecystography	0.93			0.11	
Skull	0.02			0.01	
Barium enema	3.70	0.58	3.4	1.30	2.33
GI tract	0.95		0.3	0.02	
Thorax	0.04			0.01	

Examination	Japan [H4]	Poland [J1]	Switzerland a/ [P14]	Turkey [Y1]	USSR [V7]	United States [R8]
Cervical spine					0.02	
Dorsal spine	0.004		0.004		0.10	
Dorsolumbar spine						0.07
Lumbosacral spine	0.09	3.40		0.72	1.4	0.43
Pelvis, hip	2.7 b/	0.9	3.0 b/	6.07	1.36	
	0.48 c/					0.57 c/
Abdomen, without preparation	0.18		0.06	2.22		0.16
IV urography	0.11	17.0	6.65	4.0	0.49	
Hysterography						
Cholecystography	0.01	0.08				0.0001
Skull	0.01	0.01			0.0003	0.0001
Barium enema	2.85			0.75	38.0 d/	0.58
GI tract		1.1		0.25	0.11 d/	
Thorax	0.0001	0.01			0.01	0.0001

a/ Per film.
b/ Hip and upper femur.
c/ Pelvis.
d/ Includes fluoroscopy.

Table 27

Contribution to the annual genetically significant dose from diagnostic x-ray examinations
(per cent)

Examination	France 1982 [M1,M2]	Isl.Rep. of Iran 1980 [S19]	Italy 1983 [P1]	Japan 1979 [H11]	Turkey 1977 [Y1]	United States 1980 [N1]
Skull	0.5	0.2	-	0.01	-	-
Cervical spine	0.5	-	-	-	-	-
Dorsal spine	2.5	-	-	0.02	-	-
Dorsal lumbar spine	5.0	)	-	-	-	-
Lumbosacral spine	2.4	) 25.7	19.0	8.9	5.1	22.5
Pelvis and hip	28.5	26.7	30.5	24.2	9.1	13.7
Abdomen	6.2	6.3	9.1	6.3	5.1	10.4
Urogram	29.8	10.4	14.7	3.5	33.1	12.5
Hysterography	0.7	2.6	-	2.5	19.5	-
Cholecystography	1.8	1.4	-	-	1.9	-
Upper GI (barium)	5.4	1.7	3.1	21.3	10.3	5.5
Barium enema	6.4	18.7	6.9	18.5	6.1	28.0
Chest	1.8	0.6	0.3	0.06	-	6.7
Other	8.5	5.7	16.4	14.9	9.8	0.7
Annual genetically significant dose (mSv) a/	0.30	0.09	0.25	0.15	0.05	0.22

a/ Additional values of the annual genetically significant dose: Canada, 0.26 mSv (1979) [M3]; Switzerland, 0.23 mSv (1978) [P14].

Table 28

Mean gonadal and genetically significant dose in the Russian Federation
[K19, K20]

Year	Annual per caput gonadal dose (mSv)	Genetically significant dose (mSv)
1970	0.33	0.13
1975	0.37	0.17
1980	0.45	0.19
1985	0.47-0.5	0.25-0.5

Table 29

Mean effective dose equivalent
for different diagnostic x-ray examinations
(mSv)

Examination	China	France a/	Italy	Japan	Spain	USSR	United States
	1981 [Z8]	1982 [M2]	1983 [P1]	1986 [M14,M18]	1986 [V4]	1982 [N9]	1980 [N1]
Skull	-	1.35	0.22	0.09	0.2	0.17 c/	0.13
Cervical spine	-	1.35	0.14	0.30	)	0.23 e/	0.20
Dorsal spine	-	2.24	1.34	-	) 1.0	3.55 e/	-
Dorsal lumbar spine	-	-	-		)	-	-
Lumbosacral spine	7.2	4.73	2.51	0.60	)	4.42 e/	1.27
Chest							
Radiographic	0.21	0.28	0.18	0.05	0.16	0.36	0.07
Photofluoroscopic	3.40	-	0.25	-	-	1.15	-
Abdomen	4.5	2.56	1.92	0.29	1.5	1.52	0.56
Upper GI			9.27				
Radiographic	-	6.73		1.2	)	1.52 b/	2.44
Fluoroscopic	-	-		-	)	9.45	-
Barium enema			8.97		) 10.2		
Radiographic	-	9.96		2.0	)	3.55	4.6
Fluoroscopic	-	-		-	)	14.40	-
Cholecystography	4.3	7.21	-	0.55	-	1.97	1.9
Hysterography	-	4.78	-	-	-	-	-
Urogram	-	10.42	7.07	0.70	7.0	2.51	1.6
Pelvis and hip	-	1.59	3.20	0.25	2.3	1.45	0.6
Extremities	-	-	-	-	0.1	0.01	0.1
Computer tomography	-	-		-	5.0		
Dental f/							

a/ Dose does not include component for bone marrow.
b/ P/A projection only.
c/ P/A and LAT projection.
d/ A/P projection only.
e/ A/P and LAT projection.
f/ Values of mean effective dose equivalent for dental x-ray examinations: Japan, 0.03 (intra-oral); 0.04 (extra-oral); USSR, 0.01 (intra-oral) [N9]; United Kingdom, 0.02 (intra-oral); 0.03 (extra-oral); 0.08 (pantomographic) [W4, S15].

Table 30

Annual per caput doses from diagnostic x-ray examinations by level of health care
(mSv)

Level of health care	Country	Year	Annual per caput effective dose equivalent	Annual genetically significant dose	Reference
I	Canada	1980		0.3	[M13]
	Finland	1978	0.7		[R2]
	France	1982	1.6	0.3	[M2]
	Germany, Fed.Rep.	1979		0.5	[S5,S6,S7]
	Italy	1983	0.8	0.3	[P1]
	Japan	1979	1.3	0.2	[U5]
	Netherlands	1980		0.3	[U4, U5]
	Romania			0.3	[U4]
	Poland	1976	1.7		[J1, J2]
	Spain	1986	0.8		[V4]
	Sweden	1985	0.6		[V3]
	Switzerland	1978		0.2	[P14]
	United Kingdom	1984	0.2	0.1	[H20, S16]
	United States	1980	1.3	0.3	[N1]
	USSR	1980/81	1.4	0.2	[K19, K20, N9, V7]
	Average		1.0	0.3	
II	China	1983	0.4	0.09	[Z11]
	Islam.Rep. of Iran a/	1980		0.09	[S19]
	Iraq a/			0.05	[U4]
	Turkey a/	1977		0.05	
III	India a/	1972		0.01	[U4]
	Thailand a/	1970		0.05	[U4]
IV	No data				

a/ Does not include fluoroscopy. If frequency of examinations is 1/10 of level I but fluoroscopy is 30-70% of the total, then the effective dose equivalent and the genetically significant dose may be comparable to those of health care level I.

Table 31

Diagnostic x-ray examination frequency and contribution to per caput absorbed dose in countries of level of health care I

Examination	Annual examinations per caput	Effective dose equivalent per examination (mSv)	Annual per caput effective dose equivalent (mSv)	Annual genetically significant dose a/ (mSv)
Skull	0.050	0.15	0.008	< 0.003 (<1)
Cervical spine	0.020	0.30	0.007	< 0.003 (<1)
Dorsal spine	0.013	1.00	0.013	0.006 (2)
Dorsal lumbar spine	0.013	1.00	0.013	0.006 (2)
Lumbosacral spine	0.025	1.50	0.038	0.045 (15)
Chest, radiographic	0.240	0.10	0.024	0.006 (2)
Abdomen	0.55	1.00	0.055	0.024 (8)
Barium meal and enema	0.070	8.0	0.560	0.066 (22)
Cholecystography	0.013	1.5	0.057	0.006 (2)
Urogram	0.024	3.5	0.084	0.060 (20)
Pelvis and hip	0.038	1.5	0.020	0.006 (2)
Extremities	0.157	0.10	0.016	-
Computer tomography	0.010	1.0	0.010	-
Other	0.096	1.0	0.096	0.030 (10)
Dental	0.250	0.03	0.008	< 0.003 (<1)
Total (rounded)			1.0	0.3

a/ Percentage given in parentheses.

Table 32

Estimated effective dose equivalent and genetically significant dose from diagnostic medical radiography world-wide

Level of health care	Population (millions)	Annual per caput effective dose equivalent (mSv)	Annual genetically significant dose (mSv)	Annual collective effective dose equivalent (thousands of man Sv)
METHOD 1 a/				
I	1300	1.0	0.3	1300
II	1750	0.2	0.07	350
III	1220	0.07	0.02	85
IV	730	0.03	0.01	22
Total				1760
METHOD 2 b/				
I-IV	5000	1.0	0.3	5000

a/ Method 1 assumes that in levels II-IV dose is related to the frequency and rate of examinations.

b/ Method 2 assumes that increased dose from fluoroscopy in levels II-IV countries makes absorbed dose comparable to level I.

Table 33

Estimated annual dose from dental radiography world-wide

Level	Population (millions)	Annual per caput effective dose equivalent (mSv)	Annual collective effective dose equivalent (thousands of man Sv)	Genetically significant dose (μSv)
I	1300	0.01 a/	13.0	0.08
II	1750	0.002	3.5	0.02
III	1220	0.0006	0.7	0.004
IV	730	0.0003	0.2	0.002
Total	5000		17.4	

a/ Data from Poland and United Kingdom [H20, K5, J1, U4, U5].

Table 34

Occupational exposures from diagnostic x-ray examinations
(mSv)

Category	Average annual dose equivalent (mSv)				Reference
	1974-1976	1978-1979	1980-1981	1984	
MEDICAL					
Radiologists					
Canada		0.4		0.25	U5,C3
Japan		0.3			M19
Norway			2.7		W17
Switzerland		0.6			U5
United Kingdom			0.51		H20
United States			1.7		N1
Technologists					
Canada		0.2		0.12	U5,C3
Japan		0.5			M19
United Kingdom			0.37		H20
United States			0.5		N1
Nurses					
Canada		0.4		0.15	U5,C3
Japan		0.2			M19
United Kingdom			0.35		H20
Aides, porters					
Canada		0.4		0.08	U5,C3
United Kingdom			0.14		H20
Physicists					
Canada				0.36	C3
Norway			0.74		W17
United Kingdom			0.14		H20
All medical workers					
Japan		0.5			M19
Poland a/	0.5-1.0				J3
United Kingdom			0.14		H20
DENTAL (all workers)					
Australia		0.1			U5
Canada	0.04	0.05		0.02-0.05	U4,U5,C3
France	0.5	0.4			U5
Switzerland		0.2			U5
United Kingdom				0.1	H20
United States b/			0.2		K23

a/ 1966-1978.
b/ Earlier values: 1.1 (1960); 0.6 (1970) [K23].

Table 35

Frequency of diagnostic nuclear medicine examinations
(per 1000 population)

Numbers in parentheses indicate percent of total

Examination	Australia	China	Denmark	
	1980 [U5]	1981 [Z6]	1981 [U5]	1985 [E2]
Brain	1.5 (18.4)	< 0.1 (0.3)	1.8 (14.3)	1.1 (7.4)
Biliary	0.1 (1.7)	-	0.1 (0.9)	0.2 (1.3)
Liver/spleen	1.7 (21.5)	0.2 (25.1)	0.8 (8.6)	1.0 (7.0)
Bone	2.0 (24.4)	< 0.1 (<0.1)	2.5 (17.7)	2.8 (19.3)
Pulmonary	1.2 (14.7)	-	0.6 (4.1)	1.1 (7.7)
Thyroid a/	0.8 (10.5)	0.4 (60.0)	2.0 (14.0)	1.7 (11.9)
Renal	0.2 (1.9)	< 0.1 (13.5)	2.5 (17.6)	4.8 (33.8)
Tumour/abcess	-	-	-	0.2 (1.6)
Cardiovascular	0.1 (1.7)	-	< 0.1 (0.2)	1.1 (8.0)
Other	0.4 (5.2)	< 0.1 (2.3)	3.2 (22.6)	0.3 (2.0)
Total	8.0 (100)	0.6 (100)	14.1 (100)	14.3 (100)

Examination	Poland 1981 [S25]	United Kingdom 1982 [W8, W9]	United States 1982 [U5]	b/
Brain	0.1 (5.5)	0.90 (15.0)	3.58 (11.0)	
Biliary	-	-	0.79 (2.4)	
Liver/spleen	0.3 (13.2)	0.88 (14.7)	6.27 (19.2)	
Bone	0.1 (2.9)	1.67 (27.8)	7.98 (24.5)	
Pulmonary	< 0.1 (0.4)	0.69 (11.5)	5.25 (16.1)	
Thyroid a/	1.2 (55.2)	0.41 (6.8)	2.98 (9.1)	
Renal	0.4 (19.1)	0.45 (7.5)	1.04 (3.2)	
Tumour/abcess	< 0.1 (0.4)	0.06 (1.0)	0.53 (1.6)	
Cardiovascular	< 0.1 (1.4)	0.18 (3.0)	4.19 (12.9)	
Other	< 0.1 (1.7)	0.76 (12.7)	-	
Total	2.2 (100)	6.0 (100)	32.6 (100)	

a/ Thyroid scans and uptakes.
b/ Additional reported values of total frequency: Canada (1981), 49.0 [C1]; Finland (1982), 17.7 [T3]; France (1982), 8.7 [B9].

Table 36

Annual number of diagnostic nuclear medicine examinations in the United States
(thousands)
[M28]

Examination	1966	1972	1973	1975	1978	1980	1982
Brain	62	1250	1510	2120	1546	1176	812
Hepatobiliary		26					179
Liver	60	455	535	676	1302	1349	1424
Bone	7	81	125	220	1160	1307	1811
Respiratory	23	332	417	597	1053	898	1191
Thyroid (uptake and scans)	454	647	460	627	699	606	677
Urinary		108	122	154	205	164	236
Tumour		10	14	22	166	130	121
Cardiovascular		25	33	49	160	558	950
Other	120	405	294	338	115	186	4
Total a/	726 (4)	3339 (16)	3510 (17)	4803 (22)	6406 (29)	6374 (29)	7405 (32)

a/ Figures in parentheses refer to number of procedures per 1000 population.

Table 37

Type and percent of diagnostic nuclear medicine examinations in some western hemisphere countries, 1981-1982
[I7]

Country	Thyroid	Hepatic/ Biliary	Brain	Bone	Lungs	Other
Brazil	50	10	15	10	5	10
Colombia	20	25	10	20	10	15
Ecuador	60	15	10	5		10
El Salvador	40	30	20	10		
Mexico	30	25	10	15	20	
Peru	50	15		25		10
United States	9	22	11	24	16	18

Table 38

Annual frequency of diagnostic nuclear medicine examinations
(per 1000 population)

Country	1970-1972	1973-1975	1977-1979	1980-1982	Reference
Australia	4			8	W8, U5
Austria			18		W8
Bulgaria				13	W8
Burma	0.1		0.2		U5
Canada				49	C1
China				0.6	Z5
Cuba	0.8		0.8		U5
Denmark		8	14	14 a/	U5,W8,E2
Finland				18	T3
France				9	B9
Japan			5	8	M15, H9
Poland				2	S25
Sweden	8	12	15	15	U5,W8
United Kingdom				7	W8
United States b/	16	11	29	31	M31
USSR				4	V7

a/ 1985 value.
b/ Earlier value: 4 (1966).

Table 39

Nuclear medicine examinations by level of health care

Level of health care	Country	Annual examinations per 1000 population	Population per scanner or camera (thousands)
I	Australia (1980) [U5]	8	75 a/
	Austria (1977) [W7]	18	57 a/
	Bulgaria (1980) [W7]	13	76 a/
	Canada (1981) [C2]	49	20 a/
	Denmark (1987) [E2]	14	71 a/
	Finland (1982) [T3])	18	-
	France (1982-87) [B9, P11]	9	160 a/
	German Dem.Rep.	8	122 a/
	Sweden (1982) [W8,U5]	15	50 a/
	United Kingdom (1982) [W8]	7	160
	United States (1982) [M25]	32	31 a/
	Rounded average	16	160
II	Bolivia	-	650
b/	Brazil	-	613
	China	0.6	
	Colombia	-	830
	Cuba	0.8	
	Ecuador	-	810
	Mexico	-	800
	Philippines	-	1600
	Uruguay	-	340
	Rounded average		800
III	Burma	0.2	
b/	India	0.1	4700
	Malaysia	-	1300
	Thailand [K17]	-	500
	Rounded average		2300
IV	Bangladesh		4750
b/	Indonesia		4000
	Nigeria [F4]	< 0.0001	-
	Pakistan		3600

a/ Estimated from the number of examinations, assuming 1000 examinations annually per machine [W8, W9].
b/ Except for referenced entries, the data were obtained between 1978 and 1984 and provided to UNSCEAR by the IAEA.

Table 40

Estimated average nuclear medicine examinations by level of health care

Level of health care	Annual examinations per 1000 population	Population per scanner or camera (thousands)
I	16	160
II	1.2 a/	800
III	0.4 a/	2300
IV	0.2 a/	4100

a/ Estimated from the number of machines, assuming 1000 examinations annually per machine [W7].

Table 41

Estimated world-wide diagnostic nuclear medicine examinations and number of machines

Numbers in parentheses indicate per cent of total

Level of health care	Population (millions)	Cameras or scanners	Annual diagnostic examinations (millions)
I	1300 (26)	20800 (89)	20.8 (89)
II	1750 (35)	2100 (9)	2.1 (9)
III	1220 (24)	500 (2)	0.5 (2)
IV	730 (15)	100 (< 1)	0.1 (< 1)
Total	5000 (100)	23500 (100)	23.5 (100)

Table 42

Age and sex distribution of patients undergoing diagnostic nuclear medicine examinations
[S25, U10]

Age and sex		Brain		Thyroid		Cardiovascular		Pulmonary	
		Poland	United States	Poland	United States	Poland	United States	Poland	United States
< 15	male	4.9	1.4	0.4	0.1	1.5	-	7.3	-
	female	4.4	1.3	2.2	1.3	0.9	-	1.6	-
	both	9.3	2.7	2.6	1.4	2.4	-	8.9	-
15-29	male	10.3	4.4	3.3	2.6	16.7	1.5	22.4	2.8
	female	8.1	2.6	20.4	14.0	6.3	0.8	5.4	5.3
	both	18.4	11.0	23.7	16.6	23.0	2.3	27.8	8.1
30-44	male	11.1	4.9	3.9	4.5	22.4	8.1	13.3	6.0
	female	11.4	7.7	30.2	23.0	7.1	4.3	11.9	8.8
	both	22.5	12.6	34.1	27.5	29.5	12.4	25.2	14.8
45-64	male	21.0	14.4	5.2	7.1	31.6	33.3	16.3	18.2
	female	15.5	15.1	29.7	28.5	6.4	20.4	6.4	18.9
	both	36.5	29.5	34.9	35.6	38.0	53.7	22.7	37.1
> 64	male	7.1	19.7	1.0	3.6	4.2	16.2	11.0	17.5
	female	6.2	24.5	3.7	15.3	2.9	15.4	4.4	22.5
	both	13.3	44.2	4.7	18.9	7.1	31.6	15.4	40.0
All ages	male	54.4	45.5	13.8	17.9	76.4	59.1	70.1	44.5
	female	45.6	54.5	86.2	82.1	23.6	40.9	29.9	55.5
	both	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Age and sex		Liver/spleen		Renal		Bone		All examinations	
		Poland	United States	Poland	United States	Poland	United States	Poland	United States
< 15	male	2.3	0.1	4.0	1.4	5.2	1.1	2.0	0.9
	female	2.4	-	5.6	2.1	3.2	0.9	3.3	0.7
	both	4.7	0.1	9.6	3.5	8.4	2.0	5.3	1.6
15-29	male	6.4	3.4	11.2	5.7	11.3	2.9	6.4	3.3
	female	6.9	3.1	9.4	5.7	11.6	2.3	15.4	4.9
	both	13.3	6.5	20.6	11.4	22.9	5.2	21.8	8.2
30-44	male	9.5	5.4	11.5	10.7	8.7	3.6	7.1	5.2
	female	10.5	5.7	12.9	10.0	15.0	6.4	21.7	8.7
	both	20.0	11.1	24.4	20.7	23.7	10.0	28.8	13.9
45-64	male	22.4	18.8	17.7	21.5	13.3	14.9	11.7	15.8
	female	21.5	21.7	17.4	15.8	22.9	23.9	24.0	21.6
	both	43.9	40.5	35.1	37.3	36.2	38.8	35.7	37.4
> 64	male	8.8	19.5	4.3	15.7	4.3	20.5	3.3	17.0
	female	9.3	22.3	6.0	11.4	4.5	23.5	5.1	21.9
	both	18.1	41.8	10.3	27.1	8.8	44.0	8.4	38.9
All ages	male	49.5	47.2	48.5	55.0	42.8	43.0	30.5	42.0
	female	50.5	52.8	51.5	45.0	57.2	57.0	69.5	58.0
	both	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Table 43

Average activity used for some common nuclear medicine examinations

Organ	Radiopharmaceutical	Average activity (MBq)			
		Germany Fed.Rep. [K2,K3]	Poland [S25]	Sweden a/ [M6]	United Kingdom a/ [W9]
Thyroid b/	^{99m}Tc-pertechnetate	37	37	81 (37-146)	75 (15-200)
	^{123}I-iodide	3.7	-	1.5 (0.7-1.9)	
	^{131}I-iodide	1.9	2.7	3 (1- 8)	
Liver/ spleen	^{99m}Tc-colloid	167	148	100 (75-191)	90 (37-200)
	^{198}Au-colloid	5.6		18.5	
Renal	^{131}I-Hippuran	1.5	1.2	4.5	28 (10-185) c/
	^{99m}Tc-DTPA	370		163 (40-560)	248 (37-555)
	^{99}Tc-glucoheptonate	370	136	370	
	^{99m}Tc-DMSA	111	192	177 (75-600)	102 (37-500)
Bone	^{99m}Tc-phosphate	555		409 (150-600)	520 (330-740)
Cardiac	^{99m}Tc-erythrocytes	740		540 (529-600)	658
	^{201}Tl-chloride	74		65 (42-350)	68 (40-100)
Lung	^{99m}Tc-microspheres	167		80 (42-110)	88 (37-222)
Brain	^{99m}Tc-DTPA	463		601 (337-750)	536
	^{99m}Tc-pertechnetate	463		403 (200-560)	490
	^{99m}Tc-glucoheptonate		592	403 (200-560)	570

a/ Numbers in parentheses indicate ranges where data are available.
b/ Additional values from the United States are 237 (37-555), 0.8 (0.3-1.6), 0.3 (0.04-0.4) for ^{99m}Tc-pertechnetate, ^{123}I-iodide and ^{131}I-iodide, respectively.
c/ Iodine-123.

Table 44

Annual collective effective dose equivalent for in vivo diagnostic nuclear medicine examinations
(man Sv)

Examination	China a/ 1981 [Z6]	Finland 1982 [T3]	Poland 1981 [S25]	Sweden 1983 [J8]	USSR 1981 [V7]	United Kingdom 1982 [H20]	United States 1982 [M30]
Brain	13	142	28	41	-	253	5280
Hepatobiliary	-	-	-	-	-	-	660
Liver/spleen	924	14	23	19	-	55	3420
Bone	4	45	-	81	-	319	7970
Pulmonary	-	7	4	9	-	34	1790
Thyroid	3740	203	1922	268	-	49	3980
Renal	1	-	5	1	-	8	730
Tumour/abcess	-	-	10	-	-	30	1480
Cardiovascular	-	10	3	-	-	54	6750
Other	12	13	24	121	-	148	-
Annual collective effective dose equivalent (total) (man Sv)	4700	430	2020	540	8700	950	32000
Annual per caput effective dose equivalent (mSv)	0.005	0.090	0.057	0.060	0.034	0.017	0.14

a/ Assumes that Shandong Province is representative.

Table 45

Radionuclide contribution to annual collective effective dose equivalents from diagnostic nuclear medicine
(per cent)

Radionuclide	China 1981 [Z6]	Finland 1982 [T3]	Poland 1981 [S25]	Sweden 1983 [J8]	United Kingdom 1982 [H20]	United States 1982 [M30]
Technetium-99m	-	51	2	33	89	68
Iodine-131	77	47	96	62	4	17
Other	23	2	2	5	7	15 a/

a/ Two-thirds of this is due to thallium-201.

Table 46

Annual per caput doses from diagnostic nuclear medicine examinations
(mSv)

Level of health care	Country	Year	Per caput effective dose equivalent	Annual genetically significant dose equivalent	Ref.
I	Australia	1980	0.02		[U5]
	Denmark	1985	0.05		[E2]
	finland	1982	0.09	-	[T3]
	Japan	1982	0.04	0.004	[M15]
	Sweden	1983	0.06		[J8]
	United Kingdom	1982	0.017	0.003	[H20]
	United States	1982	0.14	0.19	[M30]
	USSR	1981-1982	0.03-0.04		[V7, K13]
	Average		0.05 a/	0.01 b/	
II	China	1981	0.005		[Z6]
III	c/				
IV	c/				

a/ Population weighted average collective effective dose equivalent = 0.067 mSv.
b/ Assumes genetically significant dose is approximately 20% of effective dose equivalent.
c/ No data available.

Table 47

Estimated collective effective dose equivalent and genetically significant dose from in vivo diagnostic nuclear medicine

Level of health care	Population (millions)	Annual per caput effective dose equivalent (mSv)	Annual genetically significant dose (mSv)	Annual collective effective dose equivalent (thousands of man Sv)
I	1300	0.050	0.010	65.0
II	1750	0.004	0.0008	7.0
III	1200	0.001	0.0002	1.2
IV	730	0.0005	0.0001	0.4
Total	5000			74

Table 48

Average annual individual occupational doses for nuclear medicine technologists from diagnostic nuclear medicine

	Austria	Canada	France	Norway	United Kingdom
	1978 [U5]	1951-1983 [S20,S21]	1979 [U5]	1983 [W17]	1984 [H20]
Nuclear medicine technologists:					
Average annual dose (mSv)	0.4	2.0	0.5	0.6	0.3-1.4
Collective dose (man Sv)	0.4	-	1.2	0.3	-

Table 49

Radiation therapy treatments in Canada a/ [C1]

Type of treatment	Year		
	1978-1979	1979-1980	1980-1981
Superficial x ray	19 098	12 028	11 827
Deep x ray	109 702	20 925	29 392
Cobalt	333 355	274 470	244 422
Radium	2 202	2 199	1 289
Other	103 913	314 187	375 270
Total	568 270	623 809	662 197

a/ For eight provinces, which comprise approximately 91.5% of the Canadian population.

Table 50

Estimated annual number and type of cases treated by radiation therapy in some western hemisphere countries
[U7]

Country	New cancer cases treated	New cancer cases treated with radio-therapy	Type of case treated (%)					
			Breast	Lymph-phoma	Gynae-colog-ical	Dige-stive	Other cancers	Non-malig-nant
Columbia	3 000	2 000	11	8	31	17	7	5
Costa Rica	2 880	500						3
Ecuador	3 000	1 900		20	35	30	3-4	1-2
El Salvador	817	817	10		60		17	0
Mexico	580 000	50 000	15		20		9	1
Peru	10 000	6 000	15	10	65		8	2
United States	800 000	390 000						5
Venezuela	10 000	6 000	30		50	5	4	1

Additional values: head and neck, 10 (Venezuela); skin, 17 (Colombia) and 10 (Ecuador); lungs, 4 (Colombia) and 10 (Mexico).

Table 51

Number of megavoltage radiotherapy units in the United States and annual number of new patients per unit
[K15, R7]

Year	Cobalt	Linear accelerators and betatrons	New patients per unit
1975	970	407	227
1978	900	606	232
1980	980	801	233

Table 52

Radiation therapy experience by level of health care

Level of health care	Country	Year	Annual procedures per million population: Brachytherapy and teletherapy	Annual procedures per million population: Unsealed radionuclides	Machines per million population a/	Reference
I	Argentina	1981	-	-	14	[I7]
	Denmark	1985	-	205	-	[E2]
	France	1987	-	-	8	[P11]
	Germany, Fed.Rep.	1975	-	260	-	[U5]
	Japan	1983	1000	-	-	[H6, M13]
	Sweden	1978	-	375	-	[U5]
	United Kingdom	1984	2400	600	-	[D3]
	United States	1981	2400	-	10	[K14, R6]
	Average		~ 2400	~ 400		
II	Brazil	1981	-	-	2.5	[I7]
	Chile	1981	-	-	4.9	[I7]
	Ecuador	1981	-	-	2.3	[I7]
	Mexico	1981	-	-	1.2	[I7]
	Peru	1981	-	-	1.1	[I7]
	Venezuela	1981	-	-	2.4	[I7]
	Average		~ 600	~ 400		
III	Burma	1978	-	6	-	[U5]
	India	1976	125 b/	-	0.5	[M21]
	Sri Lanka	1978	350	2	-	[U5]
	Sudan	1985	70 b/	-	0.3	[S36]
	Average		100 b/		0.4	
IV	Indonesia	1978	7	-	-	[U5]

a/ Data also indicate approximately 200 new patients (or 250 total patients) per machine annually. Machines include teletherapy, cobalt and accelerators.
b/ Estimated based on 250 patients annually per machine.

Table 53

Estimated radiation therapy activity by level of health care
[M27]

Level of health care	Annual procedures per million population: Brachytherapy and teletherapy	Annual procedures per million population: Unsealed radionuclides	Machines per million population a/
I	2400	400	10
II	600	100 a/	2.5
III	100	16 a/	0.4
IV b/	50	8	0.2

a/ Estimates based on percentage of brachytherapy and teletherapy procedures.
b/ Estimates based on regression from nuclear medicine activity.

Table 54

Estimated worldwide radiation therapy procedures and machines [M27]

Level of health care	Population (millions)	Annual procedures or courses of treatment: Brachytherapy and teletherapy (thousands)	Annual procedures or courses of treatment: Unsealed radiotherapy (thousands)	Number of machines
I	1300	3120	520	13000
II	1750	1050	175	4400
III	1220	120	20	490
IV	730	40	6	150
Total	5000	4300	720	18000

Table 55

Estimated genetically significant dose from radiation therapy

Level of health care	Population (millions)	Annual genetically significant dose (mSv)
I	1300	0.015 a/
II	1750	0.0037
III	1220	0.0006
IV	730	0.0003

a/ Average of five reported values [H18, U5].

Table 56

Average annual individual occupational dose from radiation therapy (mSv)

Category	Australia 1978 [U5]	Canada 1984 [C3]	Norway 1983 [W17]	United Kingdom 1981 [H20]	United States 1975 [U5]
Beam therapy	0.8-1.5	-	-	1.0	-
Brachytherapy					
Radiotherapists	-	0.41	-	1.8	-
Anaesthetists	-	-	-	1.3	-
OR nurses	-	-	-	23.0	-
Ward Nurses	-	-	-	3.0	-
Laboratory staff	-	-	-	14.8	-
Mould room staff	-	1.23	-	1.5	-
Physicists	-	-	-	0.6	-
All workers	1.0-2.0	-	1.04	2.57	3.0
Collective dose (man Sv)	0.4	-	1.05	-	60

Table 57

Estimate of world-wide collective effective dose equivalent and genetically significant collective dose from medical uses of radiation
(thousands of man Sv)

Source	Annual collective effective dose equivalent	Annual genetically significant collective dose
Diagnostic medical	1800-5000	500-1500
Dental	17	0.14
Diagnostic nuclear medicine	74	15
Radiation therapy	-	27
Per caput (mSv)	~ 0.4-1.0	~ 0.1-0.3

Table 58

Occupational exposure from medical uses of radiation

Country	Year	Annual average dose (mSv)	Annual collective dose (man Sv)	Collective dose per million population (man Sv)	Reference
Canada	1974		10.5		U4
	1984	0.3	7.2	0.3	C3, W17
France	1974	1.3	29		U4
	1979			0.8	U5
Germany, Federal Rep.	1984	1.1	27	0.4	N12
Japan	1978	0.5	55	0.5	M19
Norway	1983	1.3	5.8	1.4	W17
United Kingdom	1984	0.7	28	0.5	H20
United States	1960	1.9	580		K23
	1970	1.1	500		K23
	1980	0.7	410	1.8	K23

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back to first page

ANNEX D

Exposures from the Chernobyl accident

CONTENTS

Introduction

1. The accident in April 1986 at the Chernobyl nuclear power station in the Union of Soviet Socialist Republics, in which large amounts of radioactive materials were released into the environment, was the most serious to have occurred in connection with the use of nuclear energy to generate electricity. Swift emergency response was required, first of all in the USSR to control and contain the damaged reactor and then, also, in other countries to monitor and evaluate the radiation levels. Because of the attention focused on the accident and its aftermath and the large data base that was accumulated, the Committee has decided to assess in detail the population exposures that resulted from the accident in order to improve the comparability of results between countries and to develop further the methodology for dose assessment from this type of radiation source.

2. The radiation levels from released radionuclides were highest in the immediate vicinity of the reactor. The released radioactive materials affected then mainly the western part of the USSR and the countries of Europe. Extensive measurements have been made in these regions, allowing the radiation doses to the affected populations to be evaluated in some detail. Because the released materials became further dispersed throughout the northern hemisphere, estimates of exposures to populations in other countries have also been made.

3. In presenting the results of the assessment, a short account is given of the conditions under which the accident took place, mainly to convey information that will help to evaluate the radiological impact. General aspects of the dispersion of the released radioactive materials are described. The environmental concentrations and radiation levels encountered are systematically evaluated and then applied in a common methodology for estimating radiation doses.

4. One of the major uncertainties in this dosimetric assessment is that pertaining to projected future exposures from the residual radioactive materials in the environment. Environmental levels and radiation doses continue to be measured, and the Committee plans to use these data to refine the values of the parameters required for the calculations. It will, for example, consider further the regional variabilities due to different meteorological or ecological conditions. Such analyses would greatly help in refining the transfer factors and the models used by the Committee in dose assessments.

5. The Committee has received a great deal of assistance and co-operation from many individuals and organizations in carrying out this assessment. A team of experts was formed in the UNSCEAR Secretariat by staff seconded by the Institute of Biophysics at the Ministry of Health in Moscow, USSR; by the National Cancer Institute and the Department of Energy in the United States; by the Monitoring and Assessment Research Centre in London and the National Radiological Protection Board in the United Kingdom; and by the National Committee for the Research and Development of Nuclear Energy and Alternative Energies in Italy.

6. Many countries submitted scientific data either directly to the UNSCEAR Secretariat or to the data bank set up in Vienna by the International Atomic Energy Agency. The UNSCEAR team of experts had free access to this data bank for the purpose of deriving data for the assessment. To obtain additional data, the UNSCEAR Secretariat also maintained frequent and extensive contacts with experts in various countries and discussed with them the interpretation and evaluation of results. These contacts were so numerous that it would be impossible to acknowledge them separately. They proved essential to the conduct of the project and they are here collectively recognized with appreciation.

7. In approving this Report, the Committee wishes to acknowledge this help and express its gratitude. It would also like to draw attention to and commend the spirit of full collaboration and free exchange of data

and ideas between countries, international organizations, laboratories and scientists, which has greatly enhanced the outcome of this study.

I. THE ACCIDENT

8. On 26 April 1986 at 0123 hours local time an accident occurred at the fourth unit of the Chernobyl nuclear power station. The accident destroyed the reactor core and part of the building in which the core was housed. The radioactive materials released were carried away in the form of gases and dust particles by air currents. In this manner, they were widely dispersed over the territory of the Soviet Union, over many other (mostly European) countries and, in trace amounts, throughout the northern hemisphere.

A. THE REACTOR

1. Location

9. The Chernobyl nuclear power station is located in the Ukrainian Soviet Socialist Republic in the western USSR, near the boundary with the Byelorussian Soviet Socialist Republic. It lies about 100 km north-west of Kiev and 310 km south-east of Minsk, on the River Pripyat, which flows into the Dnieper (Figure I). The nearest boundaries with neighbouring countries, Poland (eastern part) and Romania (northern part), are 450 km away.

10. The eastern Byelorussian-Ukrainian woodlands region is characterized by a relatively flat landscape, with minor slopes down to the river or its tributaries. The soils of the region are mostly soddy-podzolic, distinguished by low natural fertility. They are, as a rule, acid (pH 4.5-5.5) and have a low content of minerals. The area north of the reactor consists of about 50% agricultural land and 50% natural complexes (forests, bogs, water basins). Ploughed land makes up about half of the agricultural land, with the remainder devoted to natural fodder grasses (cereals and sedge meadows). Dairy and cattle husbandry is well developed in the region. Potato crops occupy 8% of the territory. To the south of the reactor, in the Ukraine, the agricultural use of the land increases, and only 10% of it consists of natural landscapes [12].

11. The average population density in the region had been approximately 70 inhabitants per km^2 up to the start of construction work on the Chernobyl power plant. At the beginning of 1986, the total population within an area of 30 km radius around the power plant was approximately 100,000; of this total, 49,000 lived in the town of Pripyat, situated to the west of the plant's 3-km safety zone, and 12,500 in the town of Chernobyl, the regional centre, about 15 km to the south-east of the plant.

12. The construction of the Chernobyl nuclear power station was carried out in three stages; each comprised two 1,000-MW reactor units. The first stage (Units 1 and 2) was constructed between 1970 and 1977 and

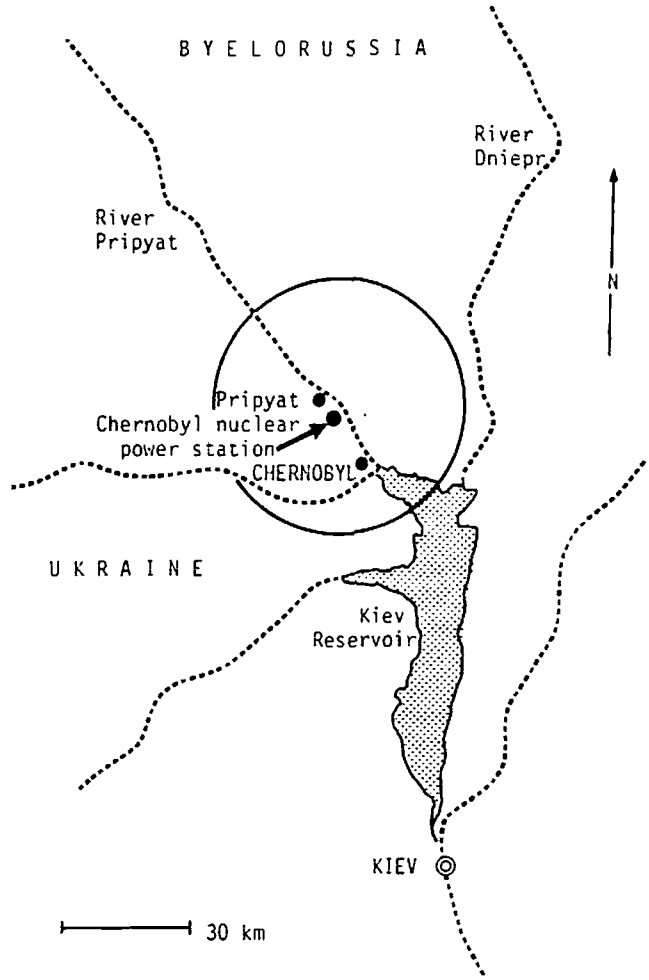

Figure I. The site of the Chernobyl nuclear power station.

the second (Units 3 and 4) was completed in late 1983. In 1981, work was started on two more units of the same type at a site 1.5 km to the south-east of the existing site [11].

2. Design characteristics

13. The reactors of the Chernobyl nuclear power station are graphite-moderated, light-water-cooled systems known as RBMK-1000. The installed electrical generating capacity of each unit is 1,000 MW. Each pair of reactors at the station shares a turbine generator room that houses four turbine generators and their associated multiple forced circulation systems. The reactor pairs are located in separate blocks adjoining the central service unit.

14. The core matrix of the RBMK-1000 reactor consists of graphite blocks (250 mm × 250 mm, 600 mm high) stacked together to form a cylindrical configuration 12 m in diameter and 7 m high. It is located in a leak-tight cavity formed by a cylindrical shroud, the bottom support structure and the upper steel cover. Apart from the solid graphite blocks forming the radial reflector, each block has a central hole providing the space for the fuel channels or absorber rod channels. There are 1,661 individual vertical fuel channels. Fuel and control rod channels penetrate the lower and upper steel structures and are connected to

two separate cooling systems, below and above the core.

15. The fuel, in the form of UO_2 pellets, is sheathed in a zirconium-niobium alloy. Eighteen fuel pins, approximately 3.5 m long, are arranged in a cylindrical cluster; two of these clusters fit on top of each other into each fuel channel. Fuel replacement is done on-power by a fuelling machine located above the core. One or two fuel channels can be refuelled each day.

16. The coolant system consists of two loops. The coolant enters the fuel channels from the bottom at 270°C. heats as it moves upward, and partially evaporates. The mass steam content at the core outlet is approximately 14.5% at full-power operation. The outlet pressure and temperature are 7 MPa (70 bars) and 284°C. The wet steam of each channel is fed to steam drums, of which there are two for each cooling loop. The dry steam from the drums is fed into one of two 3,000 rpm 500-MW(e) turbine generators. The circulation pumps supply the coolant to headers, which distribute it to the individual fuel channels of the core. In each loop, four pumps are provided, one of which is normally on stand-by during full-power operation. The coolant flow of each fuel channel can be independently regulated by an individual valve to compensate for variations in the power distribution. The flow rate through the core is controlled by feed pumps [11].

17. Approximately 95% of the energy from the fission reaction is transferred directly to the coolant. The remaining 5% is absorbed within the graphite moderator and mostly transferred to the coolant channels by conduction, which leads to a maximum temperature within the graphite of approximately 700°C. A mixture of helium and nitrogen gases enhances the gap conductance between the graphite blocks and provides chemical control of the graphite and pressure tubes.

18. The Chernobyl Unit 4 reactor had the following principal specifications [11]:

Thermal power	3,200 MW
Fuel enrichment	2.0%
Mass of uranium in fuel assembly	114.7 kg
Fuel burn-up	20 MW d/kg
Maximum design channel power	3,250 kW
Isotopic composition of unloaded fuel	
U-235	4.5 kg/t
U-236	2.4 kg/t
Pu-239	2.6 kg/t
Pu-240	1.8 kg/t
Pu-241	0.5 kg/t

19. At equilibrium fuel irradiation, the reactor has a positive void reactivity coefficient. However, the fuel temperature coefficient is negative and the net effect of a power change depends on the power level. Under normal operating conditions, the power coefficient is negative at full power and becomes positive below approximately 20% of full power. The operation of the reactor below 700 MW(th) is therefore restricted by normal operating procedures. The radionuclide composition of the Chernobyl Unit 4 core is shown in Table 1.

3. Cause of the accident

20. The accident happened while a test was being carried out on a turbine generator during a normal, scheduled shutdown of the Unit 4 reactor. The test was intended to ascertain the ability of a turbine generator, during station blackout, to supply electrical energy for a short period until the stand-by diesel generators could supply emergency power. Written test procedures that were unsatisfactory from the safety point of view, and serious violations of basic operating rules put the reactor at low-power [200 MW(th)] operation in coolant flow rate and cooling conditions that could not be stabilized by manual control. In view of the design features already mentioned (the positive power coefficient at low power levels), the reactor was being operated in an unsafe regime. At the same time, the operators, deliberately and in violation of rules, withdrew most control rods from the core and switched off some important safety systems [11].

21. The subsequent events led to the generation of an increasing number of steam voids in the reactor core, which enhanced the positive reactivity. The beginning of an increasingly rapid rise in power was detected, and a manual attempt was made to stop the chain reaction (the automatic trip, which the test would have triggered earlier, had been blocked). However, there was little possibility of rapidly shutting down the reactor as almost all the control rods had been completely withdrawn from the core. The continuous reactivity addition by void formation led to a prompt critical excursion. It was calculated that the first power peak reached 100 times the nominal power within four seconds [11]. Energy released in the fuel by the power excursion suddenly ruptured part of the fuel into minute pieces. Small, hot fuel particles (possibly also evaporated fuel) caused a steam explosion.

22. The energy released shifted the 1,000-tonne cover plate of the reactor, cutting all the cooling channels on both sides of the reactor cover. After two or three seconds, another explosion occurred, and hot pieces of the reactor were ejected from the damaged reactor building. The damage to the reactor permitted the influx of air, which then caused the graphite to burn.

B. RADIONUCLIDE RELEASE AND DISPERSION

1. Release sequence and composition

23. Damage to the reactor containment and core structures led to the release of large amounts of radioactive materials from the plant. The release did not occur in a single massive event. On the contrary, only 25% of the materials released escaped during the first day of the accident; the rest escaped over a nine-day period. The estimated percentages of various radionuclides released from the total in the inventory are shown in Table 1. Soviet experts were able to reconstruct the overall release process, as shown in the time-dependent release-rate curve in Figure II.

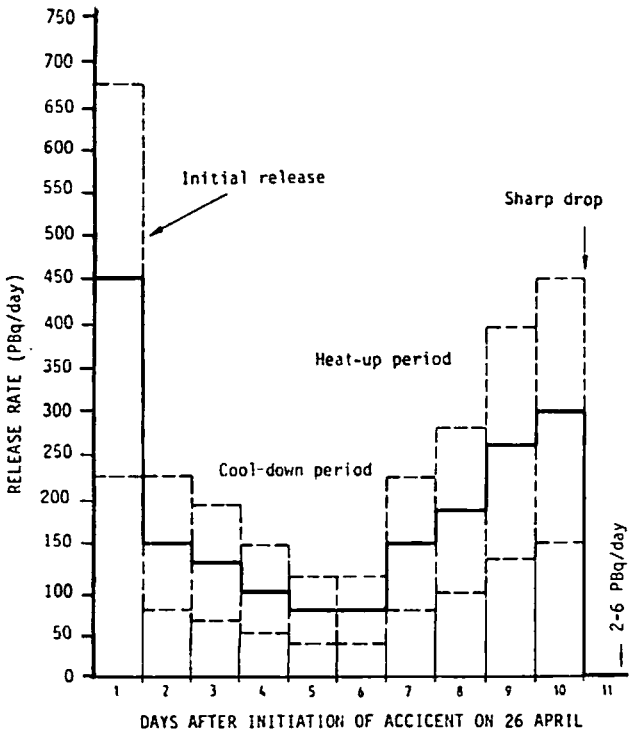

Figure II. Daily release rate to the atmosphere of radioactive materials, excluding noble gases, during the Chernobyl accident. The values are decay-corrected to 6 May 1986 and have a range of uncertainty of ± 50%. [I1]

24. The release-rate curve may be subdivided into four stages:

(a) The initial release on the first day of the accident. During this stage, the mechanical discharge of radioactive materials was the result of the explosion in the reactor;

(b) A period of five days during which the release rate declined to a minimum approximately six times lower than the initial release rate. In this stage, the release rate decreased owing to the measures taken to fight the graphite fire. These measures, which consisted of dropping about 5,000 tonnes of boron carbide, dolomite, clay and lead on to the core from helicopters, led to the filtration of the radioactive substances released from the core. At this stage, finely dispersed fuel escaped from the reactor directly with a flow of hot air and with the fumes from the burning of the graphite.

(c) A period of four days during which the release rate increased again to about 70% of the initial release rate. Initially, an escape of volatile components, especially iodine, was observed; subsequently, the composition of the radionuclides resembled that in spent fuel. These phenomena were attributed to heating of the fuel in the core to above 2000°C, owing to residual heat release.

(d) A sudden drop in the release rate nine days after the accident to less than 1% of the initial rate and a continuing decline in the release rate thereafter. This final stage, starting on 6 May, was characterized by a rapid decrease in the escape of fission products and a gradual termination of discharges. These phenomena were the consequence of the special measures taken, which caused the fission products to be included in compounds that were chemically more stable.

25. On the basis of radiation measurements and analyses of samples taken within a 30 km radius of the plant and throughout the USSR, it was estimated that materials with activity in the range of 1-2 EBq had been released from the fuel during the accident. An error range of ± 50% has been quoted. These figures do not include the release of the noble gases xenon and krypton, which are thought to have been released completely from the fuel. About 10-20% of the volatile radionuclides iodine, caesium and tellurium and 3-6% of other more stable radionuclides, such as barium, strontium, plutonium, cerium etc., were estimated to have been released (Table 1). The estimate of the ^{137}Cs release is compared in section VI.D with the amount calculated from estimated deposition in the northern hemisphere. The agreement is reasonable, considering the wide uncertainties associated with both estimates.

26. Only two earlier reactor accidents caused significant releases of radionuclides: the one at Windscale (United Kingdom) in October 1957 and the other at Three Mile Island (United States) in March 1979 [U1]. While it is very difficult to estimate the fraction of the Windscale radionuclide core inventory that was released to the atmosphere, it has been estimated that that accident released twice the amount of noble gases that was released at Chernobyl, but 2,000 times less ^{131}I and ^{137}Cs [D5]. The Three Mile Island accident released approximately 2% as much noble gases and 0.00002% as much ^{131}I as the Chernobyl accident.

27. From the composition of air samples taken during the Chernobyl release and the total release-rate data, tentative isotopic release rates for individual radionuclides were constructed [I1]. These generally follow the pattern of the total release rate (Figure II), with decreasing release rates initially and increasing rates until the end of the release period. Additional information has been presented [I3] that shows changing isotopic ratios during the release period (Table 2): for example, variable ^{131}I relative to ^{137}Cs in initial emissions and higher ^{103}Ru, ^{106}Ru, ^{141}Ce and ^{144}Ce in later emissions. The changing physical conditions and, possibly, the involvement of fuel of varying burn-up may explain these features. The chemical form of the materials released as aerosols was quite variable. The particle size of aerosols ranged from less than 1 micrometre to tens of micrometres.

28. For the region around the Chernobyl site detailed maps of radionuclide deposition could be drawn in 1986 and 1987 based on measurements of external dose rates and analyses of environmental samples [A9, I12]. The pattern of deposition within other regions of the Soviet Union was also established through gamma dose-rate measurements from aircraft and analyses of the radionuclide content of soil samples taken at a limited number of locations. These procedures enabled an estimate to be made of the total amounts of radionuclides deposited in the Soviet Union. This estimate was used in deriving the total amount of

radionuclides released, as mentioned before. The proportions of the core inventory deposited at various distances from Chernobyl were estimated to be as follows [I1]:

On-site:	0.3-0.5%
0-20 km:	1.5-2%
Beyond 20 km:	1-1.5%

2. Atmospheric transport

29. At the time of the accident, surface winds at the Chernobyl site were very weak and variable in direction. However, at 1,500 m altitude the winds were 8-10 m/s from the south-east. The initial explosions and heat from the fire carried some of the radioactive materials to this height, where they were transported by the stream flow along the western parts of the USSR toward Finland and Sweden. The arrival of radioactive materials outside the USSR was first noted in Sweden on 27 April [D1]. The transit time of 36 hours over a distance of some 1,200 km indicates transfer at an average wind speed of 10 m/s.

30. According to aircraft measurements within the USSR, the plume height exceeded 1,200 m on 27 April, with the maximum radiation occurring at 600 m [I4]. On subsequent days, the plume height did not exceed 200-400 m. The volatile elements iodine and caesium, were detectable at greater altitudes (6-9 km), with traces also in the lower stratosphere [J1]. The refractory elements, such as cerium, zirconium, neptunium and strontium, were for the most part of significance only in local deposition within the USSR [I3, I4].

31. Changing meteorological conditions, with winds of different directions at various altitudes, and continuing releases over a 10-day period resulted in a very complex dispersion pattern. The plumes of contaminated air that spread over Europe are described in a highly simplified manner in Figure III, along with the reported initial arrival times of radioactive material.

32. The initial plume, depicted as A in Figure III, arrived on 27 April in Sweden and Finland. A portion of this plume at lower altitude was directed southward to Poland and the German Democratic Republic. Other eastern and central European countries became

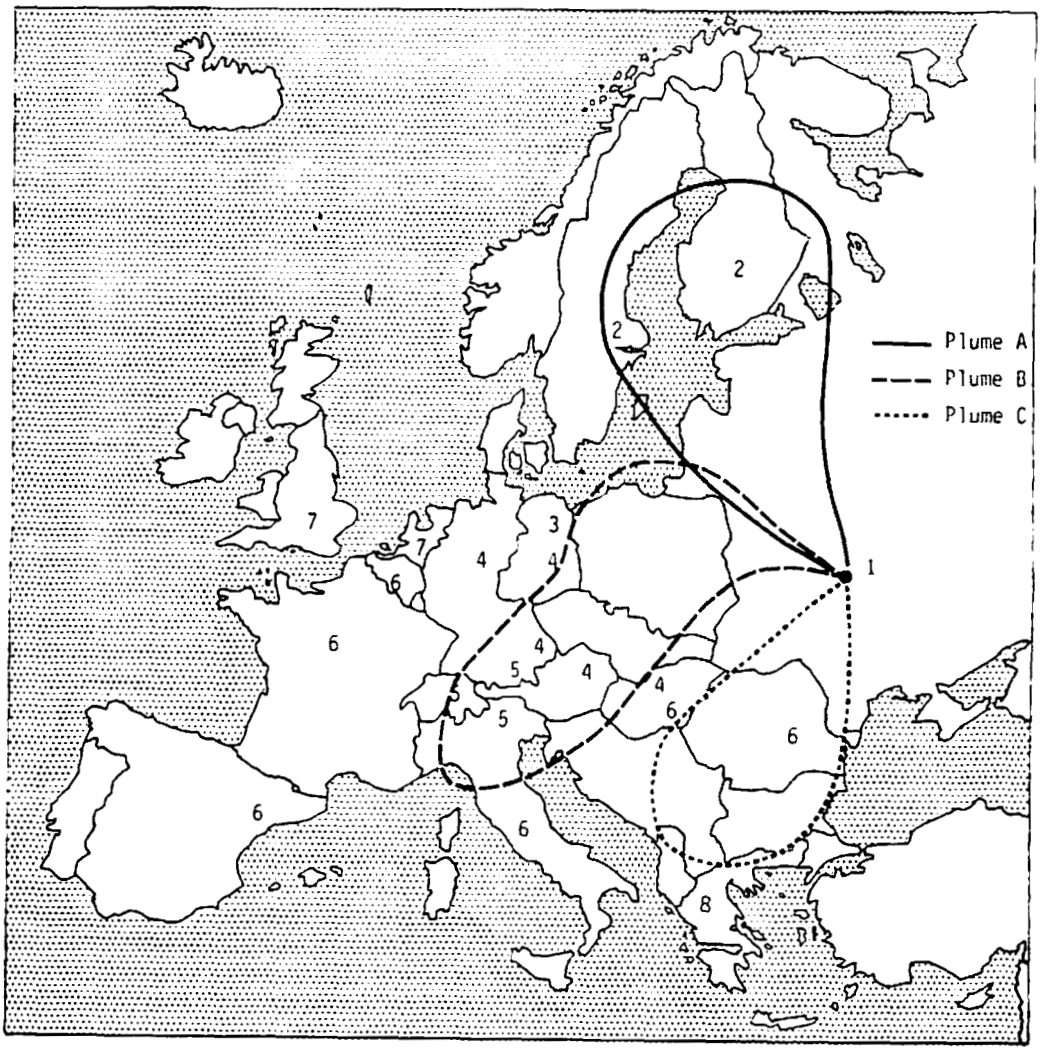

Figure III. Descriptive plume behaviour and reported initial arrival times of detectable activity in air. Plumes A, B, and C correspond to air mass movements originating from Chernobyl on 26 April, 27-28 April, and 29-30 April, respectively. The numbers 1 to 8 indicate initial arrival times: 1 (26 April), 2 (27 April), 3 (28 April), 4 (29 April), 5 (30 April), 6 (1 May), 7 (2 May), 8 (3 May).

affected on 29 and 30 April (plume B). Activity in air entered north-east Italy during 30 April (also plume B). Central and southern Italy first had evidence of the plume's arrival during the following day. Switzerland reported its first arrival on 30 April. The generally northward flow air across western Europe brought detectable activity to eastern France, Belgium and the Netherlands on 1 May and to the United Kingdom on 2 May. Contaminated air (plume C) arrived in Greece on 2 May in the north and on 3 May in the south [G2]. Airborne activity was also reported in Israel, Kuwait and Turkey in early May [K1, S6, T1].

33. Long-range atmospheric transport spread the released activity throughout the northern hemisphere. Reported initial arrival times were 2 May in Japan, 4 May in China, 5 May in India, and 5-6 May in Canada and the United States [B1, C7, L2, L6, N4]. The simultaneous arrival at both western and eastern sites in Canada and the United States suggests a large-scale vertical and horizontal mixing over wide areas [L2, R8]. No airborne activity from Chernobyl has been reported in the southern hemisphere.

C. EMERGENCY MEASURES

34. After the accident, the first emergency measures taken at the nuclear station were fire-fighting and short-term operations to stabilize the reactor. During the night of 25-26 April 1986, 176 reactor operational staff and workers from different departments and maintenance services were on duty at stages one and two (Units 1-4) of the nuclear power station. In addition, 268 builders and assemblers were at work on the night shift at the construction site of the third stage.

35. Of the on-site personnel and fire-fighters, about 300 had to be hospitalized for burns and the diagnosis of possible radiation injuries. These individuals were observed and given care and, if necessary, specialized treatment. The short-term effects and treatment of radiation injuries caused by the accident are discussed in the Appendix to Annex G, "Early effects in man of high doses of radiation".

36. A system of meteorological and radiological monitoring was organized to survey the contamination levels in the surrounding area. Aerial radiological monitoring was carried out by aircraft and helicopters equipped with air samplers and radiation-detection instruments. On the morning of 26 April, people in the town of Pripyat were instructed to remain indoors and to keep their windows and doors shut. Schools and kindergartens were closed. Late at night on 26 April, radiation levels in Pripyat started rising, reaching about 10 mSv/h on 27 April. It soon became apparent that both the lower intervention level for evacuation (250 mSv whole-body dose) and eventually even the upper intervention level (750 mSv whole-body dose) could be exceeded if the population remained in their homes and no other countermeasures were taken. The evacuation of Pripyat started on the morning of 27 April, after safe evacuation routes had been established on the basis of the first results of radiological monitoring. Provisions were made for decontaminating people's skin and, in some cases, for changing their clothing.

37. In view of the duration of the release of radioactive gases and aerosols from the damaged reactor, it was decided that the accident zone should be further evacuated. As a result of this decision, over 88,000 people, including 21,000 children, were evacuated from the Kiev region and a further 25,000 people, including 6,000 children, were evacuated from the Gomel region of Byelorussia. After the radiation situation had been verified, about 1,000 people were evacuated from the Zhitomir region in the Ukraine and a similar number from the Bryansk region in the RSFSR. The total number of evacuees rose to 115,000. All of these people were medically examined and resettled in neighbouring districts [A9, I12].

38. To prevent the iodine radioisotopes (mostly ^{131}I) present in the plume from accumulating in the thyroid, potassium iodide preparations were distributed to the population in the surrounding zone starting on the morning of 26 April. During the following days, iodine prophylactics were given to 5.4 million people in the USSR, including 1.7 million children [I12, I16].

39. Some tens of thousands of cattle also had to be removed from the contaminated area. Measures were taken to prevent or reduce the contamination of water bodies and ground-water supplies. The extensive environmental radiological monitoring that took place from the very beginning revealed many foodstuffs had been contaminated. On the basis of derived intervention levels for the most important items in the diet, the consumption of locally produced milk and other foodstuffs was banned over a considerable area [I12].

40. According to measured levels of contamination, the area within a 30-km radius of the reactor was divided into three zones: (a) a zone of some 4-5 km around the plant, where no re-entry of the general population is foreseeable in the near future and where no operations other than those required at the installation will be permitted; (b) a 5-10 km zone, where partial re-entry and special operations may be allowed after some time; and (c) a 10-30 km zone, where the population may eventually be allowed to re-enter and agricultural activities may be resumed, subject to strict radiological surveillance. Personnel and vehicles are being controlled at the zone boundaries to reduce the spread of contamination.

41. Great effort has been devoted to decontaminating off-site areas. In a 7,000 km^2 area surrounding the reactor, houses and, particularly, public buildings (schools, nurseries, etc.) were repeatedly treated. Houses that could not be brought to acceptable levels and contaminated, old buildings of low value were dismantled and buried. Roads and other contaminated surfaces were covered with asphalt, gravel, broken stone, sand or clean soil, which brought about 10- to 100-fold decreases in gamma dose rates. In contaminated agricultural areas, deeper ploughing was carried out and more mineral fertilizers were added. Grasslands and pastures were also ploughed and reseeded.

All of these measures substantially reduced radionuclide transfers and radiation levels.

42. In many countries the countermeasures taken immediately after the accident were effective in reducing individual and collective doses. Thyroid dose equivalents were reduced by 80-90% in the most contaminated region of the USSR. Estimates of the effectiveness of the ^{137}Cs countermeasures in that country varied between 20% and 90%, depending on the level of contamination. In Austria, the Federal Republic of Germany and Norway, doses were reduced between 30% and 50% by countermeasures, and in other European countries they were reduced somewhat less [N5]. These countermeasures were taken into account in the Committee's assessment, as far as possible, by considering the reduction in intakes of contaminated foods.

II. METHODOLOGY FOR THE DOSE ASSESSMENT

A. SCOPE AND APPROACH

43. Since the accident, a sufficient number of measurements have been made to show the basic features to consider in a dosimetric evaluation. The main pathways and radionuclides contributing to doses are external irradiation from deposited radioactive materials (primarily ^{137}Cs in the longer term) and the dietary ingestion of radionuclides (^{131}I in milk and leafy vegetables during the first month and, after that, ^{134}Cs and ^{137}Cs in foods).

44. The inhomogeneous deposition of dispersed materials makes it necessary to take a regional approach to dose calculation. Enough information is available to calculate doses in the most affected region, which includes most of the European countries (some of these countries were further subdivided). The input values for the calculations make full use of measurement results through the first year following the accident. Thereafter, projections are required to estimate future environmental behaviour, primarily of ^{137}Cs, and the continued contribution to dose for a few decades. These projections were made on the basis of long-term observations of global fallout from nuclear weapons testing.

45. It may be instructive to consider the differences between this dose assessment and the previous UNSCEAR dose assessments carried out in connection with nuclear fallout or routine, low-level releases from nuclear fuel-cycle installations; namely, that (a) much of the radioactive debris from nuclear weapons tests in the atmosphere was injected into the stratosphere, from which altitude there was rather more uniform hemispheric deposition over the course of several years. Doses could be assessed on the basis of a latitudinal deposition distribution derived from a relatively small number of measurements and on the basis of transfer factors inferred from measurements in only a few countries. Representative rather than comprehensive results were required. Short-term deposition (local fallout) was largely ignored; its distribution was very uneven and its contributions to the total collective dose commitments were small, and (b) following releases from nuclear installations, environmental concentrations and body burdens are often below the detection limits of the measuring instruments. Doses are calculated using generic source terms characteristic of the particular type of nuclear installation under consideration and using environmental transfer models, the parameter values of which are largely independent of the location of the installation.

46. In the case of the accident at Chernobyl, a different set of conditions prevailed: (a) the release was into the troposphere and took place from a single location at a specific time of year; (b) even so, the duration of the release over several days, the large size of the affected region and changing weather throughout the region resulted in a locally varying deposition pattern; (c) the accident occurred at different stages in the agricultural growing season: in the north of Europe, the season had not yet begun, in the south it was already under way; (d) protective measures varied from country to country; (e) a large number of environmental measurements were made available, providing input data for comprehensive dose assessments.

47. In these circumstances, UNSCEAR was able to perform its dose assessment for the Chernobyl accident in some detail, accounting for regional variabilities but applying uniform calculational methods to achieve comparability of results between countries. The Committee relied as much as possible on measured results and used a general model to project the dose commitment.

48. This report includes estimates of average doses to populations of countries. Occupational exposures are not included, because dose information for workers participating in the restoration work in the USSR is not yet available.

1. Geographic coverage

49. There are practical reasons for considering countries as the basic geographic units: most measurements have been co-ordinated and averaged country by country and much of the secondary data, such as population, food production and consumption, is available only on a similar basis. This approach also allows the Committee to compare its calculations of first-year dose equivalents with the calculations of the individual countries. Dose commitments are then calculated on a regional basis.

50. Although it was the countries of Europe that were most affected by the Chernobyl accident, the radioactive materials became dispersed throughout the northern hemisphere, and so the dose assessment considers the entire hemisphere. It is well established that, for an atmospheric release into the lower troposphere, there is very little transfer of particles from one hemisphere to another. Although there may be some transfer of dose to southern hemisphere

residents through imported foods, this increment in the collective dose equivalent can be accounted for by considering total production as well as consumption of foods in the affected regions.

51. Because they were closest to the release point, the countries of northern, eastern and western Europe and the western part of the USSR require the most detailed consideration. It was in these places that deposition was greatest and most non-uniform. In countries further removed from the release point, the more widely dispersed material was deposited with more regional uniformity and was, at any rate, less significant from a dosimetric standpoint.

52. For almost all the countries of eastern and western Europe, enough radiation-monitoring data and other information were available to allow detailed dose calculations for the first year. In so far as was possible, each country was considered as a single geographic unit. However, to avoid averaging wide-ranging dosimetric data, several countries were subdivided. These geographical breakdowns within the various countries of Europe are indicated in Figure IV. For the calculation of dose equivalent commitments, countries were combined into broad geographical regions.

53. In Asia and North America, only low levels of radioactivity could be detected. The approximate dose estimates for some countries in these regions have been extrapolated to obtain estimates for larger geographic areas. Although they were not significantly affected by the airborne transport of radioactive materials from the accident, other developing countries have been concerned about the possible contamination of imported foods. Further, the accident has prompted several countries to engage in activities to evaluate and assess immediate and late effects of this and other possible accidents. It is clear that international agencies must become involved in the training of scientists and technicians; the procurement of equipment; the development of simplified techniques for measurement and assessment; and procedures on which to base setting of restrictions on imports of contaminated foods.

2. Pathways

54. There are two primary pathways to be considered in this dose assessment: (a) external irradiation from radioactive materials deposited on the ground and (b) ingestion of contaminated foodstuffs. Two secondary pathways have been considered as well,

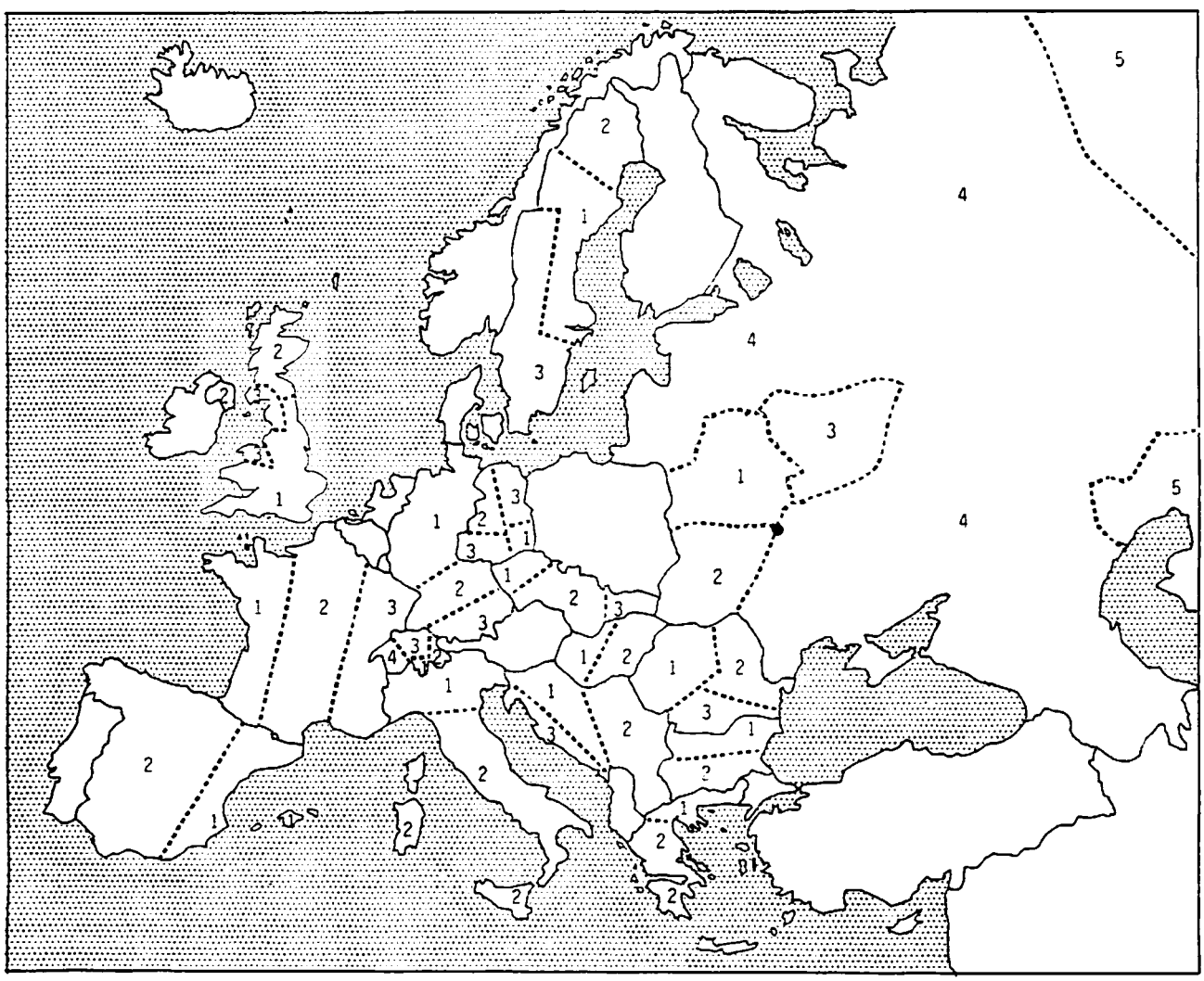

Figure IV. Division of Europe by country, or by subregions within countries, for purpose of the dose assessment.

since the concentrations of radionuclides in air, on which they depend, have been generally available: (a) external irradiation from radioactive materials present in the cloud, referred to as "cloud gamma", and (b) inhalation of radionuclides during passage of the cloud. The inhalation pathway can, in fact, be important right after an accident and if people are subsequently evacuated and received no further exposure, it can turn out to have been the most important pathway.

55. Some data available from different countries show a small amount of resuspension of the deposited material that led to measurable concentrations in air some weeks or months after the accident. The contribution of resuspension to further inhalation doses is considered to be small in comparison to that of the other exposure pathways.

56. The pathways of cloud-gamma exposure and inhalation of radionuclides are effective only for the short period before the airborne material has been deposited. Transfers along the two primary pathways continue for a length of time that depends on the half-lives of the radionuclides, some tens of days for ^{131}I, for example, and some tens of years for ^{137}Cs.

57. For the ingestion pathway, only the basic food items have been considered: milk products, grain products, leafy vegetables, other vegetables and fruit, and meat. Those five categories are sufficient to account for the food ingestion of most individuals. Radionuclide uptakes in other foods, such as mushrooms and lake fish, have been noted. Although these other foods may be important for some consumers, they, like other possible, but minor, pathways, have little effect on collective dose estimates.

3. Radionuclides considered

58. Only ^{131}I, ^{134}Cs and ^{137}Cs, the most important contributors to the total dose, have been considered systematically by the various countries. Other radionuclides (^{95}Zr, ^{103}Ru, ^{106}Ru, ^{132}Te, ^{140}Ba and ^{141}Ce) were reported in air or deposition. Several of the latter were important short-term contributors to external irradiation from deposited material; when not measured directly, they may be accounted for by scaling to ^{137}Cs or ^{131}I deposition. The long-lived radionuclides ^{3}H, ^{14}C, ^{85}Kr and ^{129}I are discussed later. They, too, are but minor contributors to the total dose.

4. Doses evaluated

59. The assessment of doses has two components: (a) the committed dose equivalents resulting from exposures and intakes during the first year following the accident and (b) the collective effective dose equivalent commitment due to the accident. In assessed countries and subregions, estimates are made of the first-year effective dose equivalent, i.e. the dose received in the first year from external irradiation and the dose committed from first-year inhalation and ingestion of radioactive materials. First-year dose equivalents to the thyroid of adults and one-year-old infants are also estimated.

60. The evaluations of dose for the first year reflect as nearly as possible the prevailing conditions, taking into account not only measured values but also shielding and occupancy factors and protective measures. The recently observed and reported reduction in exposure levels in urban areas as a result of runoff has been incorporated into the dose models. Other factors are introduced and described along with the calculational methods.

61. The second component of the dose assessment is the collective effective dose equivalent commitment, which requires projection of doses to be received in the future from deposited materials. For this purpose the models developed by the Committee for estimating dose commitments from fallout have been used. Because the parameters for these models were obtained by averaging results from widely separated regions, wider groupings of countries have been selected to reflect regional deposition patterns. The dose commitments have been evaluated for each large region and used for calculating the collective dose commitment. The estimates are based on both consumption and production of foods.

B. CALCULATIONAL METHODS FOR FIRST-YEAR DOSES

62. For the most part, the calculations simply involve multiplying integrated concentrations by dose factors, with reduction factors taken into account. The integrated concentrations in food are derived, where possible, from measurements through the first year following the accident. To supply missing data, use is made of ratios to other measurements or to "default" values, which are values derived from measurements at other sites or averaged from representative results from neighbouring locations. The methods for each pathway are described below.

1. External irradiation during cloud passage

63. During a very brief period, usually only hours but sometimes a few days, the passing cloud of contaminated air exposes people to external irradiation. This exposure is referred to as cloud-gamma irradiation. Although this exposure rate could in theory be measured directly, in practice it is not possible to distinguish this component from radiation caused by deposited activity on the ground. The doses from cloud-gamma exposure can be easily calculated from measured air concentrations. The equation for radionuclide i is

$$H_{E,c}(i) = C_a^*(i)\,\Phi_c(i)\,(1 - F_o) + C_a^*(i)\,\Phi_c(i)\,F_o\,F_s$$

where $H_{E,c}(i)$ is the cloud-gamma effective dose equivalent (Sv); $C_a^*(i)$ is the integrated concentration in outdoor air (Bq d/m^3); Φ_c (i) is the effective dose equivalent factor per unit integrated air concentration

(Sv per Bq d/m^3); F_o is the indoor occupancy factor (the fractional time spent indoors); and F_s is the building shielding factor (the ratio of indoor to outdoor dose rates).

64. The first term in the equation is the outdoor component of effective dose equivalent and the second term is the indoor component. An additional small component of dose from contaminated air indoors has been neglected in this calculation. The effective dose equivalent factors have been derived for uniform semi-infinite cloud geometry. A list of effective dose equivalent factors is given in Table 3. The same values are assumed to apply to both infants and adults.

65. For the calculations here, an indoor occupancy factor of 0.8 and a building shielding factor of 0.2 have been used for all countries. The values of these factors had been previously used by the Committee [U1, U2]. It is to be noted, however, that measurements as well as calculations of the shielding factor afforded by buildings show a large range of variation depending on the kind of building: from 0.01 to 0.1 for multi-storey buildings and from 0.1 to 0.7 for single-family houses in Sweden [C25], while in Norway the mean shielding factor of houses was reported as 0.5 during the first month and 0.29 during the sixth month following the accident [S14]. For typical European houses, calculations for ^{137}Cs deposition yield values of 0.44, 0.084, and 0.0063 for the ground floors of prefabricated, semi-detached, and multi-storey houses, respectively [M8].

66. To calculate cloud-gamma (and also inhalation) doses, it is necessary to know the integrated concentrations in air of many short-lived radionuclides. In some countries, complete data were available. In others, only one or a few radionuclides were reported, so concentrations of other radionuclides were inferred from ratios measured in nearby countries. In a few cases, no measured air concentrations were available, so the integrated air concentration of ^{137}Cs was inferred from its ground-deposition density and a nominal quotient of ground deposition to integrated air concentration of 1,000 m/d; the integrated concentrations of other radionuclides were then inferred from ratios to ^{137}Cs measured at nearby locations.

2. Inhalation

67. Contaminated air is inhaled during the short time that the radioactive materials remain airborne. This is a straightforward calculation from measured integrated concentrations in air. The equation for radionuclide i is:

$$H_{E,h}(i) = C_a^*(i)\ B\ \Phi_h(i)\ (1 - F_o) + C_a^*(i)\ B\ \Phi_h(i)\ F_o\ F_r$$

where $H_{E,h}(i)$ is the inhalation effective dose equivalent (Sv); $C_a^*(i)$ is the integrated concentration in outdoor air (Bq d/m^3); B is the breathing rate (m^3/d); $\Phi_h(i)$ is the dose per unit intake from inhalation (Sv/Bq); F_o is the indoor occupancy factor; and F_r is the indoor air concentration reduction factor (the ratio of indoor to outdoor air concentrations).

68. The first term is the outdoor component and the second term is the indoor component. The breathing rates are taken to be 22 m^3/d for adults and 3.8 m^3/d for infants [I6]. Indoor occupancy is the same as in the previous calculation. Air concentrations are assumed to be lower indoors due to filtration effects. For all countries, the value of the indoor air concentration reduction factor is taken to be 0.3. Experiments in Finland and Norway showed a range of values, from 0.23 to 0.47, for this factor [C23]; in Denmark they ranged from 0.1 to 0.5 [R9]. Calculations have been made both for the thyroid and for the effective dose equivalents. This calculation also depends upon data of integrated concentration in air with ^{131}I being of particular importance. Such data were inferred where needed as discussed under the section above. Dose equivalent factors are listed in Table 4.

3. External irradiation from deposited material

69. External irradiation from radioactive materials deposited on the ground makes a significant contribution to the total dose equivalent. During the first month after deposition, a number of short-lived emitters, including ^{132}Te, ^{132}I, ^{131}I, ^{140}Ba, ^{140}La and ^{136}Cs, were important components of the total external gamma exposure rate. For several months, ^{103}Ru and ^{106}Ru made contributions, but since then only ^{134}Cs and ^{137}Cs have been of significance. External gamma exposure rates will remain elevated for some years due to ^{134}Cs and for some tens of years due to ^{137}Cs.

70. Calculation of the effective dose equivalent from external irradiation from deposited material proceeds in two steps: the exposure in the first month is considered separately from exposure in subsequent months.

(a) During the first month

71. The outdoor exposure X_1 (C/kg) during the first month was assessed by four different methods, with the choice dependent upon the data available. If continuous or daily data were provided, the exposure rates were integrated. If incomplete data were provided, an attempt was made to fit a power function of the form at^b to the data, where t is time (days) and a and b are constants to be determined. X_1 is then the integral of this function from arrival day 1 to day 30.

72. If measurements of external gamma-exposure rate were not available, two approaches were used. If data on the ground deposition of the radionuclides were provided, the exposure rate from each was computed using the factors published by Beck [B10] for a relaxation depth of 0.1 cm. The term relaxation depth follows from the assumption that the activity mass concentration S(z) of a radionuclide decreases exponentially with depth z in soil:

$$S(z) = S(0)e^{-\alpha z}$$

and the relaxation depth is defined by α^{-1}. In this case, X_1 was evaluated as the sum of the integrated exposure rate from each radionuclide.

73. In several cases, only data on the deposition of ^{137}Cs were available, and X_1 was evaluated on the basis of the relationship of the exposure to ^{137}Cs deposition density as measured at a specific location, e.g., Neuherberg, Federal Republic of Germany [G1].

74. The effective dose equivalent during the first month was calculated from X_1 by:

$$H_{E,e1} = AX_1(1 - F_o) + AX_1F_oF_s$$

where $H_{E,e1}$ is the effective dose equivalent from external exposure during the first month (Sv), A is the conversion factor (23.6 Sv per C/kg, i.e., 33.7 Gy per C/kg × 0.7 Sv/Gy), F_o is the indoor occupancy factor and F_s is the building-shielding factor. The values of the latter two factors are 0.8 and 0.2, the same as used for the calculation of effective dose equivalent from cloud-gamma irradiation.

(b) After the first month

75. The calculation of external gamma dose beyond one month is based on the measured total deposition of ^{134}Cs and ^{137}Cs and, although less important, ^{103}Ru, ^{106}Ru and ^{131}I. The conversion factors for long-term deposition to dose rate depend on the penetration of these radionuclides in soil. Change with time is accounted for by using factors appropriate for a relaxation depth of 1 cm during the first year and 3 cm thereafter. The latter value had been previously used by the Committee for its assessment of doses from nuclear weapons fallout [U1, U2].

76. Following the deposition of radioactive material from the Chernobyl accident, several groups observed that the measured external gamma exposure rate decreased more rapidly over urban surfaces than over grass surfaces [J2, K6, S18]. Although varied, these results are consistent with the loss of half of the material with a half time of 7 days and the other half being firmly fixed on urban surfaces. This urban runoff effect has been reflected in this assessment by applying these factors to that portion of a country's population considered to be urban.

77. The equation for the calculation of external gamma effective dose equivalent for the time period between one month and one year for radionuclide i is

$$H_{E,e2}(i) = [F(i)/\lambda(i)]\,[\Phi_{e2}(i)\,(e^{-\lambda(i)1a/12} - e^{-\lambda(i)\,1a})]$$
$$[1 - F_o(1 - F_s)]\,[1 - F_p(1 - F_u)]$$

where $H_{E,e2}(i)$ is the external gamma effective dose equivalent for the time from one month to one year (Sv); F(i) is the deposition density (Bq/m^2); $\Phi_{e2}(i)$ is the deposition density to effective dose equivalent conversion factor during the period between one month and one year (relaxation depth of 1 cm) (Sv per Bq/m^2); $\lambda(i)$ is the radioactive decay constant (a^{-1}); F_p is the urban fraction of a country's population; F_u is the fraction of the deposition that remains fixed on urban surfaces (assumed in this Annex to be equal to 0.5) and F_o and F_s are as previously defined.

78. The equation applies to the period between 30 days and 1 year. The overall reduction for occupancy and shielding of buildings is 0.36 and the reduction for urban areas is 0.75 with the assumed parameters. The proportion of populations living in urban and rural areas is given in national statistical reports. The urban proportion is around 80% in most European countries, according to the various definitions of urban areas. However, as urban populations also include people living in suburban locations, the urban fraction (F_p), for purposes of this calculation, was assumed not to exceed 0.5. Effective dose equivalent conversion factors are listed in Table 5.

79. Data were available from almost all countries in Europe and elsewhere on the deposition of ^{137}Cs. If data were not reported for ^{134}Cs, a measured ratio in air was used, or a nominal ratio of 0.5. Data were also typically available for ^{131}I, but if not, deposition was inferred based on ratios measured on airborne particles or ratios of deposition in nearby countries. Data on ^{103}Ru and ^{106}Ru were available from about half of the countries; if they were not, the calculations were made on the basis of the ratio to ^{137}Cs measured in air or deposition in nearby countries.

4. Ingestion

80. The ingestion of radionuclides in foods is a second primary pathway for radiation doses. As determined by an initial sensitivity analysis, only the radionuclides ^{131}I, ^{134}Cs and ^{137}Cs make significant contributions and need be considered. The dose estimation is based on measured or inferred concentrations during the first year, but projections are required to take account of caesium transfer in future years.

81. The food categories considered include milk and milk products, grain products, leafy vegetables, other vegetables and fruit, and meat. The occurrence of ^{131}I in foods was of significance only for milk and milk products and leafy vegetables, with the exception of high relative values reported for the radish in Japan [N4]. Root vegetables and fruits were, in general, less affected, and they have been considered together. An integrated food concentration (Bq a/kg) has been calculated or inferred for each food category; it is based on all types of individual foods to the extent data were available, weighted by consumption amounts. For example, the concentration for meat was calculated on the weighted average concentration in beef, pork, lamb, poultry, game and fish. Similarly, the concentration in milk products was calculated as a weighted average of the concentration in milk (of cows, sheep and goats), cheese, butter etc.

82. Food consumption by adults has been taken from national estimates or from data tabulated by the United Nations Food and Agriculture Organization [F10]. There are substantial variations in these values from country to country. National consumption estimates for infants were more variable than would be reasonable, probably because different age groups were considered. Accordingly, consumption estimates for infants up to one year old were standardized and used uniformly in calculations for all countries: milk products, 200 kg/a; grain products, 20 kg/a; leafy

vegetables, 5 kg/a; vegetables/fruit, 15 kg/a; and meat, 5 kg/a.

83. Doses from ingestion of contaminated foods are calculated simply as the product of integrated concentrations in foods during the first year (from the beginning of May 1986 to the end of April 1987), consumption amounts and dose equivalent factors. The integrated concentrations are summations of measured values averaged over the regions considered. In some cases, extrapolations were required to complete the full year of data.

84. If countermeasures were known to have been taken in different countries, the effects were included in the integrated concentrations in foods. For example, Austria banned leafy vegetables, so the concentration of ^{131}I in leafy vegetables is given as 0.0 [M3]. In other countries, foods with radionuclide concentrations above certain limits were withheld from markets; any reported concentrations in foods above that limit were therefore, disregarded.

85. Nearly all countries reported measurements of ^{131}I in milk and leafy vegetables. Levels of ^{134}Cs and ^{137}Cs were usually reported for milk and leafy vegetables. The reporting of concentrations in grain, meat and other vegetables and fruits was more limited. Methods of inferring concentration varied depending upon what other data had been reported and the general relationships among food categories deduced previously [U1]. The concentration of ^{134}Cs or ^{137}Cs, if necessary, was typically inferred using a first-year transfer factor. Specific values varied from region to region. As an example, ^{137}Cs in meat was estimated from ^{137}Cs deposition using a first-year transfer factor of 3-4 Bq a/kg per kBq/m^2, in some west European countries; in others, it was inferred from a ratio of integrated concentrations of meat to milk of 2-3. The concentration in other vegetables and fruits was similarly deduced using a transfer factor of 0.8-1.6 Bq a/kg per kBq/m^2 or by using a ratio of 0.3 for integrated concentration relative to milk. Grain presented a special difficulty because measurements were lacking and because some data showed a very strong effect of time of contamination before harvest, as noted earlier by Aarkrog [A4]. A more complete discussion of how concentrations in grain were calculated is provided in the next section.

86. The equation for this part of the ingestion pathway calculation for food category g and radionuclide i is

$$H_{E,gI}(i) = C_g^*(i) I_g \Phi_g(i)$$

where $H_{E,gI}(i)$ is the effective dose equivalent from first-year ingestion of food group g (Sv); $C_g^*(i)$ is the weighted integrated concentration in food group g (Bq a/kg); I_g is the consumption rate for food group g (kg/a); $\Phi_g(i)$ is the effective dose equivalent per unit intake from ingestion (Sv/Bq). Summation is required over the relevant food categories for the total dose equivalent from each radionuclide. Values of the dose factors are listed in Table 6. Specific values of consumption rates are taken as reported by the individual countries or as derived from FAO data [F10].

87. The dose assessment for the first year after the Chernobyl accident depends on the use of measured concentrations of radionuclides in foodstuffs. Such concentrations are assumed to represent consumption-weighted averages for the area concerned. Reliable estimates of such averages depend on systematic sampling plans specially designed for this purpose. For some types of foodstuffs, the prime example being dairy milk, it is relatively easy to achieve reasonably reliable estimates, because a measurement on a single sample can be assumed to typify both a large production area and a large consumer group. For other dietary components, reliable estimates necessitate both large numbers of samples and well-designed sampling plans. This is especially the case when there has been both small-scale and large-scale variability of the deposition density, as was the case after the Chernobyl accident.

88. After the Chernobyl accident, the affected countries started sampling and measurement programmes. These programmes were in many cases control programmes, designed to assure that foodstuffs contaminated above a particular level did not reach consumers. Such programmes are often characterized by a planned or unplanned bias, such that sampling is concentrated in areas where high contamination levels are suspected. The average calculated from such programmes therefore tends to overestimate consumption-weighted averages, and there is little possibility of correcting afterwards for a bias of this kind.

89. For the long-lived caesium isotopes, there will be a time-averaging that results in less variability for contamination levels in such foodstuffs as milk, green vegetables and meat. Since the short half-life of ^{131}I precluded such averaging, the estimated average levels must in many cases be regarded as tentative.

C. CALCULATIONAL METHODS FOR PROJECTED DOSES

1. External irradiation

90. External exposure from radioactive materials deposited on the ground was evaluated by the following equation:

$$H_{E,e3}(i) = [F(i)/\lambda(i)]\,[\Phi_{e3}(i)\, e^{-\lambda(i)Ia}]\,[1 - F_o(1 - F_s)]\,[1 - F_p(1 - F_u)]$$

The symbols were defined in paragraph 77. The deposition density to effective dose equivalent factor, $\Phi_{e3}(i)$, to be used beyond one year after deposition, uses a relaxation depth of 3 cm, as has been assumed previously in UNSCEAR assessments. Values of this factor are listed in Table 5.

2. Ingestion

91. Projections are required to estimate ingestion doses beyond the periods for which measurements are available. Over many years, a deposition-diet transfer model has been developed and used by the Committee

to describe the behaviour of fallout radionuclides, ^{90}Sr and ^{137}Cs, in the environment and to estimate dose equivalent commitments [U1]. The basic transfer relationship for radionuclide i and for food category g of the weighted diet total is:

$$C_g^*(i) = P_{23}(g,i)\,F(i)$$

where $C_g^*(i)$ is the integrated concentration in food over all time (Bq a/kg); $P_{23}(g,i)$ is the transfer factor from deposition density (compartment 2) to food or total diet (compartment 3) (Bq a/kg per Bq/m^2); and F(i) is the total deposition density (Bq/m^2).

92. The values of deposition density and concentrations in food have been determined on an annual basis and the parameters in the transfer function evaluated by regression fitting. The model for the transfer function is

$$P_{23} = b_1 + b_2 + b_3 e^{-\lambda t}$$

where b_1 is the component of first-year transfer; b_2 is the second-year transfer; and $b_3 e^{-\lambda t}$ is the subsequent transfer (the latter accounts for both environmental loss and radioactive decay). This model was developed for the rather more uniform and continuing deposition pattern of radioactive fallout from atmospheric nuclear weapons testing. Thus it is not specifically intended to predict time-integrated concentrations in foods in specific countries for a release such as that which occurred from the Chernobyl reactor. However, in so far as seasonal and local conditions are largely accounted for by direct measurements of the first year, the model may be applied to obtain projected behaviour for the second year and beyond over large areas, such as groups of countries. The part of the transfer function that accounts for the time-integrated concentrations beyond the first year, the second and third terms, is referred to as $P_{23,2+}$:

$$P_{23,2+} = b_2 + b_3 e^{-\lambda t}$$

93. Detailed evaluation of the P_{23} factor for ^{137}Cs for all food categories is available from fallout measurements in Denmark and Argentina, reported in [U1]. A similar analysis has been made for Chicago in the United States [E7]. The values of these parameters are listed in Table 7. The three locations are far apart, and the results show some of the variability that can be expected as a result of different soil types, agricultural practices and other local conditions. These results have been combined and the averaged values of $P_{23,2+}$ used in the dose calculations for all food categories except grain products.

94. A separate assessment is required for grain products, whose contamination has been shown to be very dependent on the maturity of the plant [A4, C13]. Contamination by root uptake is negligible in comparison to contamination by direct deposition, as is generally the case for any vegetable product. Under controlled conditions, the transfer of caesium to grain has been studied in relation to time of harvest [A4]. Uniform deposition to a test area of a barley field three months before harvest resulted in a 100-fold lower concentration in grain than applications two months before harvest. There was little difference in transfer for applications at other times within two months of harvest.

95. Grain is usually harvested in the summer months and later processed into flour and bran or used as animal feed. The transfer factors from grain to bread or other products for human consumption and the composition of grains in the consumed products have been reported for Denmark [A5].

	Transfer from grain to bread	*Percentage of grain consumption*
Rye	1	36
Wheat	0.5	55
Oats	0.5	9

Applying these factors to the measured ^{137}Cs activity mass concentrations in grains harvested in 1986 results in P_{23} transfer factors of 0.5 Bq a/kg per kBq/m^2 in Finland; 0.25 in Norway, 3.3 in Denmark, 4 in France, 4.5 in Czechoslovakia and 16 in Japan. The average P_{23} for grain products delivered after the atmospheric testing of nuclear weapons was 15 Bq a/kg per kBq/m^2 (Table 7). The latitudinal dependence of the Chernobyl contamination reflects the different stages of grain maturity at the time of the accident. Where grain contamination is not reported for a particular country, values of P_{23} for grain products have been assumed to be 0.5 Bq a/kg per kBq/m^2 for latitudes above 55° N, 5 for temperate latitudes (40-55° N), and 20 for latitudes below 40° N. Higher values are not likely because the grain at latitudes below 40° N was about to be harvested when the contamination occurred.

96. Assuming that the grain products derived from a given summer harvest are available from November of that year to November of the following year, the grain contaminated by the deposition in May 1986 can be considered to have been distributed for six months (November to April) during the first year after the accident and for six months during the second year, so that $b_1 = P_{23}/2$ and $P_{23,2+} = P_{23}/2$.

97. Estimates of projected doses from the ingestion pathway are obtained by multiplying the factor $P_{23,2+}$ by the deposition in the region, the consumption rate and the dose per unit intake from ingestion:

$$H_{E,g2}(i) = P_{23,2+}(g,i)F(i)I_g\Phi_g(i)$$

where $H_{E,g2}(i)$ is the effective dose equivalent from ingestion of radionuclide i in food group g beyond the first year (Sv); $P_{23,2+}(g,i)$ is the deposition density to diet transfer factor; F(i) is the total deposition density (Bq/m^2); I_g is the consumption rate (kg/a); and $\Phi_g(i)$ is the effective dose equivalent per unit intake (Sv/Bq).

98. Collective dose estimates are made for each pathway by multiplying doses by the relevant population of each region. For the ingestion pathway two estimates are made: namely, (a) a consumption-based estimate, whereby the intake per individual is multiplied by the number of individuals and (b) a production-based estimate which is derived from the country's total production. The estimates are usually in fairly close agreement, certainly within the uncertainty of the two methods. The production-based estimates account for any additional collective dose outside the country if large amounts of food are exported.

99. Countries were grouped together, and population-weighted values of deposition density and transfer factors were used in evaluating the collective effective dose equivalent commitments.

III. EVALUATED INPUT DATA

100. Following the Chernobyl accident, extensive national monitoring programmes were undertaken to determine the extent and degree of contamination from the radionuclides released and to evaluate the need for countermeasures. Continued measurements in many countries of the environmental levels and of concentrations in the diet and in the human body provide a basis for evaluating the radiation exposures.

101. The material in this chapter is not intended to document the many results obtained; rather, it comprises, in summary form, the representative input data required for the dose calculations. In most cases, these data are the first-year integrated concentrations for each country or subregion. Relationships between integrated quantities have been used to check the consistency of the results and to form the basis for estimates where data are incomplete or missing, as indicated in the previous chapter. The input data used in the dose assessment are presented in tabular form, and measured and inferred data are carefully distinguished.

102. Various types of input data are required to complete the dose calculations. These include non-radiological data, such as population, area, food production and consumption, and radiation data, such as integrated concentrations in air and foods and deposition densities. The values of the non-radiological parameters for each country or subregion are listed in Table 8. Food-production estimates, when not reported directly by countries, were obtained from reports of the Food and Agriculture Organization of the United Nations [F10, F11], adjusted to reflect food-use amounts by accounting for feed and non-food processed amounts. Other sources for non-radiological data included publications of the United Nations and European and other regional publications [E4, E5, E6, P5, U3].

103. It has not been possible to substantiate fully all of the reported radiation measurement results. In selecting representative values for specific regions, considerable care and judgement are required. Although scientists in each country were asked to review the input data, some inconsistencies and questionable values remain. However, these should not affect the more general results of the assessment.

104. The sources of radiological data have been numerous; some of the data was obtained directly from scientists in the relevant countries and some of it came from published reports. The references for the countries are as follows: North Europe: Denmark [A3, R1, R2]; Finland [A8, F1, I14, I15, N6, P1, R3, R7, R10, R11, R12, R13, S22, S23, S24, S25, S26, S27]; Norway [B4, B5, S14, S15, W3]; Sweden [A6, E1, E8, F4, F5, F6, H5, K2, K3, K6, L3, S1, S8, S9, S13]; Central Europe: Austria [A1, B7, D2, F7, K7, M3, O1, S18, S19, S20]; Czechoslovakia [B12, I11, M7, M9]; German Democratic Republic [L1]; Federal Republic of Germany [B13, D4, D6, G1, I10, J5, K4, S2, S16, W2, W4]; Hungary [A2, B9, H1, H4, S7]; Poland [C1, C2]; Romania [R6]; Switzerland [B2, B3, C14, H2, P2, S12, V2, W2]; West Europe: Belgium [C10, G4, S4, S5]; France [C5, C21, C22, D3, L5, S3, S17, S21]; Ireland [C9]; Luxembourg [C10, S4, S5]; Netherlands [C8, C26]; United Kingdom [C3, C11, F2, F3, F12, M2, W1]; South Europe: Bulgaria [C4, P4]; Greece [G2, G3]; Italy [C15, C16, C17, C18, C19, C20, E2, M4, M5, M6, R4, R5]; Portugal [L4]; Spain [C6, G5, G6]; Yugoslavia [F8, F9, I7, I8, J3]; USSR: [A9, I1, I2, I3, I4, I12, I13, I16, P6, U5, U6]; West Asia: Cyprus [C12]; Israel [S6]; Syrian Arab Republic [S11]; Turkey [T1, T2]; East Asia: China [B8, C24, L6]; India [B1]; Japan [A7, N2, N4, S10]; North America: Canada [C7, R8]; United States [D5, E3, U4].

A. AIR

1. Radionuclide composition

105. Radionuclides in air, identified by filter sampling, were predominantly volatile elements (iodine, caesium, tellurium) rather than non-volatile ones. The radionuclides detected by gamma spectrometry included ^{99}Mo, ^{99m}Tc, ^{103}Ru, ^{127}Sb, ^{129}Te, ^{132}Te, ^{131}I, ^{132}I, ^{133}I, ^{134}Cs, ^{136}Cs, ^{137}Cs, ^{140}Ba, and ^{140}La. Some additional radionuclides (^{95}Nb, ^{106}Ru, ^{110m}Ag, ^{125}Sb, ^{129m}Te, ^{141}Ce, ^{144}Ce) could be detected only after the decay of interfering gamma lines.

106. Other radionuclides in air were determined by beta or alpha spectrometry. Strontium radionuclides were present in low concentrations, the ^{137}Cs/^{90}Sr ratio being approximately 110 to 1 as measured at Munich-Neuherberg and the ^{89}Sr/^{90}Sr ratio about 10 to 1 (on 1 May). Transuranic elements were estimated to be present on 1 May at concentrations of 130 μBq/m^3 (^{238}Pu), 200 μBq/m^3 (^{239,240}Pu) and 1,500 μBq/m^3 (^{242}Cm) [W4]. Other radionuclides assumed to have been present but which were below the detection limits were ^{129}I and ^{14}C [W4]. The noble gases ^{85}Kr and ^{133}Xe were detectable in air, as was ^{3}H in rain water.

107. The composition of iodine activity in air at the Munich site on initial arrival was found to be 40% aerosol form, 35% elemental gaseous form and 25% organically bound; however, these fractions changed somewhat in subsequent days as rainfall depleted the aerosol and elemental forms more than the organic form (Figure V) [W4]. The particulate iodine fraction measured at Nurmijärvi in Finland on 28 April was 15% [S7] in a sample collected between 29 April and 2 May and 3-24% in samples collected through June [S1]. Other determinations were 33% in Belgium on 2 May [S4], 29-31% at two sites in Hungary on 2 and 4 May [H1], 50% on 29 April and about 33% on following days in Austria [A1], 20% on 4 May and decreasing to 10% thereafter in Switzerland [H2], 25% in the United Kingdom during 7-12 May [C3], about 33% in China on 4-5 May [L6] and 30% on 5-6 May

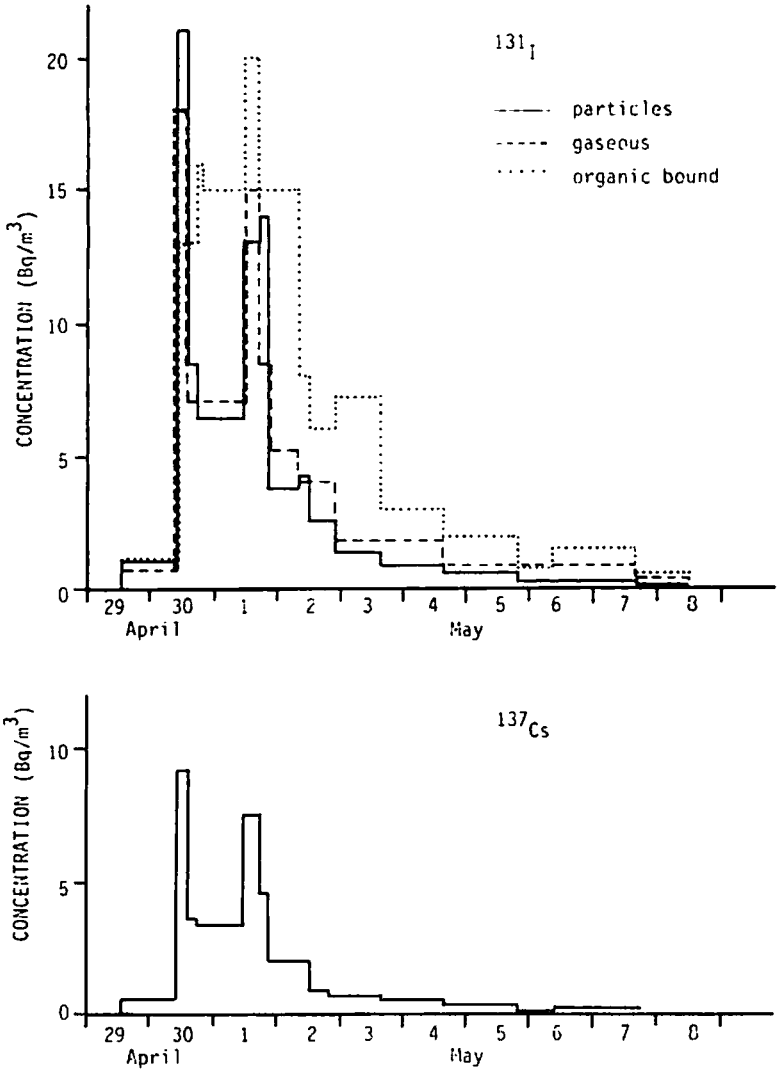

Figure V. Measured concentrations of iodine-131 and caesium-137 in air at Munich-Neuherberg, Federal Republic of Germany. [W4]

in Japan [A7]. Over the monitoring period shown in Figure V, the integrated concentration of ^{131}I was 23% aerosol, 27% gaseous and 50% organically bound. Approximately similar results were obtained for ^{133}I. Ninety-eight per cent of ^{132}Te was associated with particles, as was 65% of its daughter ^{132}I. Of the remaining ^{132}I, 30% was gaseous and 5% organically bound.

2. Concentrations in air

108. The first arrival of contaminated air at the affected places usually brought the peak concentrations of radionuclides in air. The continuing releases from the reactor and the complex air movements often caused secondary peaks on subsequent days, as illustrated in Figure V. The integrated concentrations of radionuclides in air for the duration of elevated levels are listed in Table 9.

109. Reported peak concentrations of ^{131}I and ^{137}Cs in air at several locations gives an indication of the levels encountered. For ^{131}I, the peak values were 400 Bq/m³ at the Berezinsky National Park 120 km north-east of Minsk, 300 Bq/m³ at Varyshevka 140 km south-east of Chernobyl [13], 210 Bq/m³ at Helsinki, 170 Bq/m³ at Vienna, 52 Bq/m³ at Munich-Neuherberg, 31 Bq/m³ at Brussels, 2.5 Bq/m³ at Fukui and 0.3 Bq/m³ at Beijing. For ^{137}Cs, the peak values were 12 Bq/m³ at Helsinki and Berlin, 9.6 Bq/m³ at Vienna, 9 Bq/m³ at Munich-Neuherberg, 6 Bq/m³ at Brussels, 0.04 Bq/m³ in Japan, and 0.02 Bq/m³ at Beijing.

110. Relationships between peak and integrated concentrations of radionuclides in air varied with local meteorological conditions, the sampling times, and whether more than one wave of contaminated air passed the site. The quotients of integrated to peak air concentrations (Bq h/m³ per Bq/m³) were comparable for ^{131}I and ^{137}Cs at individual sites. Values of this quotient were determined to be 15 at Helsinki and Nurmijärvi in Finland, where a sharp peak occurred, 39 at four sites in Germany (West Berlin, Braunschweig, Karlsruhe, Neuherberg), 83 at two sites in Hungary (Budapest, Paks), where three peaks occurred, and about 70 in Japan (Chiba), where a more diffuse peak occurred.

3. Ratios of integrated concentrations

111. The radionuclide composition of contaminated air masses varied depending on when the material had been released from the reactor and the time it took for dispersion to the particular location. The ratios of radionuclides of ruthenium, cerium and caesium suggest that the average irradiation periods of fuel in the reactor had been 400-600 days during the initial release period [C3].

112. The ratios of integrated concentrations in air relative to ^{137}Cs are listed in Table 10. The $^{131}I/^{137}Cs$ ratio was around 25 in Scandinavia and 5-10 in most other European locations. The $^{134}Cs/^{137}Cs$ ratio varied from 0.4 to 0.7 on separate days during May [C3, W4], but the ratio of integrated concentrations was relatively constant, around 0.5, in most places. The ratios of other radionuclides to ^{137}Cs showed some variability, but there were no significant differences between regions. The median values for all countries are indicated in Table 10.

113. The ratios of refractory elements relative to ^{137}Cs differed significantly with distance from the reactor. For example, the ratios of ^{90}Sr, ^{141}Ce and ^{239}Pu to ^{137}Cs in dust samples from within the Soviet Union were 35 times higher than in air samples in western Europe [A4]. The refractory components of the debris and also ^{90}Sr were deposited closer to the accident site than the more volatile constituents.

B. DEPOSITION

1. Deposition of caesium-137

114. The deposition of radioactive materials is associated mainly with rainfall, and since rainfall occurred very sporadically throughout Europe during the passage of the contaminated air, the deposition pattern was very irregular. The highest deposition of ^{137}Cs outside the USSR was recorded in Sweden north of Stockholm, where the deposition density exceeded 85 kBq/m^2. The region of Tessin (Region 1) in Switzerland received 43 kBq/m^2 and southern Bavaria in the Federal Republic of Germany up to 45 kBq/m^2. The provinces of Upper Austria, Salzburg and Carinthia in Austria received estimated average deposition densities of 59, 46 and 33 kBq/m^2, respectively.

115. Average deposition densities for ^{137}Cs of >1 and >5 kBq/m^2 in Europe are illustrated in Figure VI.

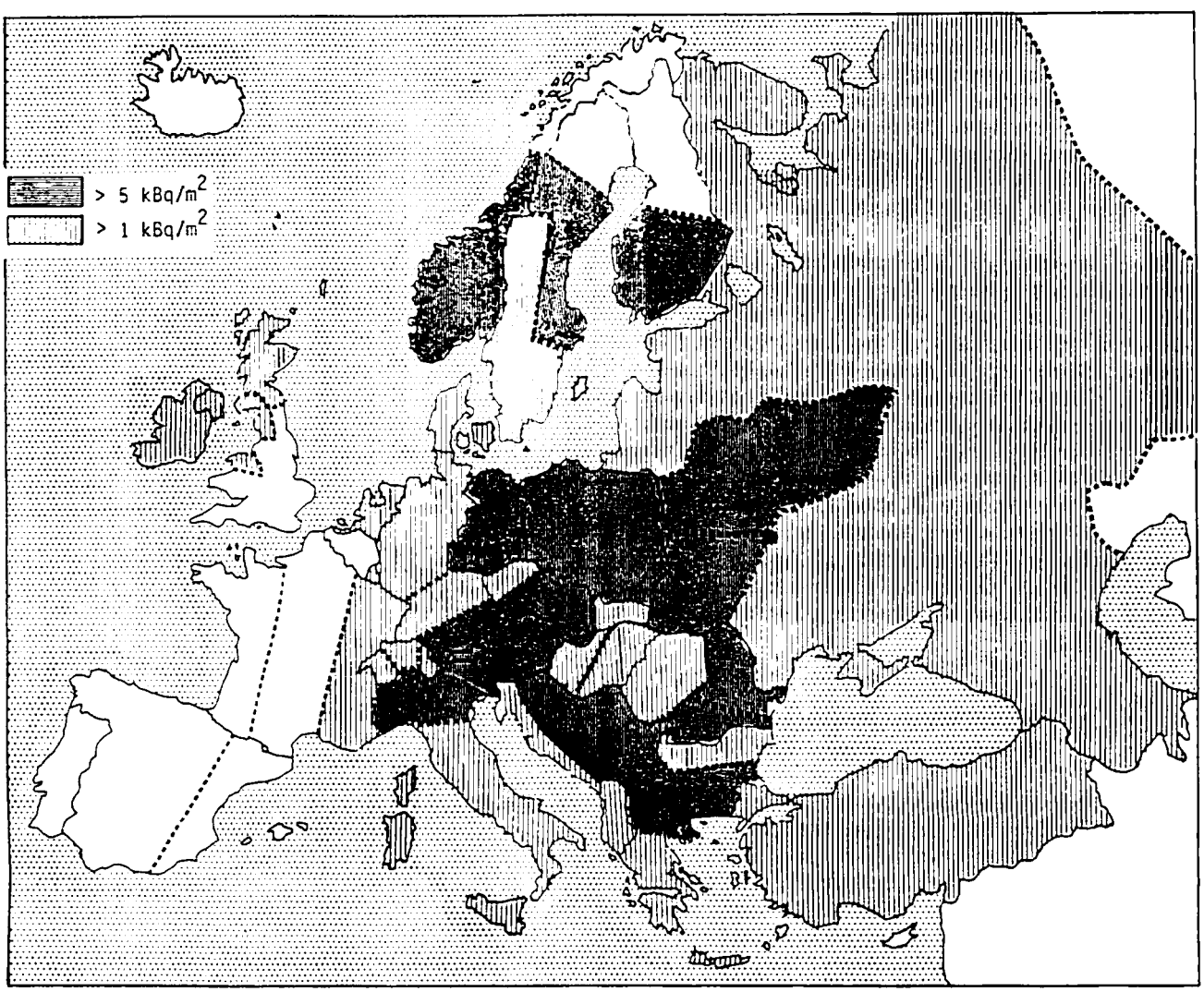

Figure VI. Average caesium-137 deposition density in countries or larger subregions in Europe.

Country-wide deposition densities of >5 kBq/m² for entire country averages are indicated for Austria, German Democratic Republic and Poland. Table 11 lists these average deposition densities.

116. The deposition of ^{137}Cs and other radionuclides outside Europe and the USSR was, accordingly, much less. Representative values of ^{137}Cs deposition densities were 16-300 Bq/m² in Japan, 20-90 Bq/m² in the United States and 20-40 Bq/m² in Canada.

2. Deposition of other radionuclides

117. Radionuclides of importance to the external gamma-irradiation dose from deposited materials beyond the first month include ^{137}Cs, ^{134}Cs, ^{106}Ru and ^{103}Ru. The deposition of ^{131}I, ^{134}Cs and ^{137}Cs is of importance in determining doses from the ingestion pathway. The deposition densities of these radionuclides in different countries and the ratios to ^{137}Cs are given in Table 11. The ratio of ^{131}I to ^{137}Cs is higher in Norway and Sweden than in other countries. The ratios of other radionuclides to ^{137}Cs are relatively uniform. The median ratios of radionuclide deposition to that of ^{137}Cs for all countries are ^{103}Ru, 1.6; ^{106}Ru, 0.5; ^{131}I, 6.2; and ^{134}Cs, 0.5.

118. On an individual measurement basis, there are differences of more than an order of magnitude in the ratios of radionuclide depositions, particularly in the iodine/caesium ratio. There appear to be two reasons for this: the first is the difference in isotopic release at different times during the course of the accident itself; the second is the effect of different rates of precipitation during the passage of the radioactive plume.

119. The release of radionuclides took place over about 10 days and the fire spread through fuel of varying burnup and power rating, resulting in a different relative release of nuclides over the 10-day period. Moreover, the plumes of radioactive material left the Chernobyl site travelling in different directions and were subjected to different meteorological conditions. Some experience showed that where the plume radionuclide content was fairly similar, deposition was related to the intensity of rainfall. Where the plume passed and there was no rainfall, caesium deposition was significantly less than that of iodine. Where it rained through the plume, iodine deposition was higher, and caesium deposition was similar to that of iodine [C11].

3. Quotient of deposition density and integrated air concentration

120. Values of the quotient of the deposition density of a radionuclide to its integrated concentration in air depend on the proportions of wet and dry deposition, as well as on the nature of the particles or vapour and of the receiving surface. Table 12 lists these country average results for ^{137}Cs. The quotients are mostly in the range between 0.6 and 1.2 cm/s. The higher values (those observed, for instance, in Sweden and in Ireland) are strongly influenced by rainfall.

4. External exposure from deposited materials

121. External irradiation from deposited radioactive materials is, in the long term, due primarily to ^{134}Cs and ^{137}Cs. In the first month after initial deposition, however, a number of short-lived emitters, including ^{132}Te, ^{132}I, ^{131}I, ^{140}Ba, ^{140}La, ^{103}Ru and ^{106}Ru, were more significant contributors to the external exposure rate.

122. The exposure rate in air from natural background is about 0.7 pC/(kg s). Off-site external exposure rates in air following the accident were, at maximum, 40-60 pC/(kg s) at Kiev, USSR, 27 in south-west Finland; 12 at Sofia, Bulgaria; 12 at Salzburg, Austria; 7.9 at Munich-Neuherberg and 1.5 at Karlsruhe, Federal Republic of Germany; and 1.4 at Athens, Greece. The component of the external exposure rate attributable to the Chernobyl release was typically lower than the initial value by a factor of 5 by the end of the first month.

123. The exposure rates in air over the first month have been summed in order to evaluate the specific contribution of short-term emitters to effective dose equivalent. These results have been normalized to ^{137}Cs deposition density in Table 13. While the outdoor effective dose equivalent in the first month is not due primarily to ^{137}Cs, the normalized values can be useful for estimating effective dose equivalents where measurements were incomplete or absent. Anomalies in results can point to errors in data. With a few exceptions, the results range from 5 to 40 μSv per kBq/m². The median value is 15 μSv per kBq/m². These results are illustrated in Figure VII.

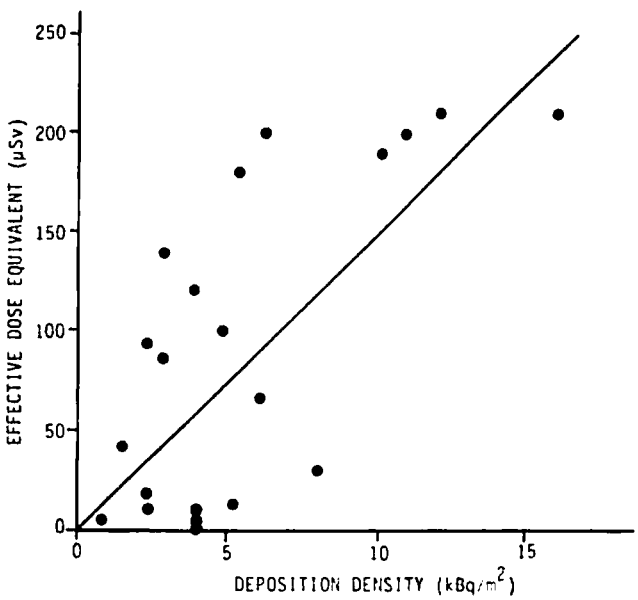

Figure VII. Outdoor effective dose equivalent from external irradiation in the first month after the accident relative to caesium-137 deposition density. The regression line corresponds to 15 μSv per kBq/m².

C. DIET

124. Ingestion of contaminated foods is an important pathway leading to radiation doses from ^{131}I and ^{137}Cs, and all countries paid particular attention to

this pathway following the accident. These radionuclides are rapidly transferred to man through the consumption of milk and leafy vegetables, following their direct deposition on to pasture grass and plants. Other basic foods, such as cereals, root vegetables, fruit and meat, are produced during longer growing periods and are, therefore, not so relevant for short-lived ^{131}I.

125. Numerous measurements are available for ^{131}I and ^{137}Cs concentrations in foods in the first weeks after the accident (data for ^{137}Cs are available for longer periods). The great variability in results reflected the irregular deposition pattern. As indicated in chapter II, attention often centred on the highest levels in foods from areas of greater deposition; however, for the dose assessment, it is representative levels in widely consumed foods that are needed. Assessed results of representative integrated concentrations of ^{131}I and ^{137}Cs in foods during the first year are given in Tables 14 and 15.

126. A degree of comparability between areas can be achieved by considering the integrated concentrations in foods normalized to the deposition densities, and this is the basis for the discussion below. Such relative transfer factors can be used to help establish representative levels in foods from more widely based deposition measurements and to fill in gaps in food data. Of course, the relative transfer depends on local conditions, such as feeding practice during May 1986, so differences in widely separated regions can be expected.

1. Iodine-131 in foods

127. Integrated concentrations of ^{131}I in milk and leafy vegetables relative to ^{131}I deposition density are listed in Table 14. In the case of ^{131}I, there may be some additional variability because of uncertainties in determining total ^{131}I deposition, but a general pattern emerges. In Scandinavia, cows were not yet on pasture at the time of the accident. By keeping cows indoors for some days more, the integrated concentrations of ^{131}I in milk were kept rather low. Some grazing restrictions were also imposed in the Netherlands. In other areas, cows were already on pasture. Normalized transfer of ^{131}I to milk ranges from 0.01 Bq a/kg per kBq/m^2 in Scandinavia to 0.1-1 in central Europe and to 1-3 in some southern and Asian countries. This suggests a latitudinal dependence, which in turn reflects agricultural conditions; this is illustrated in Figure VIII. Only results based largely on measurements are included. The probability distribution of normalized integrated concentrations of ^{131}I in milk is illustrated in Figure IX.

128. At several locations, concentrations of radioactivity in milk were higher for sheep and goats than for cows; this phenomenon is associated with differences in metabolism and feeding habits. For example, during the first week after the accident, the average concentrations of ^{131}I in milk in Greece were 9,000 Bq/l (sheep), 2,000 Bq/l (goats) and 200 Bq/l (cows) [G2]. If a non-typical food makes an important contribution to radionuclide intake in a food category (milk or milk products in this case), the food has been included, weighted by consumption amount.

129. The extent to which ^{131}I is transferred to leafy vegetables depends on the growing season, which was not far advanced in Scandinavia but was well under way in southern Europe. The values of normalized integrated concentrations in Table 14 generally reflect this. The latitudinal dependence of all measured values is illustrated in Figure VIII. The probability distribution is shown in Figure IX.

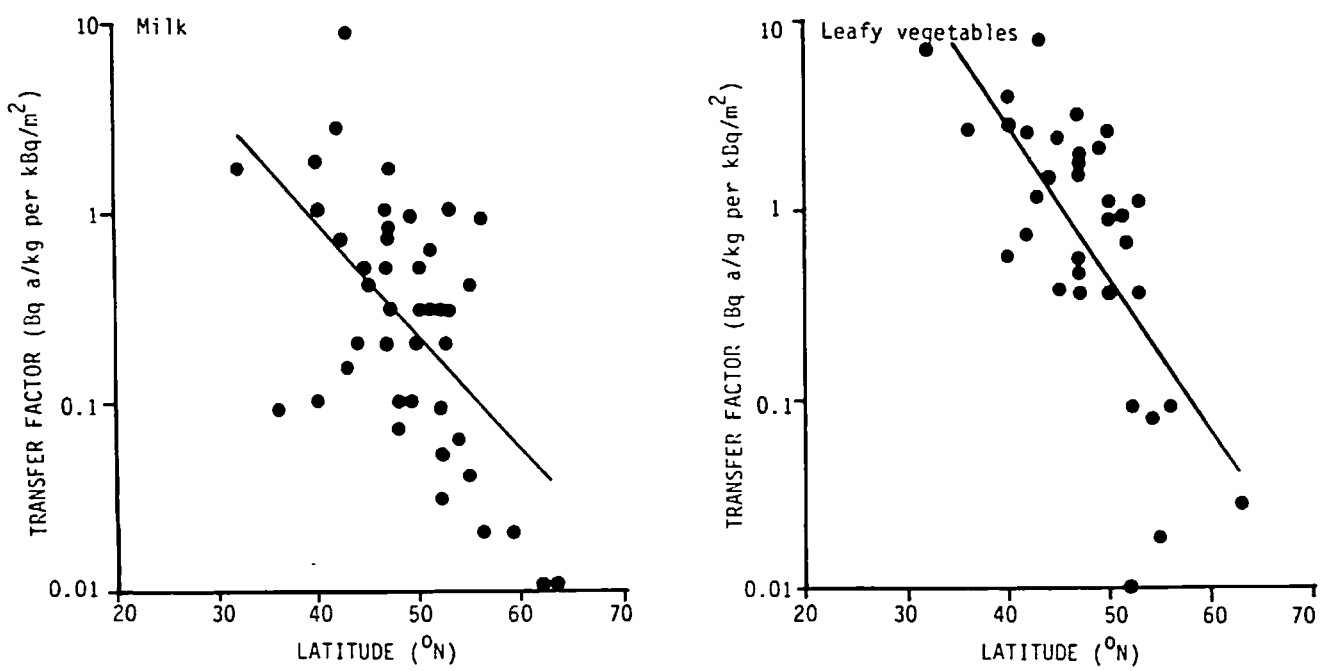

Figure VIII. Integrated concentrations of iodine-131 in milk and leafy vegetables per unit iodine-131 deposition density.

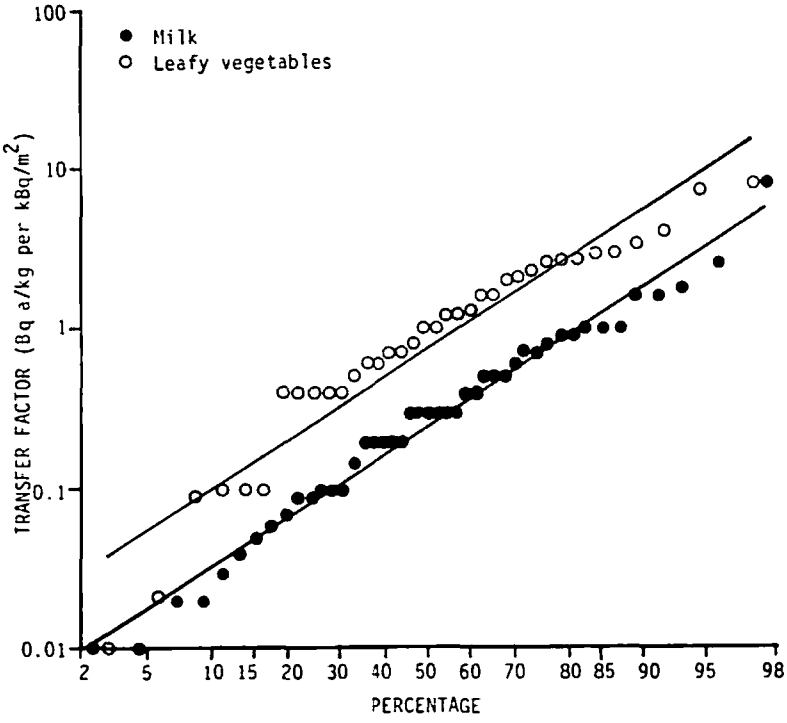

Figure IX. Probability distribution of integrated concentrations of iodine-131 in milk and leafy vegetables per unit iodine-131 deposition density.

130. The ratios of integrated concentrations of ^{131}I in leafy vegetables to those in milk are given in Table 14. This comparison removes uncertainties in ^{131}I deposition, but there is still great variability among regions, suggesting differences in definition of the individual results, the use of milk of different sources, differences in local agricultural practice and the effect of various countermeasures. The majority of values of this ratio lie in the range 1-5 with a median of 2.

2. Caesium-137 in foods

131. The assessed first-year integrated concentrations, normalized to unit deposition density, of ^{137}Cs in the basic food categories are listed in Table 15. These concentrations are based on measurements, as reported and averaged over the countries or subregions. Generally the transfer for all food categories is higher in southern Europe. The latitudinal dependence of integrated concentrations of ^{137}Cs in foods is illustrated in Figure X. The probability distributions of all measured values are shown in Figure XI.

132. The ratios for leafy vegetables/milk and for meat/milk are compared in Table 15. Relative to its concentrations in milk, the integrated concentrations of ^{137}Cs in leafy vegetables are lower by a factor of about 2 and in meat are higher by a factor of about 2, with some deviations.

133. The longer-term monitoring of ^{137}Cs in milk from a dairy farm in the south-eastern part of the Federal Republic of Germany [J5] gave the results shown in Figure XII. Concentrations of ^{137}Cs in milk decreased through the summer of 1986, primarily because the ^{137}Cs was diluted in pasture grass of fresh growth. Increases later in the year were due to the use of animal feeds produced earlier in the year. These changes can be adequately modelled by an appropriate choice of parameters [J5]. Similar variations have been noted elsewhere. Also shown in Figure XII is the country-wide average concentration of ^{137}Cs in milk in Finland [R3]. The initial peak was relatively small and occurred a few weeks after the accident because the cows had initially been off pasture; also, the variability with time was less marked, presumably because the data came from wider-ranging samples.

134. Country-wide monitoring results for ^{137}Cs in meat in Finland are shown in Figure XIII. For reference, the concentrations in milk are also shown. The curve labelled "average meat" is weighted to reflect average consumption of three parts pork for every two parts beef. A beef/milk ratio of about 4 is seen to prevail and an average meat/milk ratio of about 2, as referred to in paragraph 132. Owing to differences in feed sources, the concentrations of ^{137}Cs were generally lowest in pork and poultry, higher in beef and lamb and highest in game.

135. Some foodstuffs that are consumed in small amounts by most people or in large amounts by relatively few people had, on average, much higher activity mass concentrations of ^{137}Cs than the foods presented in Table 15. Foods that should be mentioned in this regard are reindeer meat, mushrooms and lake fish: (a) the feeding habits of reindeer (consuming lichens) lead to exceptionally high levels of ^{137}Cs, as was observed in the 1960s following atmospheric nuclear testing. After the accident, a large fraction of the reindeer in Sweden had ^{137}Cs levels of more than 10,000 Bq/kg [S1]; (b) enhanced levels of ^{137}Cs have been found in mushrooms, although there was consider-

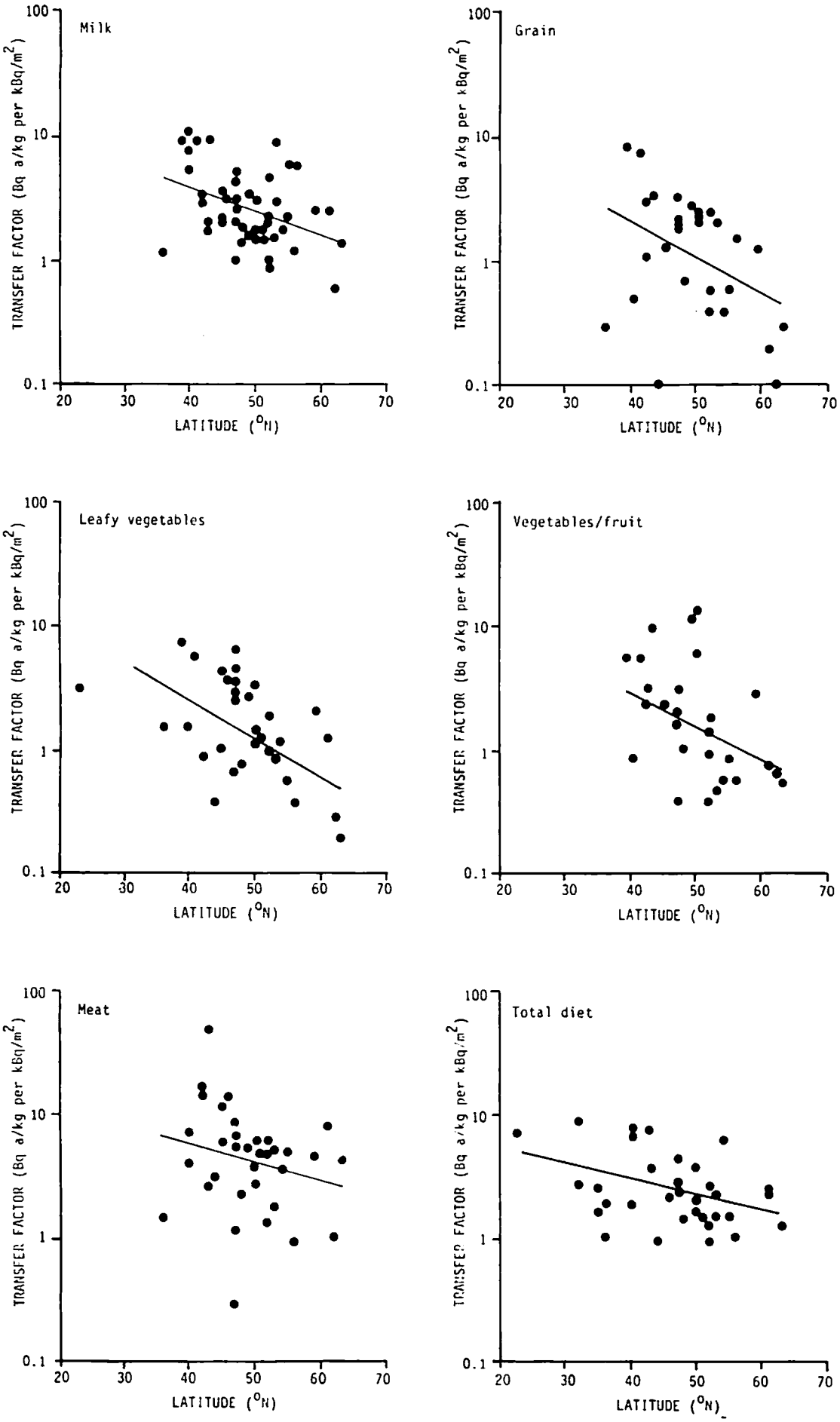

Figure X. Integrated concentrations of caesium-137 in foods and total diet in the first year after the accident per unit caesium-137 deposition density.

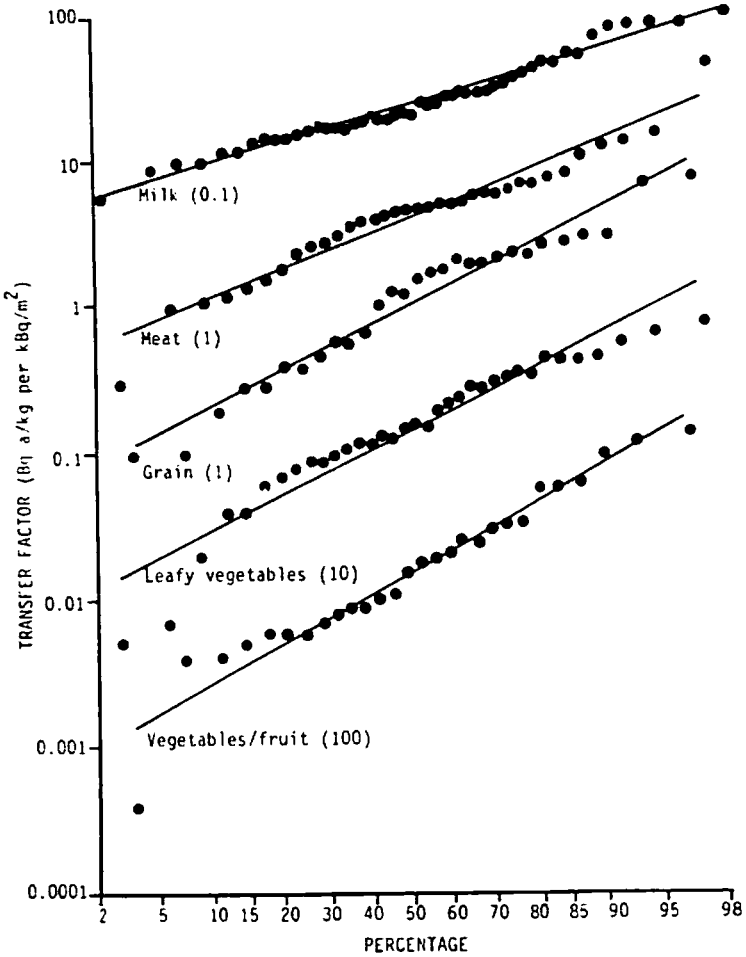

Figure XI. Probability distribution of integrated concentrations of caesium-137 in foods in the first year per unit caesium-137 deposition density. Because of sliding scale on left axis, multiply values by numbers in parentheses. Geometric mean values are 2.7 milk, 4.5 meat, 1.1 grain, 1.5 leafy vegetables and 1.6 vegetables/fruit (Bq a/kg per kBq/m²).

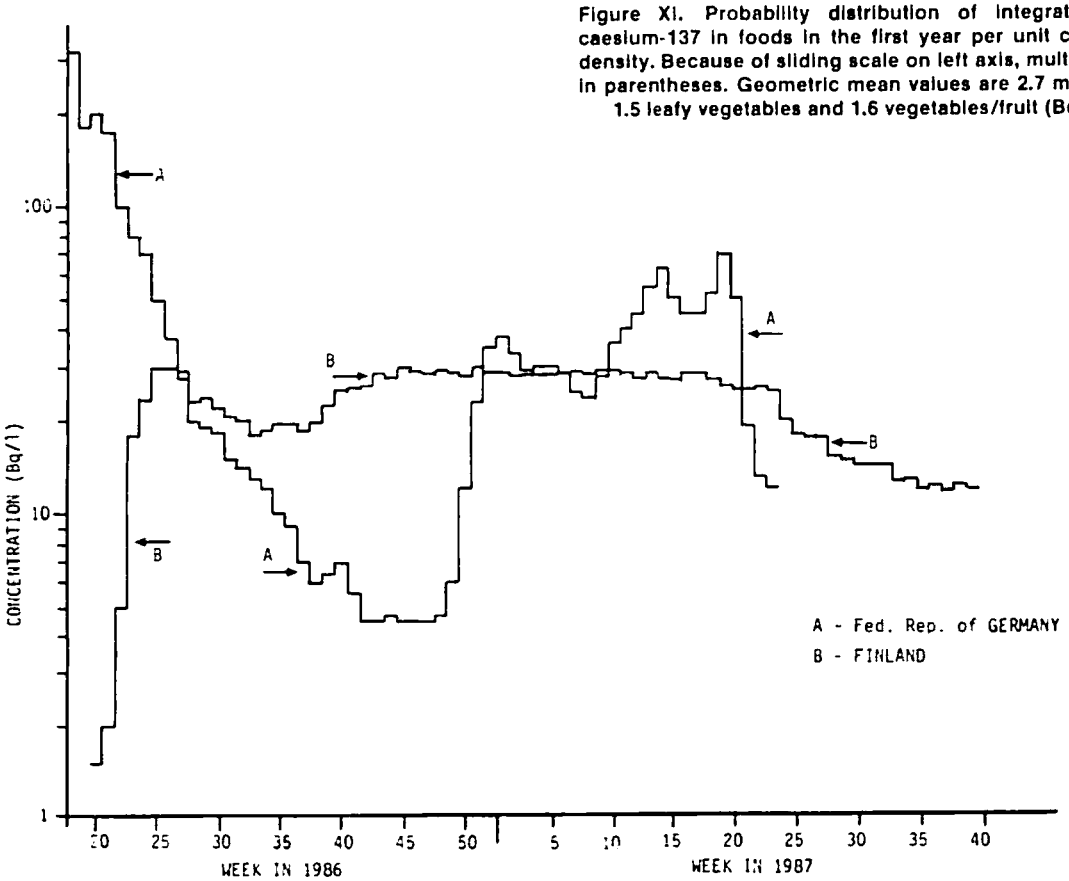

Figure XII. Weekly monitoring results of caesium-137 concentrations in milk from the Federal Republic of Germany (dairy farm in south-east Bavaria) and Finland (country-wide mean). [J5, R3]

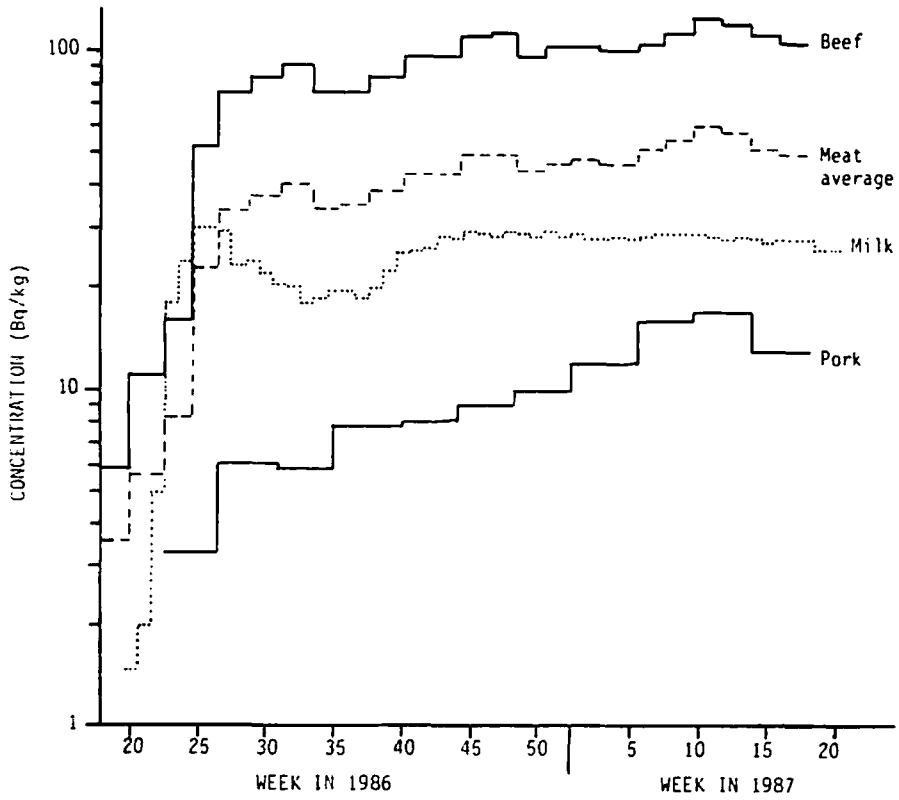

Figure XIII. Country-wide mean concentrations of caesium-137 in meat and milk in Finland [R3, R7]. (Meat average obtained by weighting according to consumption).

able variability depending on type and location. The highest levels were measured in mushrooms of the family Boletaceae that live in symbiosis with trees (mycorrhiza), e.g., in *Xerocomus badius* (Maronen-röhrling). In this species, the ^{137}Cs levels were around 250 Bq/kg, but peak values of around 20,000 Bq/kg were measured at the beginning of September 1986 in the Federal Republic of Germany [W2], and 800 Bq/kg average and 7,800 Bq/kg maximum were measured in the German Democratic Republic, also in September 1986 [L1]. In other Boletaceae, e.g., the popular *Boletus edulis* (Steinpilz or cèpe), the levels were lower, usually below 100 Bq/kg. In non-mycorrhizal mushrooms, e.g., mushrooms of the genus *Agaricus*, such as the common mushroom, ^{137}Cs levels were very low; and (c) concentration of ^{137}Cs in freshwater fish were in some places, e.g., Sweden, found to be many thousands of Bq/kg, though there were large differences between types of fish and even between nearby lakes [S1]. Values of about 300 Bq/kg in plankton-eating lake fish were measured in the Federal Republic of Germany [W2]. Marine fish accumulate only very low concentrations of ^{137}Cs.

D. THE HUMAN BODY

136. Following the accident, extensive measurements were made of ^{131}I in the thyroid or ^{137}Cs in the body. The thyroid measurements were not always made in a standardized way, and much variability was encountered. These results cannot, therefore, be easily interpreted, although they served as a guide to general exposure levels. Measurements of thyroid burdens in the Federal Republic of Germany that were intended to evaluate estimates of ^{131}I intakes through inhalation and ingestion showed that those intakes were overestimated by a factor of about 5 [S16].

137. The amount of ^{137}Cs in the body is generally measured by whole body counting, which can be performed in a reliable, comparable way. These measurements enable a direct assessment of internal doses from ^{137}Cs. Although ingestion was responsible for most of the dose, the contribution from inhalation could also be measured during the first few weeks following the accident [O1].

138. Examples of ^{137}Cs body measurements in the Federal Republic of Germany, France and the United Kingdom are presented in Figure XIV. Generally the amounts increased until late spring or early summer 1987. Regional differences are accounted for by the varying levels of ^{137}Cs in the diet. Lower body burdens are accumulated in children and adult females than in adult males as a result of shorter retention half-times in the body [N1].

139. It is of interest to compare the internal doses estimated directly from body burden measurements and those estimated indirectly from concentrations in foodstuffs. Accordingly, the information on measured body burdens in adults that was available to the Committee was processed to obtain time-integrated body burdens corresponding to the ^{137}Cs intakes during the first year after the accident. The results, presented in Table 16, are the integrated amounts in the body (Bq a) for one year (May 1986 to April 1987)

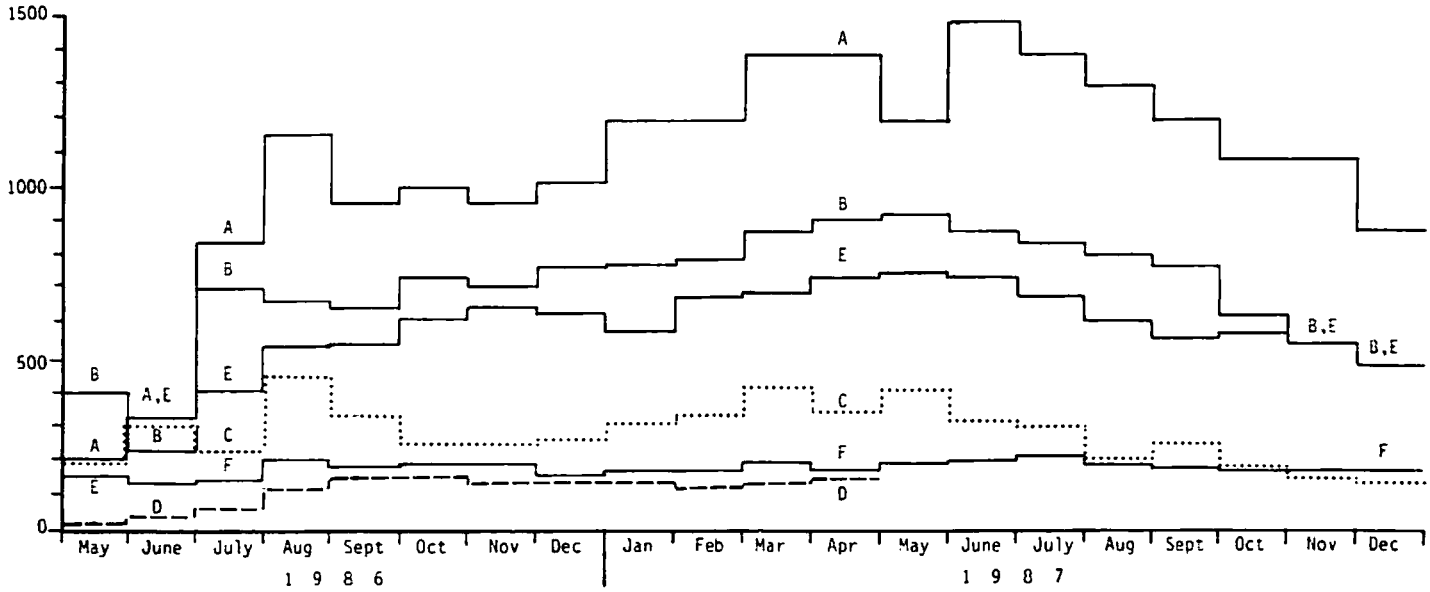

Figure XIV. Caesium-137 in the human body at Munich, Federal Republic of Germany (A: males; B: females; C: children) [S16]; in Oxfordshire, United Kingdom (D: adults) [F2]; and in France (E: Grenoble, adults; F: Saclay, adults) [J4, L5].

and include retention beyond one year of the acquired body burden. The integrated ^{137}Cs body measurements range from 100-200 Bq a in areas of the United Kingdom and France to 2,000-3,000 Bq a in Austria, Bulgaria, Finland, Italy and Norway; in Japan, they were 34 Bq a. The retention function for the adult was taken to be 10% of the burden retained with a half-life of 2 days and 90% of that retained with a half-life of 110 days [I9]. This retention function was used to estimate the time-integrated body burdens during the first year, when the measured information was limited to one or two points in time, and also to calculate the fraction of the time-integrated body burden attributable to retention beyond one year. Continuous intake of ^{137}Cs at a rate of 1 Bq/d gives an integrated concentration in the body of 87 Bq a at the end of one year and a further integrated concentration of 56 Bq a from continued retention with no further intake. Thus, 1 Bq/d for one year gives 143 Bq a in the body or 2.0 Bq a/kg, which results in an effective dose equivalent of 5.0 μSv to reference man.

140. The body burdens expected from the ^{137}Cs concentrations in diet have also been calculated, using reported concentrations in foods for the area considered, when available, or, when not, assuming that the concentrations in foods are proportional to the deposition density of ^{137}Cs. The ratios of the body burdens derived from measurements in man and expected from concentrations in diet are presented in the last column of Table 16.

141. In general, the body burdens are less than would be expected based on deposition in the country or subregion and on local concentrations of ^{137}Cs in foods. The retention function was tested in a controlled study and was found to be adequate [V1]. When food basket or total diet samples were measured, as was done in regions 2 and 3 in France, in Sweden and in the Federal Republic of Germany, the agreement was better. These findings call into question the representativeness of the concentrations in foods and the amounts consumed. This was certainly a factor in the places where people refrained from eating foodstuffs expected to present higher-than-average ^{137}Cs concentrations. Ingestion of less typical foods explain why the measured body burdens of some people, e.g., Lapps (see the Norwegian data in Table 16), are greater than those predicted from the average diet.

142. The ^{137}Cs concentrations in foodstuffs may be overestimates. These overestimates could have come from a sampling bias towards high deposition areas or they could have been due to the fact that losses during food processing or preparation are usually not taken into account; also commercial distribution could cause large scale movements of food and a smoothing of the concentrations over entire countries. This may explain why the measured body burdens in Oslo, Vienna, and regions 1 of Finland and France (low-deposition areas) were higher than predicted and why the reverse was true in the high-deposition regions of Finland and France.

IV. FIRST-YEAR DOSE ESTIMATES

143. Exposures of populations to radionuclides released in the accident have been calculated for all countries for which measurements are available. These include the USSR, most countries in Europe and a few countries in Asia and North America. Thirty-four countries are considered here. The results are used, first, as direct determinations of first-year doses and, second, as a basis for establishing transfer factors to be applied for estimating doses in other countries of the northern hemisphere.

144. The dose equivalents to individuals in the assessed countries during the first year following the accident are presented in Table 17. These are the thyroid dose equivalents to infants and adults, primarily from ^{131}I, and the effective dose equivalents from all

radionuclides and all pathways; they are average results for subregions or for the country as a whole. In each country, there were more localized areas where exposures were both higher and lower than these broad averages.

A. THYROID DOSE EQUIVALENTS

145. Thyroid dose equivalents have been evaluated because there were significant amounts of ^{131}I in the released materials. Doses to ^{131}I in the environment are generally higher to infants than to adults because the main pathway is through milk consumption, and also because infants are characterized by greater ^{131}I uptake and smaller thyroid mass.

146. The estimated average infant (one year old) and adult thyroid dose equivalents during the first year in countries or subregions are listed in Table 17. While these doses were primarily due to ^{131}I, the contributions from other radionuclides and all pathways are included.

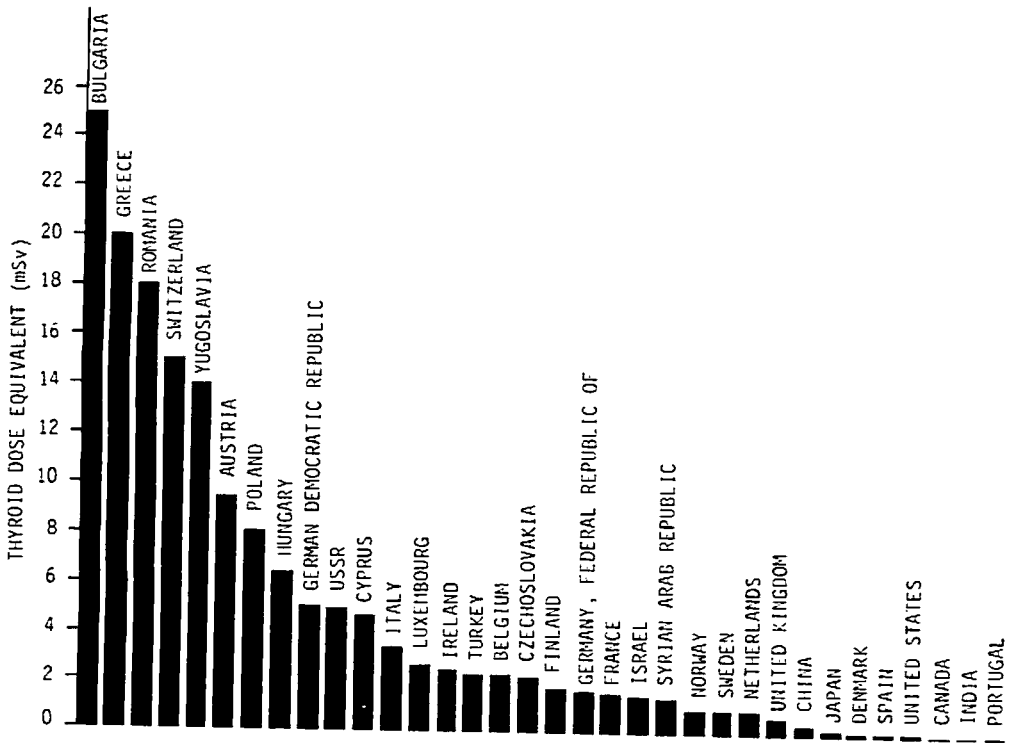

Figure XV. Country-wide average infant thyroid dose equivalents from the Chernobyl accident.

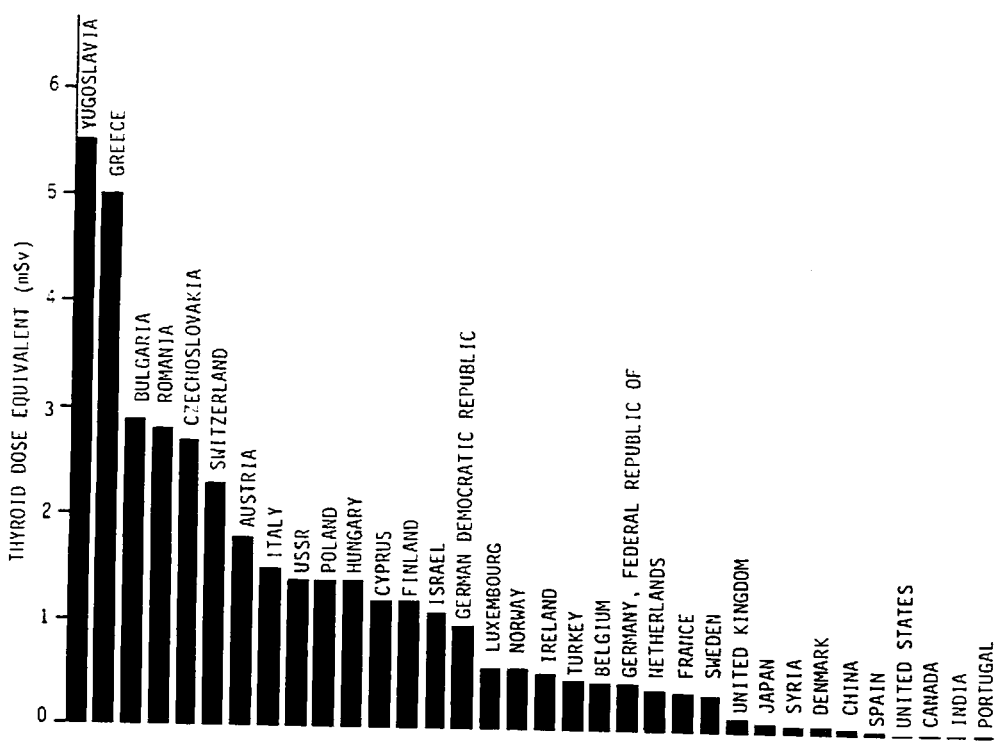

Figure XVI. Country-wide average adult thyroid dose equivalents from the Chernobyl accident.

147. The calculated results for thyroid dose equivalents, and also for effective dose equivalents, take into account, where possible, the application of countermeasures. This was usually done by adjusting the integrated concentrations in foods so that the values represented what was actually consumed. However, the Committee has not taken into consideration the use of thyroid blocking agents or stable iodine preparations. By reducing uptake, these would have afforded some additional protection against inhaled and ingested radioiodine.

148. The country averages of infant and adult thyroid dose equivalents are listed in Table 18 and shown in Figures XV and XVI. Infant thyroid dose equivalents in Europe generally ranged from 1 to 20 mSv, but there were higher doses in some parts of Romania, Greece, Switzerland, Bulgaria and the USSR. Adult thyroid doses were usually smaller than infant doses in the same country by a factor of about 5 in central and western Europe, but the differences were smaller in northern Europe, where milk was less contaminated because the cows had not been on pasture, and in regions of southern Europe and Asia, where the contamination of leafy vegetables increased adult thyroid doses.

149. The thyroid dose estimates are compared with the estimates reported by individual countries in Table 18. The country-reported results are those collected by the Nuclear Energy Agency of the OECD [N5]. Differences from unity in the ratios of the estimates to the country-reported results reflect differences in the various assumptions regarding intake, the age groupings for infants and the ways of accounting for countermeasures. The dose estimates are both higher and lower than those reported by the countries, but the differences are generally not greater than a factor of 4 for infants and a factor of 3 for adults.

B. EFFECTIVE DOSE EQUIVALENTS

150. The effective dose equivalents received by individuals (adults) during the first year following the accident are presented in Table 17, which also shows rural-urban differences. Contributions to dose from the ingestion pathway also include committed doses from caesium in the body following the first-year intake of caesium in diet.

151. The highest average first-year committed effective dose equivalent in subregions was 2 mSv in the Byelorussian Soviet Socialist Republic. Subregions where effective dose equivalents were 1-2 mSv were located in Romania and Switzerland and 0.5-1 mSv in Austria, Bulgaria, Federal Republic of Germany, Greece and Yugoslavia. The effective dose equivalent in the Byelorussian Soviet Socialist Republic approached the yearly effective dose equivalent due to natural radiation sources. The mean values for each country are listed in Table 18 and plotted in Figure XVII.

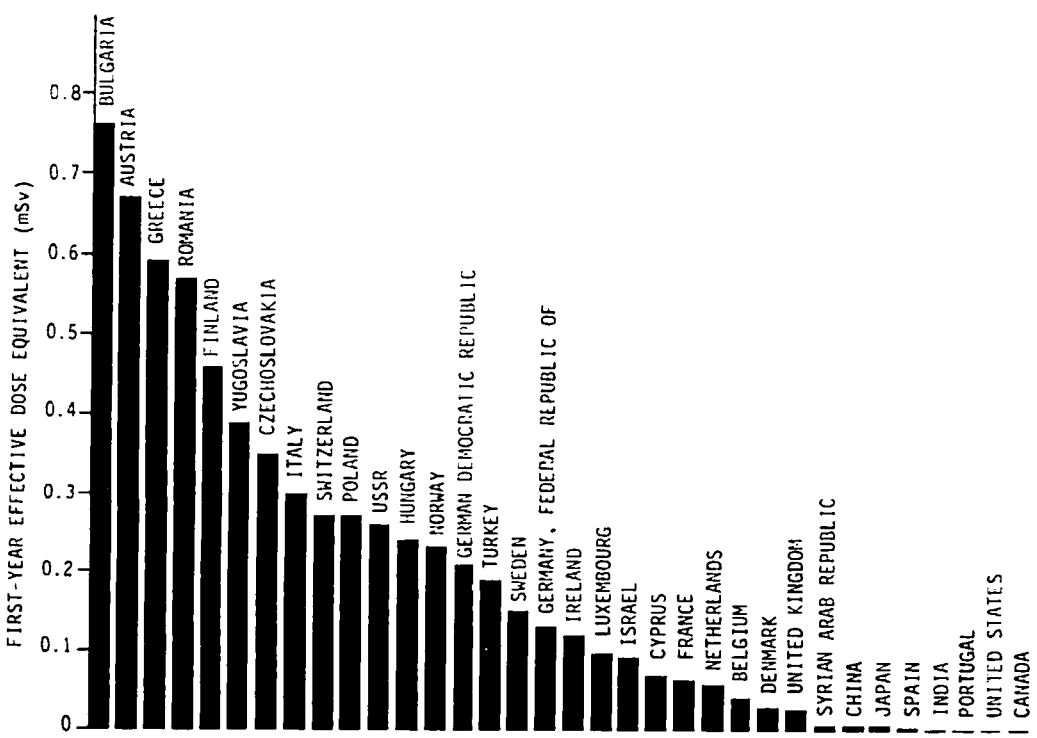

Figure XVII. Country-wide average first-year committed effective dose equivalents from the Chernobyl accident.

152. These estimates of first-year committed effective dose equivalent are in reasonable agreement with the results reported by individual countries [N5], as is also shown in Table 18. While there are some greater discrepancies between these estimates and other, provisional dose estimates [M1, D5], the latter were based on measurements made in the first months after the accident. Differences in estimates from country-reported results can be attributed to the averaging of results over larger subregions, the inclusion of additional food groups and the use of different assumptions for occupancy, shielding and food consumption. Most results from individual countries did not account for urban run-off. On average, however, the comparability of the Committee's estimates and those of individual countries is good, the average ratio being 1.06 with a standard error of 0.6.

C. PATHWAY CONTRIBUTIONS

153. The pathway contributions to the first-year committed effective dose equivalents varied substantially by location for all pathways except cloud gamma, which was everywhere less than 1%. The contribution from inhalation averaged 5%, with a range from 0.1% in Ireland to 22% in Turkey.

154. The first-year committed effective dose equivalents resulted primarily from the ingestion pathway, which in most countries accounted for over 60% of the total dose and in southern countries for over 80%. The differences in pathway contributions are illustrated in Figure XVIII for three groupings of countries: southern countries (<40° N latitude), temperate countries (41°-55° N latitude) and northern (Scandinavian)

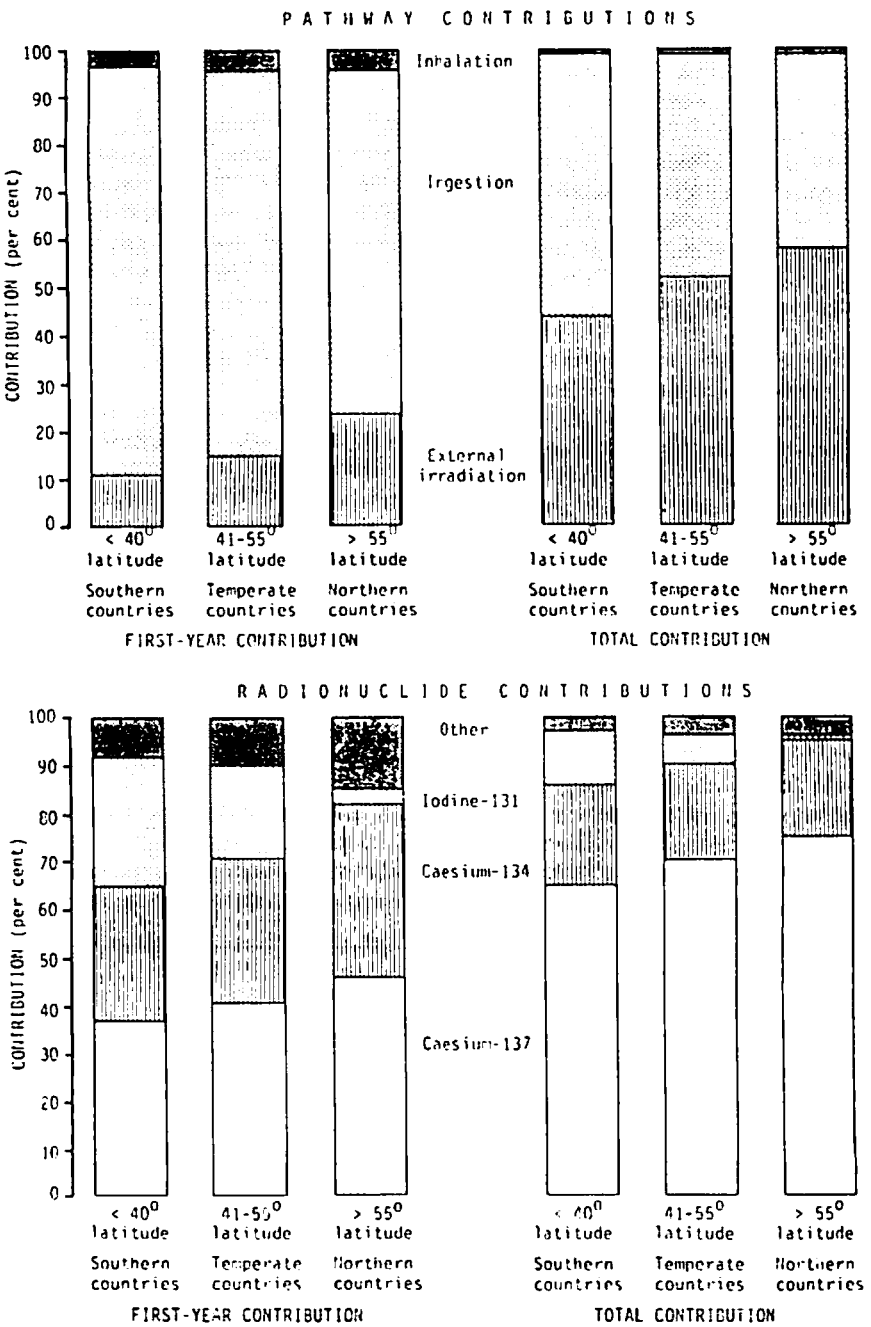

Figure XVIII. Pathway and radionuclide contributions to the first-year effective dose equivalents and the effective dose equivalent commitments.

countries (>55° N latitude). The contributions to committed first-year effective dose equivalents average 11%, 19% and 27% from external irradiation and 86%, 76% and 69% from ingestion in the southern, temperate and northern countries, respectively.

155. The pathway contributions to the thyroid dose equivalents in the first year also varied from north to south. Average results for all age groups showed the significance of the ingestion pathway (through milk and leafy vegetables), which was generally responsible for over 70% of the total dose but in northern Europe was responsible for only 40%. The inhalation pathway contributed 20-50% of the first-year thyroid dose in some northern countries.

D. RADIONUCLIDE CONTRIBUTIONS

156. Contributions to the first-year committed effective dose equivalents were dominated by the radionuclides ^{131}I, ^{134}Cs and ^{137}Cs. For the cloud gamma and inhalation pathways, some other radionuclides in air were important, specifically ^{132}Te and ^{103}Ru. For the external irradiation and the ingestion pathways, some other short-lived radionuclides were also significant. Caesium-137 and ^{134}Cs together contributed over 50% of the dose from ingestion in most countries. For the committed first-year thyroid dose equivalent, ^{131}I typically contributed over 90%.

157. A seasonal dependence of the radionuclide contribution to the committed first-year effective dose equivalent is indicated in Figure XVIII. The dose from ^{131}I ranged from less than 4% in Scandinavia, where cows were not on pasture and leafy vegetable production was minimal, to some 20% in countries at lower latitudes, where quite different agricultural conditions prevailed. The remainder of the main dose contribution from ^{137}Cs varied in an inverse way, becoming increasingly more important in northern countries.

E. TRANSFER RELATIONSHIPS

158. The input data for the assessment of the committed first-year dose equivalents have been based on measurements through the first year. These can be analysed to infer transfer relationships to dose equivalents. Because of the differences in local conditions and varying assumptions with regard to food consumption and in determining integrated concentrations, it would not be reasonable to expect uniformly consistent values of transfer factors. Nevertheless, it is useful to indicate the range of values that applied to conditions at the time.

1. Transfer from deposition to dose from external irradiation

159. Doses due to external irradiation from deposited radionuclides are delivered directly. The transfer factor for external radiation in the first month after the accident depended upon the presence of many short-lived radionuclides. As shown in Figure VII, the average outdoor effective dose equivalent was around 15 μSv per kBq/m^2 of ^{137}Cs. This multiplied by the shielding/occupancy factor of 0.36 [0.2 (outdoor occupancy) plus the product of 0.8 (indoor occupancy) and 0.2 (shielding)] gives an average contribution of 5 μSv per kBq/m^2.

160. Transfer factors for the period between one month and one year may be taken directly from Table 5. For ^{137}Cs, the value is 8.04 μSv per kBq/m^2. When this is multiplied by the shielding/occupancy factor of 0.36 and the urban population/runoff factor of 0.75 [0.5 (rural population) plus the product of 0.5 (urban population) and 0.5 (urban removal)], the average contribution from ^{137}Cs alone is seen to be 2.2 μSv per kBq/m^2.

161. The one-month to one-year transfer factors for other important radionuclides in deposited material, from Table 5, are 18.6, 0.691, 2.09 and 0.015 μSv per kBq/m^2 of ^{134}Cs, ^{103}Ru, ^{106}Ru and ^{131}I, respectively. It is convenient to relate these further to ^{137}Cs deposition density by using median values of the ratios of these radionuclides to ^{137}Cs in deposition. These ratios are 0.5 for ^{134}Cs and ^{106}Ru, 1.6 for ^{103}Ru and 6.2 for ^{131}I (Table 11). The total contribution, using the same factors (shielding/occupancy and urban population/runoff), to the effective dose equivalent from these radionuclides per unit ^{137}Cs deposition density is 3.1 μSv per kBq/m^2.

162. The components of the first-year transfer to effective dose equivalent due to external irradiation from deposited radionuclides relative to unit ^{137}Cs deposition density may be summarized as follows:

Radionuclide	*Outdoor effective dose equivalent (μSv per kBq/m^2)*	*Shielding/ occupancy factor*	*Urban population/ runoff factor*	*Ratio to caesium-137*	*Transfer factor components (μSv per kBq/m^2)*
First month					
All	15	0.36			5
Second to twelfth month					
Cs-137	8.04	0.36	0.75		2.2
Cs-134	18.6	0.36	0.75	0.5	2.5
Ru-103	0.691	0.36	0.75	1.6	0.30
Ru-106	2.09	0.36	0.75	0.5	0.28
I-131	0.015	0.36	0.75	6.2	0.025
Total (first year)					10

2. Transfer from deposition to thyroid dose equivalent of iodine-131

163. The derivation of the transfer factor from deposition to thyroid dose equivalent in the first year is presented in Table 19. Since the thyroid dose calculation includes inhalation and ingestion contributions, some differences may result from relating the total dose only to ^{131}I deposition.

164. The results vary by orders of magnitude. The very low values for Scandinavian countries reflect the early stage of the growing season there and the consequently low transfer to milk and leafy vegetables. The relatively high values are due to several factors. In southern countries, animals were already on pasture and in addition, in some areas contributions from extensive use of sheep's milk was included, in which the concentrations were about 10 times higher than in cow's milk. Protective actions that were taken further increased the variability of these results. The latitudinal dependencies in the transfer factor from deposition to thyroid dose equivalent from ^{131}I for infants and adults are shown in Figures XIX and XX.

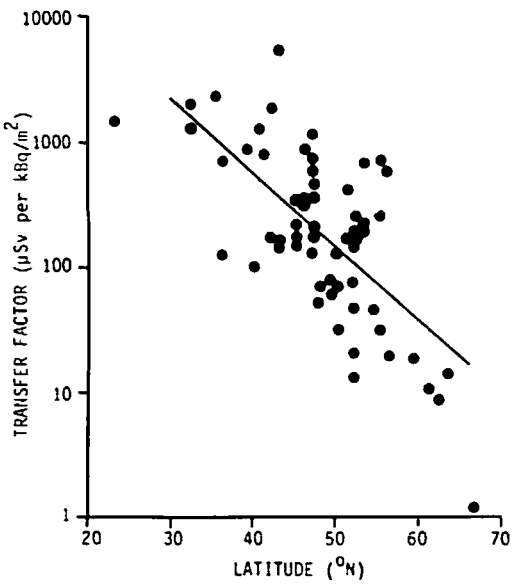

Figure XIX. Transfer factor for infant thyroid dose equivalent from ingestion and inhalation relative to iodine-131 deposition density.

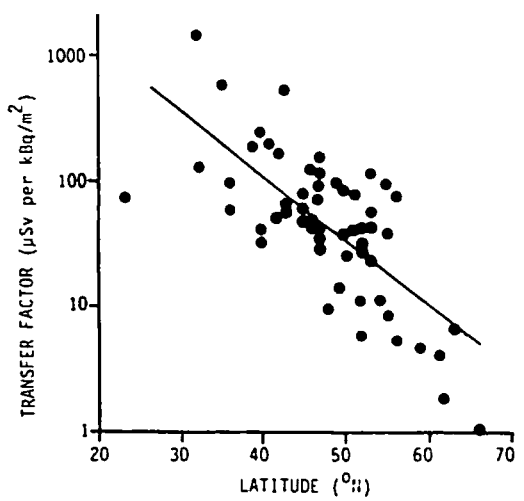

Figure XX. Transfer factor for adult thyroid dose equivalent from ingestion and inhalation relative to iodine-131 deposition density.

3. Transfer from deposition to dose from ingestion of caesium-137

(a) Transfer from deposition to diet

165. The quotients of the first-year integrated concentrations of ^{137}Cs in foods and the ^{137}Cs deposition density, which were presented in chapter III (Table 15) for the individual food categories, define the first-year deposition to diet transfer factors for ^{137}Cs, the b_1 values. These values have been combined and weighted by consumption amounts to obtain the average deposition to first-year total diet transfer factors for each country or subregion listed in Table 20. Also listed are the average integrated concentrations of ^{137}Cs in diet and the total first-year intakes of ^{137}Cs.

166. The results of first-year transfers of ^{137}Cs to total diet under the conditions that prevailed at the time of the accident are included in Figure X. The least-squares fit through the measured values shows a trend toward increasing transfer per unit deposition at southern latitudes, as seen also in the individual components of diet from first-year measurements (also in Figure X). Most countries and subregions in temperate latitudes are in the range 1-4 Bq a/kg per kBq/m^2. There are, however, greater deviations in some countries that reported higher levels in foods than would have been expected from estimated deposition. In some cases, there is uncertain transfer to some food items as well as higher transfer to diet due to the inclusion of certain foods, such as milk and meat from goats and sheep. It would be of interest to study in more detail the local conditions that cause deviations from the more widely applicable transfer factors derived here.

167. The log-normal distribution of b_1 transfer factors for ^{137}Cs in total diet is shown in Figure XXI. A single population-weighted value is plotted for each country; for some countries, the values were largely inferred, but these have also been included. The values range from 1 to 9 Bq a/kg per kBq/m^2, with a geometric mean of 2.6 Bq a/kg per kBq/m^2. This mean may be compared with the average value of 4.1 Bq a/kg per kBq/m^2 (range 1.9 to 6.3) for the first-year transfer of fallout ^{137}Cs, derived from long-term measurements (Table 7).

(b) Transfer from diet to body

168. The transfer factor from diet to body burden, P_{34}, is derived in Table 20. The integrated concentration of ^{137}Cs in the body is obtained by multiplying the dietary intake of ^{137}Cs in the first year by a standard factor, 143 d/70 kg (the mean residence time of ^{137}Cs in the body divided by the body mass). The integrated concentration includes retention in the body beyond the first year. The transfer factor from total diet to body burden is the ratio of integrated concentrations in the body and in diet. Variability in this factor reflects only differences in food consumption. The median value for this transfer factor is 2.9 Bq a/kg per Bq a/kg.

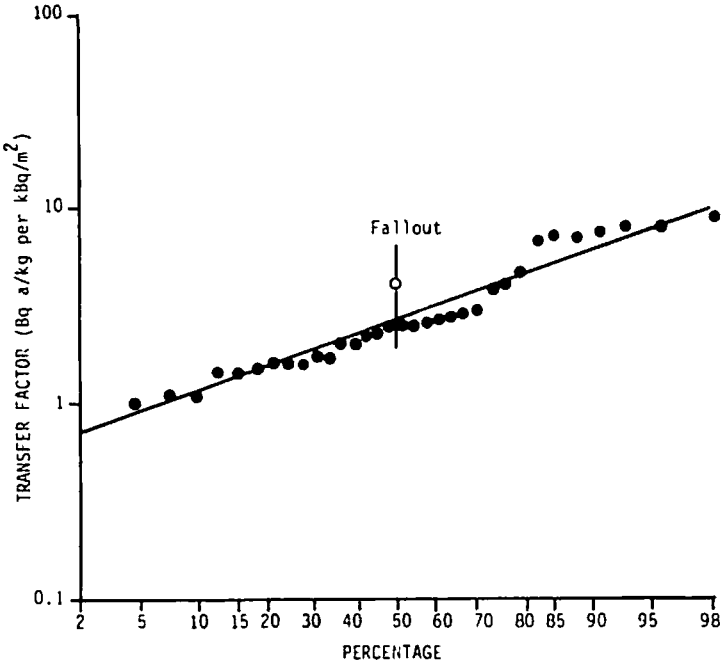

Figure XXI. Probability distribution of caesium-137 in first year total diet relative to caesium-137 deposition density.

(c) Transfer from body to effective dose equivalent

169. The transfer factor from ^{137}Cs in the body to the effective dose equivalent, P_{45}, is based on the dose factor given in Table 6. For adults, this factor is 0.014 μSv per Bq intake. The retention function for caesium in the body was discussed in paragraph 139. Since the mean retention time is 143 days, an intake of 1 Bq corresponds to 1 Bq × 143 d ÷ 70 kg = 5.6 10^{-3} Bq a/kg in the body. The transfer factor from integrated concentration in the body to the effective dose equivalent is 0.014 ÷ 5.6 10^{-3}, or 2.5 μSv per Bq a/kg.

170. The overall transfer factor from deposition to the first year effective dose equivalent, $P_{25,1}$, is obtained by sequential multiplication of the transfer factors P_{23} (which is referred to as b_1 for the first-year transfer), P_{34} and P_{45}. These values for the ingestion of ^{137}Cs in countries or subregions are listed in the last column of Table 20.

V. DOSE COMMITMENTS

171. Dose equivalent commitments have been calculated using transfer factors developed and used by the Committee for its assessments of the dose commitments resulting from atmospheric nuclear weapons tests [U1, U2]. Since those transfer factors were developed for the rather more uniform and continuous deposition patterns of fallout, they are here applied only to regional groups of countries. Because first-year doses were for the most part calculated from measured data, only the components of the fallout models corresponding to transfers beyond the first year following deposition were taken into consideration. For that time, i.e., more than one year after the accident, the only pathways to be considered are external irradiation due to activity deposited on the ground and ingestion of foodstuffs, and the only radionuclides that contribute significantly to the dose equivalents are ^{134}Cs and ^{137}Cs. For these radionuclides, the effective dose equivalent and the thyroid dose equivalent have the same value for a given exposure.

172. The methods for obtaining projected dose estimates were discussed in section II.C. After specific values for the transfer factors have been derived, they are applied to the average ^{134}Cs and ^{137}Cs deposition. Since the ^{134}Cs to ^{137}Cs deposition ratio was uniform in all countries, the contributions to the dose from both radionuclides may be related to the ^{137}Cs deposition value.

A. TRANSFER RELATIONSHIPS

1. Transfer from deposition to dose from external irradiation

173. Values of the effective dose equivalent per unit deposition density of radionuclides for the period after one year are given in Table 5. These apply to a soil relaxation depth of 3 cm. Assuming an initial runoff loss of one half of deposition in urban areas, equal proportions of urban and rural residents, a shielding factor of 0.2 indoors and an indoor occupancy factor of 0.8, the transfer factors for the dose per unit deposition from external irradiation beyond one year are 71 μSv per kBq/m^2 for ^{137}Cs and 9.8 μSv per kBq/m^2 for ^{134}Cs. An additional small contribution of 0.4 μSv per kBq/m^2 comes from ^{106}Ru. Using a value of 0.5 for the deposition ratio $^{134}Cs/^{137}Cs$ as well as for $^{106}Ru/^{137}Cs$, the total dose may be estimated directly from ^{137}Cs deposition: 76 μSv per kBq/m^2. The derivation of this transfer factor may be summarized as follows:

Radionuclide	Outdoor effective dose equivalent (μSv per kBq/m^2)	Shielding/ occupancy factor	Urban population/ runoff factor	Ratio to caesium-137	Transfer factor components (μSv per kBq/m^2)
Cs-137	264	0.36	0.75		71.3
Cs-134	36.2	0.36	0.75	0.5	4.9
Ru-106	1.65	0.36	0.75	0.5	0.2
Total for the period beyond 1 year					76

2. Transfer from deposition to dose from ingestion

(a) Transfer from deposition to diet

174. The model for the transfer from deposition to diet is:

$$P_{23} = b_1 + b_2 + b_3 e^{-\lambda t}$$

where b_2 is the second-year transfer and $b_3 e^{-\lambda t}$ is the subsequent transfer, in which the elimination of radiocaesium by environmental and physical processes is taken into account. The transfer from deposition to diet beyond the first year is thus represented by:

$$P_{23,2+} = P_{23} - b_1$$

Values of $P_{23,2+}$ and P_{23} derived from long-term fallout measurements of ^{137}Cs are given in Table 7. For all foodstuffs except grain products, the average values of $P_{23,2+}$ given in Table 7 were used for ^{137}Cs in all of the large regions considered in the assessment: 2.1, 1.4, 2.0 and 8.0 Bq a/kg per kBq/m^2 for milk products, leafy vegetables, vegetables/fruit and meat, respectively. In the case of grain products, the value of $P_{23,2+}$ for ^{137}Cs in a large region was assumed to equal the population-weighted mean of the b_1 values estimated for that region (see paragraph 96).

175. The deposition to total diet transfer factor is obtained by weighting the values for the food groups by consumption amounts. Population-weighted food consumption estimates for the large regions considered in the commitment assessment are listed in Table 21. The regional value for the transfer factor for grain products is given along with the weighted total diet transfer factor.

(b) Transfer from diet to body

176. The transfer factor from total diet to body burden, P_{34}, is the quotient of normalized body burden and normalized dietary concentration. These values vary only because of consumption differences. The value can be derived by multiplying total food consumption (kg/a) by 143 Bq d per Bq (residence time in body) and dividing by 365 d/a and 70 kg (body mass). The results are listed in Table 21. The median value for these large regions is 2.8 Bq a/kg per Bq a/kg.

(c) Transfer from body to effective dose equivalent

177. The transfer factor from the time-integrated concentration in the body to the effective dose equivalent, P_{45}, is, for ^{137}Cs, equal to 2.5 μSv per Bq a/kg, as derived in paragraph 169.

178. The overall transfer factor for ^{137}Cs from deposition to total diet to body to effective dose equivalent in the time period beyond the first year, $P_{25,2+}$, is given in Table 21. The values average 20 μSv per kBq/m^2 in the northern and temperate countries and about 25 μSv per kBq/m^2 in southern countries.

179. The transfer of ^{134}Cs from deposition to effective dose equivalent may be related to ^{137}Cs deposition, taking into account the lower deposition (^{134}Cs/^{137}Cs = 0.5) and the higher dose per unit intake (^{134}Cs/^{137}Cs = 1.4). This gives effective dose equivalents from ^{134}Cs 70% of those from ^{137}Cs in the first year. Subsequent transfer is less because of the shorter half-life of ^{134}Cs, but most significant transfer to most foods occurs within the first few years of deposition. Average results for all countries show the ^{134}Cs ingestion dose to be 65% of that from ^{137}Cs, corresponding to 70% of the first-year ^{137}Cs dose and 60% of the subsequent ^{137}Cs dose.

B. AVERAGE DOSE EQUIVALENT COMMITMENTS IN LARGE REGIONS

180. The effective dose equivalent commitments from all radionuclides released in the accident are evaluated in Table 22. These are the average results for the large regions. The first-year dose is the population-weighted result of the effective dose equivalents given in Table 18. The component of dose from exposure or intake after the first year is determined by multiplying the population-weighted ^{137}Cs deposition density in the region by the total $P_{25,2+}$ transfer factor, comprising external gamma exposure (invariant across regions and derived in paragraph 173) and doses from ^{137}Cs and ^{134}Cs in foods (derived in paragraphs 178 and 179).

181. The results range from 1,200 μSv in south-eastern Europe (Bulgaria, Greece, Italy, Yugoslavia), 970 μSv in Scandinavia, 940 μSv in central Europe, 820 μSv in the USSR and 510 μSv in eastern Mediterranean countries to 20 μSv or less in other regions. These results are illustrated in Figure XXII. Further evaluations of regional effective dose equivalent commitments are presented in the following section, VI.C.

C. PATHWAY AND RADIONUCLIDE CONTRIBUTIONS

182. The relative contributions of external and internal irradiation to the effective dose equivalent com-

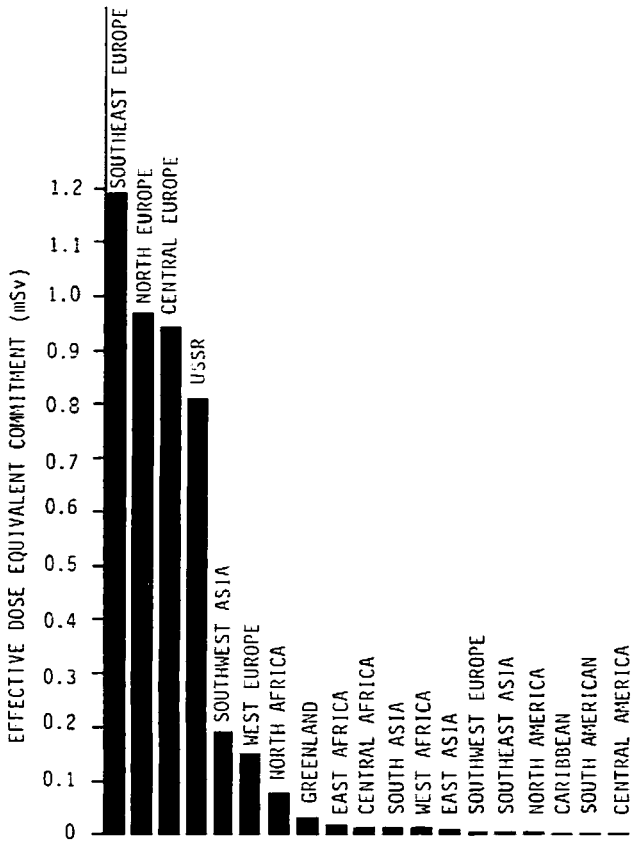

Figure XXII. Regional average effective dose equivalent commitments from the Chernobyl accident.

mitment vary from one region to another. The external irradiation dose pathway becomes relatively more important as time goes on, and is the dominant contributor to the effective dose equivalent commitment in all but the southern countries. The median contributions to the effective dose equivalent commitments from external irradiation and ingestion are approximately 60-40% in northern countries, 55-45% in temperate countries and 45-55% in southern countries.

183. Caesium-137 is the dominant radionuclide contributing to the effective dose equivalent commitment, accounting for about 75%, 70% and 65% in northern, temperate and southern countries, respectively. Because of its shorter half-life, ^{134}Cs contributes much less to the effective dose equivalent commitment than ^{137}Cs via the external exposure pathway. Overall, the contribution of ^{134}Cs to the effective dose equivalent commitment is about 20% of the total in all regions. The contribution from ^{131}I ranges from less than 1% in northern countries to about 10% in southern countries. The remaining 4% or 5% of the effective dose equivalent commitment comes from other radionuclides that caused exposures within the first year.

184. The pathway and radionuclide contributions to the effective dose equivalents, including both the first-year components and the contributions over all time, are illustrated in Figure XVIII.

VI. COLLECTIVE DOSE COMMITMENT

185. On the basis of available measurements, calculations have been completed of the first-year doses in 34 countries and the dose commitments in several large regions. Using transfer factors derived from these results, dose estimates may be made for the remaining areas of the northern hemisphere. These areas, generally far removed from the accident site, received only trace deposition of radioactive materials and therefore make only small contributions to the total collective dose equivalent. Nevertheless, for completeness, the entire northern hemisphere is considered in the dose assessment. This is done in two steps: (a) by considering the relationship between deposition and distance to estimate ^{137}Cs deposition in all regions; and (b) by applying a general transfer factor based on ^{137}Cs deposition to estimate the effective dose equivalent commitment from all pathways and all radionuclides.

A. CAESIUM-137 DEPOSITION WITH DISTANCE FROM CHERNOBYL

186. It may be expected that radionuclide deposition and radiation doses generally decrease with distance from a release by virtue of geographic spreading and dilution in the atmosphere. Of course, there may be significant variations within the first hundreds of kilometres, depending on the exact course of the plumes and the rainfall pattern. In the case of the accident at Chernobyl, however, the release lasted several days, during which the wind changed to all directions, so even these variations were minimized.

187. Figure XXIII shows the relationship between ^{137}Cs deposition and distance, based on measurements in the 33 assessed countries outside the USSR. There is seen to have been a relatively uniform decrease in the average ^{137}Cs deposition density with distance from Chernobyl. An envelope of points is shown along with the central power-function curve, from which the ^{137}Cs deposition densities in the various regions are estimated. The average ^{137}Cs deposition densities in the five main regions of Europe, based on measurements, are shown.

188. In Figure XXIII the distance to a particular region is the population-weighted average of the distances to the capital cities or to the approximate population centres of the countries in the region. The average ^{137}Cs deposition density in the region is then selected from the central curve in Figure XXIII.

B. TRANSFER FACTOR FOR TOTAL DOSE COMMITMENT BASED ON CAESIUM-137 DEPOSITION

189. For the purpose of estimating exposures from the Chernobyl accident in countries for which measurements are unavailable, it is necessary to have a general transfer factor that accounts for the total effective dose equivalent commitment from all radionuclides and all pathways based on extrapolated estimates of ^{137}Cs deposition density.

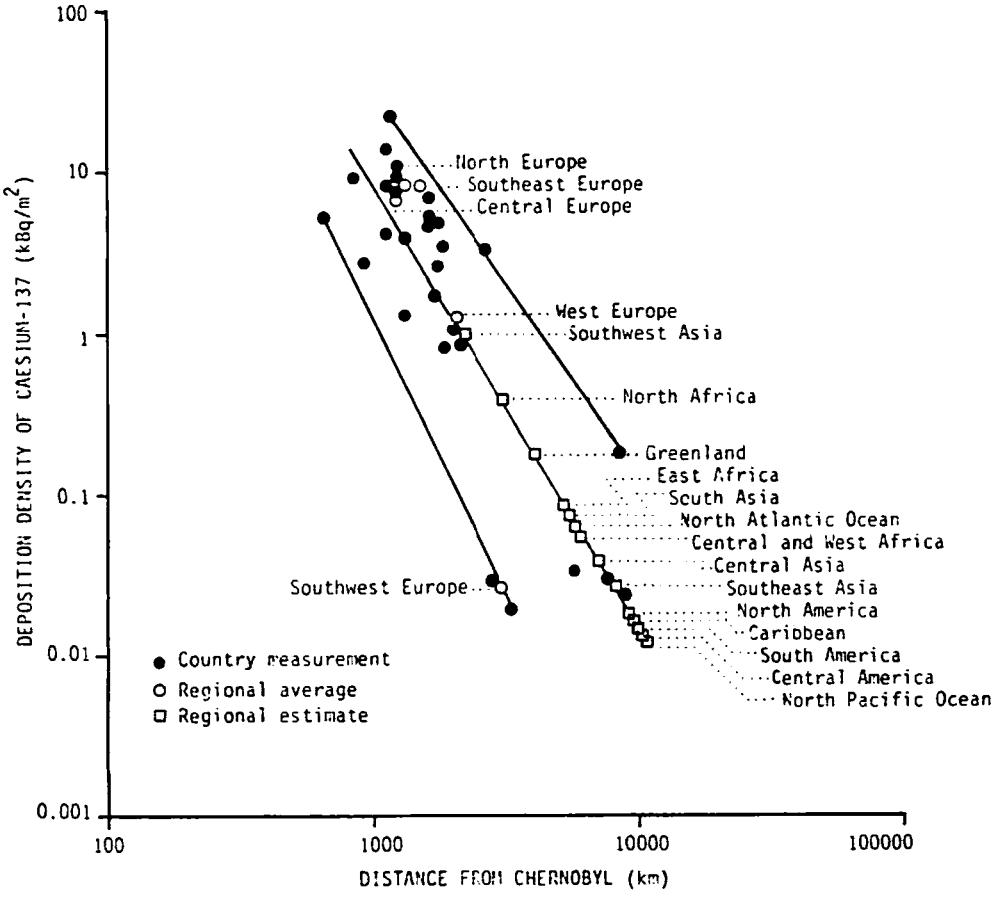

Figure XXIII. Deposition density of caesium-137 with distance from the Chernobyl reactor.

190. The first component of this transfer factor from external irradiation was derived in paragraphs 159-161 and 173. The summary values are entered in Table 23, which compiles the general transfer factors for southern (<40°), temperate (41-55°) and northern (>55°) latitudinal regions. For external irradiation, the same assumptions are used for all regions, so the components of the transfer factor per unit ^{137}Cs deposition are the same.

191. Because of differences in agricultural conditions in countries at the time of the accident, some latitudinal dependence must be introduced into the components of the transfer factor from the ingestion pathway. The transfer factors to effective dose equivalent for ^{137}Cs from first-year ingestion were derived in Table 20. The population-weighted values for northern, temperate and southern latitudes are approximately 15, 20 and 25 μSv per kBq/m^2. The values for ^{134}Cs, based on ^{137}Cs deposition amounts, are 70% of the corresponding values for ^{137}Cs. These estimates are entered in Table 23.

192. After the first year, the transfer factor components for ^{137}Cs ingestion are 20 μSv per kBq/m^2 at northern and temperate latitudes and 25 μSv per kBq/m^2 at southern latitudes (Table 21 and paragraph 178). The corresponding estimates for ^{134}Cs are 60% of the ^{137}Cs estimates (paragraph 179).

193. Regional (i.e., northern, temperate or southern) values of ^{131}I transfer factors may be selected from Table 19 and from Figure XX. Based on the fit to calculated values for individual countries or their subregions, approximate average values are 5, 50 and 100 μSv per kBq/m^2 for countries at northern, temperate and southern latitudes, respectively. These are the thyroid dose equivalents relative to ^{131}I deposition density. The contribution to the effective dose equivalent is obtained by multiplying them by the weighting factor for the thyroid (0.03). The transfer factor may be based on ^{137}Cs deposition by multiplying further by the average ratio of ^{131}I to ^{137}Cs deposition, 6.2 (Table 11). The resulting transfer factor components for ^{131}I, for the first year only, are 1, 10 and 20 μSv per kBq/m^2.

194. The components of the transfer factor based on ^{137}Cs deposition to effective dose equivalent commitment from the two major pathways and from the dominant radionuclides are summarized in Table 23. It must be understood that these factors apply to the conditions at the time following the accident and to the average composition of radionuclides in the dispersed material as observed. The latitudinal differences apply only to the ingestion pathway.

C. ESTIMATES OF COLLECTIVE EFFECTIVE DOSE EQUIVALENT COMMITMENT

195. Estimates of collective dose equivalent commitments for all regions of the northern hemisphere are compiled in Table 24. To allow an estimate to be

made of the total release of ^{137}Cs, this listing includes also the ocean areas north of the equator. The country populations given in [U3] have been adjusted, based on individual country growth rates, to values appropriate for 1986. The population of the northern hemisphere ($4.3\ 10^9$) makes up 88% of the total world population.

196. The effective dose equivalent commitments in the large regions (Table 22) were estimated on the basis of measurements in the first year and projections for subsequent times. The estimates for European regions are carried forward to Table 24, with a few additional countries having been included in some regions. The product of population and effective dose equivalent commitment is the estimated collective effective dose equivalent commitment.

197. Countries outside Europe but still in the northern hemisphere (i.e., the countries of Asia, North America and parts of Africa and South America) have been grouped in several regions. The population-weighted distances to individual countries are used as the distances to the regions for the purpose of estimating average ^{137}Cs deposition (Figure XXIII). For these regions, all of which lie at southern latitudes (<40°), the transfer factor 190 μSv per kBq/m^2 (Table 23) is used. The estimated effective dose equivalent commitments for all geographical regions are illustrated in Figure XXII. Multiplication by the populations of the regions gives the collective effective dose equivalent commitments.

198. The total collective effective dose equivalent commitment from the accident is estimated to be 600,000 man Sv. From Table 24, it is seen that 53% is experienced in European countries, 36% in the USSR, 8% in Asia, 2% in Africa and 0.3% in North, Central and South America.

199. Alternative estimates of collective effective dose equivalent commitment have been made for the 34 countries for which more detailed radiological data were available. These estimates are based on the total production for human consumption of foods in all the countries. There is no need to consider where the foods are consumed. The collective effective dose equivalent commitment estimates based on production are generally in close agreement with the estimates based on individual consumption rates in countries and populations. The production-based estimated total for all 34 countries is just 10% greater than the consumption-based estimate.

200. It is difficult to assess the uncertainty in the Committee's estimates. Much of the dose commitment has not yet been experienced, and can only be calculated on the basis of projection models. The general methodology for projections used by the Committee, has been developed after some years of studying the transfer factors for ^{137}Cs, the radionuclide of primary concern. The comparison of the calculations by the Committee for the first year and the calculations by individual countries (Table 18) showed reasonable agreement. When the first-year integrated body burdens calculated by the Committee are compared with the actual measurements (Table 16), it can be seen that the estimate of effective dose equivalent commitment from ingestion may be high by perhaps 50%. As discussed above, a possible explanation for this discrepancy is the difficulty of knowing the radionuclide content of what is actually being consumed, given the limitations of food-sampling techniques. The Committee believes, accordingly, that its estimate is unlikely to be an underestimate of the effective dose equivalent commitment that will actually occur but that it might be an overestimate by a few tens of per cent.

D. COLLECTIVE DOSE COMMITMENT PER UNIT RELEASE

201. From estimates of the average ^{137}Cs deposition density in the regions included in Table 24, an estimate can be made of the total amount of ^{137}Cs released in the accident, independent of estimates that could be made near the reactor site at the time of the accident. The sum of the products of average deposition density and area for all land and ocean regions gives an estimated total ^{137}Cs deposit of 70 PBq. Of this total, some 42% was deposited within the USSR, 37% in Europe, 6% in the oceans and the remainder in the other regions of the northern hemisphere.

202. This estimated ^{137}Cs total deposit in the northern hemisphere may be compared with the original ^{137}Cs release estimate of 38 PBq ± 50% (Table 1). These estimates are in reasonable agreement, given the magnitude of the uncertainties associated with each estimate. The estimated release of 70 PBq would correspond to about 25% of the ^{137}Cs calculated to have been in the reactor core.

203. The reported release of ^{134}Cs from the damaged reactor was about 10% of the core inventory (Table 1). Based on the higher estimate of ^{137}Cs release and on the activity relationship, the ^{134}Cs release could have been 35 PBq, corresponding to a percentage release of 18%. If the release of ^{131}I, originally estimated to have been 20% of the total ^{131}I in the core, was, instead, 25%, the estimated release would be 330 PBq.

204. From the calculations or estimates of the collective effective dose equivalent commitments listed in Table 24, it may be determined that 430,000 man Sv is due to ^{137}Cs, 120,000 man Sv to ^{134}Cs, and 37,000 man Sv (collective effective dose) to ^{131}I. The remaining 20,000 man Sv was contributed by shorter-lived radionuclides deposited immediately after the accident.

205. From these values, the collective effective dose equivalent commitments per unit release of the major radionuclides may be estimated as follows:

^{137}Cs: 430,000 man Sv/ 70 PBq = $6\ 10^{-12}$ man Sv per Bq
^{134}Cs: 120,000 man Sv/ 35 PBq = $3\ 10^{-12}$ man Sv per Bq
^{131}I: 37,000 man Sv/330 PBq = $1\ 10^{-13}$ man Sv per Bq

For the thyroid dose equivalent from ^{131}I, the estimate would be the above value divided by the thyroid weighting factor of 0.03.

206. These estimates pertain to the particular conditions that prevailed at the time of the accident, but they may be a useful point of reference for this type of radiation source. For comparison, the collective effective dose equivalent commitments per unit release from another source, atmospheric nuclear testing, are as follows [U1]:

^{137}Cs: 2,200,000 man Sv/960 PBq = $2\,10^{-12}$ man Sv per Bq
^{131}I: 110,000 man Sv/700 EBq = $2\,10^{-16}$ man Sv per Bq

These resulted from releases largely into the stratosphere and apply to world populations of $3.2\,10^9$ persons (for ^{131}I) at the time of the main releases and $4\,10^9$ persons (for ^{137}Cs) during the main exposure period. Because the fallout from weapons tests was injected into the stratosphere, a longer time elapsed for decay of ^{131}I before deposition.

207. Estimates of collective effective doses per unit release have also been made for modelled dispersion from nuclear installations (Annex B). Based on a population density of 25 persons per km^2, these estimates are [W5]:

^{137}Cs: $5\,10^{-12}$ man Sv per Bq
^{131}I: $4\,10^{-13}$ man Sv per Bq

E. COLLECTIVE DOSE COMMITMENTS FROM OTHER RADIONUCLIDES

208. This assessment has accounted for the main radionuclides contributing to the collective dose. A few other radionuclides in the release from the accident were widely dispersed and could be considered as additional contributors to the total collective dose commitment. For completeness, the collective effective dose equivalent commitments may be summarized as follows:

Radionuclide	*Release (PBq)*	*Dose factor (man Sv per PBq)*	*Collective effective dose equivalent commitment (man Sv)*
H-3	2	0.4	1
C-14	0.005	110000	550
Kr-85	33	0.21	7
Xe-133	1700	0.05	85
I-129	0.00003	170000	5
Total			650

209. The amounts of noble gases ^{85}Kr and ^{133}Xe in the reactor core, which were assumed to be entirely released, were given in Table 1. Releases of ^{3}H, ^{14}C, and ^{129}I were not reported, but their generation rates in the reactor are assumed, roughly, to be 1,000, 10 and 0.05 GBq per MW a, respectively, which may be compared to the ^{85}Kr generation rate of 14,000 GBq per MW a [W5]. The percentage release has been taken as 100% for ^{3}H and (as for ^{137}Cs) 25% for ^{14}C and ^{129}I. The ^{129}I dose has been truncated at 10,000 years. The doses from ^{129}I and ^{14}C are delivered over long times but at very low dose rates. The collective effective dose equivalent commitment from these radionuclides is negligible.

VII. SUMMARY

210. The accident at the Chernobyl nuclear power station was a serious occurrence, indeed a tragic event for the people most closely affected in the USSR. The material costs of control, resettlement and decontamination have been enormous. Some of the people who dealt with the emergency lost their lives. Although populations were exposed in the countries of Europe and, to a lesser extent, in countries throughout the northern hemisphere, the radiation exposures were, in perspective, not of great magnitude.

211. The detectability of radiation in very small concentrations has allowed extensive measurement of the released radioactive materials in the environment, and it has been possible to make a complete inventory of ^{137}Cs, the main component of the release. The amount 70 PBq of ^{137}Cs corresponds to 22 kg of caesium, which was, however, dispersed across an entire hemisphere of the earth. Radionuclides are a unique class of substance whose environmental behaviour can be studied in detail at such trace levels.

212. In Europe, the highest effective dose equivalents in the first year were 760 μSv in Bulgaria, 670 μSv in Austria, 590 μSv in Greece and 570 μSv in Romania, followed by other countries of northern, eastern and south-eastern Europe (Table 18). For reference, the average annual effective dose equivalent from natural sources is 2,400 μSv. The doses in countries farther to the west in Europe and in the countries of Asia, Africa and North and South America were much less, which is in accord with the deposition pattern.

213. Exposures, mainly from released ^{137}Cs, will continue for a few tens of years from the external irradiation and ingestion pathways. Estimates of dose commitments have been made for larger geographical regions, based on projection models developed from fallout measurement experience. Transfer factors derived for northern, temperate and southern latitudes provide estimates of the effective dose equivalent commitment from all radionuclides and all pathways referred to the deposition density of ^{137}Cs. From the ^{137}Cs deposition versus distance relationship, dose estimates for the entire northern hemisphere are obtained. The estimated collective effective dose equivalent commitment from the accident is of the order of 600,000 man Sv.

214. This assessment of radiation exposures from the Chernobyl accident has dealt with the main radionuclides and pathways that contribute to the collective dose. It is recognized that many more features of exposure from other radionuclides and other pathways have been and continue to be investigated in various countries. The Committee will undoubtedly wish to review these findings in the expectation that they will lead to a better understanding of the behaviour and effects of radionuclides in the environment and to improved methods for assessing radiation exposure.

Table 1

Core inventory and estimate of total release of radionuclides [11]

Radio-nuclide	Half-life a/	Inventory (EBq) b/	Percentage released c/
Kr-85	10.72 a	0.033	~ 100
Xe-133	5.25 d	1.7	~ 100
I-131	8.04 d	1.3	20
Te-132	3.26 d	0.32	15
Cs-137	30.0 a	0.29	13
Cs-134	2.06 a	0.19	10
Sr-89	50.5 d	2.0	4
Sr-90	29.12 a	0.2	4
Zr-95	64.0 d	4.4	3
Mo-99	2.75 d	4.8	2
Ru-103	39.3 d	4.1	3
Ru-106	368 d	2.1	3
Ba-140	12.7 d	2.9	6
Ce-141	32.5 d	4.4	2
Ce-144	284 d	3.2	3
Np-239	2.36 d	0.14	3
Pu-238	87.74 a	0.001	3
Pu-239	24065 a	0.0008	3
Pu-240	6537 a	0.001	3
Pu-241	14.4 a	0.17	3
Cm-242	163 d	0.026	3

a/ Reference: [15]
b/ Decay corrected to 6 May 1986.
c/ Stated accuracy: ± 50%, except for noble gases.

Table 2

Activity ratios of radionuclides released in the Chernobyl accident relative to caesium-137 [13]

Date	Zr-95	Ru-103	Ru-106	I-131	Ba-140	Ce-141	Ce-144
30 April 1986	0.4	2.1	0.3	4.0		0.6	0.7
1 May 1986	1.5	5.2	0.8	13		3.2	5.5
2 May 1986	6.7	2.9	0.8	4.1	5.7	5.5	4.3
4 May 1986	5.3	5.2	1.1	6.2	9.6	4.8	3.8
6 May 1986	11	6.4	3.8	4.1	5.1	10	11

Table 3

Effective dose equivalent factors for cloud gamma irradiation
[K5]

Radionuclide	Effective dose equivalent per unit time-integrated concentration in air (nSv per Bq d/m^3)
Sr-89	0.033
Sr-90	0.008
Zr-95	2.88
Nb-95	2.99
Mo-99 a/	1.04
Ru-103 a/	1.81
Ru-106 a/	0.874
Ag-110m	10.7
Cd-115 a/	1.39
Sb-125	1.61
Sb-127	2.55
Te-129m a/	0.324
Te-131m a/	2.90
I-131	1.44
Te-132 a/	9.78
I-133	2.33
Cs-134	6.03
Cs-136	8.47
Cs-137 a/	2.30
Ba-140	0.718
La-140	9.26
Ce-141	0.293
Ce-143	1.01
Ce-144 a/	0.275
Np-239	0.636

a/ Includes daughter radionuclide.

Table 4

Dose equivalent factors for the inhalation of radionuclides
[H3, N3]

Radionuclide	Inhalation class	Thyroid dose equivalent per unit inhaled activity (nSv/Bq)		Effective dose equivalent per unit inhaled activity (nSv/Bq)	
	a/	Infants	Adults	Infants	Adults
Sr-89	D	8.0	0.41	21	1.8
Sr-90	D	22	2.2	130	59
Zr-95	W	2.2	0.78	26	4.3
Nb-95	Y	0.42	0.36	27	1.6
Mo-99 b/	Y	0.23	0.033	7.9	1.1
Ru-103 b/	Y	0.82	0.26	8.0	2.4
Ru-106 b/	Y	12	1.7	900	130
Ag-110m b/	Y	38	6.4	210	22
Cd-115	Y	0.12	0.018	8.9	1.1
Sb-125	W	2.1	0.32	27	3.3
Sb-127	W	0.39	0.062	12	1.6
Te-129m b/	W	1.1	0.16	4.7	6.5
Te-131m b/	W	180	33	21	1.6
Te-132 b/	W	260	58	37	2.5
I-131	D	2200	270	66	8.1
I-133	D	420	44	14	1.5
Cs-134	D	6.5	11	7.3	13
Cs-136	D	4.2	1.7	4.7	2.0
Cs-137 b/	D	5.6	7.9	6.4	8.6
Ba-140	D	1.5	0.26	8.2	1.0
La-140	W	0.20	0.069	8.6	1.3
Ce-141	Y	0.039	0.025	17	2.4
Ce-143	Y	0.045	0.0062	6.8	0.92
Ce-144 b/	Y	1.4	0.29	700	100
Np-239	W	0.043	0.0058	4.7	0.66

a/ D, W, Y refer to retention times in the lungs (days, weeks and years, respectively).
b/ Includes daughter radionuclide.

Table 5

Effective dose equivalent factors
for external irradiation from deposited radionuclides
[B10]

Radio-nuclide	Effective dose equivalent per unit deposition density for outdoor exposure (nSv per Bq/m^2)	
	30 days to 1 year	After 1 year
Ru-103	0.691	0.00128
Ru-106	2.09	1.65
I-131	0.015	0.0
Cs-134	18.6	36.2
Cs-137	8.04	264

Table 6

Dose equivalent factors for the ingestion of radionuclides
[H3, N3]

Radio-nuclide	Thyroid dose equivalent per unit ingested activity (nSv/Bq)		Effective dose equivalent per unit ingested activity (nSv/Bq)	
	Infants	Adults	Infants	Adults
I-131	3500	430	110	13
Cs-134	11	18	12	20
Cs-137	9	13	9.3	14

Table 7

Parameters of caesium-137 deposition to diet transfer function derived from long-term fallout measurements
[U1, E7]
(Bq a/kg per kBq/m^2)

Country	First year transfer b_1	Transfer beyond first year $P_{23,2+}$	Total transfer P_{23}
Milk products			
Argentina	7.7	1.1	8.8
Denmark	3.0	2.8	5.8
United States	3.3	2.4	5.7
Average	4.7	2.1	6.8
Grain products			
Argentina	2.0	6.9	8.9
Denmark	3.3	23	27
United States	1.5	7.1	8.6
Average	2.3	12	15
Vegetables			
Argentina	2.1	2.3	4.4
Denmark	2.4	1.1	3.5
United States	1.4	0.7	2.1
Average	2.0	1.4	3.3
Fruit			
Argentina	0.5	2.6	3.1
Denmark	1.8	1.7	3.5
United States	1.7	1.8	3.6
Average	1.3	2.0	3.4
Meat			
Argentina	22	4.1	26
Denmark	12	12	24
United States	2.0	8.2	10
Average	12	8	20
TOTAL DIET			
Argentina	6.3	1.8	8.1
Denmark	4.0	8.0	12
United States	1.9	3.5	5.4
Average	4.1	4.4	8.5

Table 8

Area, population, food production and food consumption in assessed countries

Country	Area (10^3 km^2)	Population (10^6)		Food production (10^6 kg/a)					Food consumption per caput (kg/a)				
		Infants	Total	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat
NORTH EUROPE													
Denmark	43.1	0.07	5.11	1100	420	250	140	340	173	80	18	150	66
Finland	338.1	0.07	4.87	1340	460	61	420	330	263	73	6	169	71
Norway	323.9	0.06	4.16	1060	280	190	400	210	202	65	37	120	76
Sweden													
Region 1	115.8	0.02	1.30	280	25	-	46	54	222	77	36	121	56
Region 2	105.9	0.004	0.26	43	5	-	8	7	222	77	36	121	56
Region 3	219.2	0.09	6.79	1530	676	13	1190	413	222	77	36	121	56
CENTRAL EUROPE													
Austria	83.9	0.09	7.56	1260	653	253	1420	673	145	66	71	136	99
Czechoslovakia													
Region 1	18.7	0.03	2.06	800	430	67	220	200	134	132	25	107	86
Region 2	75.0	0.15	10.40	4900	2900	270	870	1300	134	132	25	107	86
Region 3	34.2	0.04	3.02	1000	530	120	390	250	134	132	25	107	86
German Dem. Rep.													
Region 1	19.3	0.03	2.34	380	350	230	190	410	115	97	45	266	92
Region 2	25.8	0.04	2.62	620	420	280	230	540	115	97	45	266	92
Region 3	63.2	0.16	11.67	1600	1000	680	4100	1500	115	97	45	266	92
Germany, Fed. Rep. of													
Region 1	142.4	0.48	40.96	3000	2600	1440	4600	3200	108	80	23	112	55
Region 2	59.5	0.15	12.39	1200	890	580	1900	750	108	80	23	112	55
Region 3	46.8	0.09	7.68	1700	700	150	470	750	108	80	23	112	55
Hungary													
Region 1	35.2	0.06	5.16	252	391	301	137	815	185	110	25	160	80
Region 2	57.8	0.07	5.46	503	783	776	678	1630	185	110	25	160	80
Poland	312.7	0.64	37.46	16000	23200	3440	6900	2740	160	180	20	132	67
Romania													
Region 1	99.2	0.12	7.63	1360	1660	390	2570	905	150	190	40	240	86
Region 2	59.9	0.09	5.62	820	1000	235	1550	546	150	190	40	240	86
Region 3	78.4	0.15	9.48	1080	1310	310	2030	716	150	190	40	240	86
Switzerland													
Region 1	3.7	0.003	0.30	102	31	11	72	48	180	99	29	230	110
Region 2	12.4	0.02	2.03	345	105	38	244	162	180	99	29	230	110
Region 3	12.5	0.03	2.36	347	105	38	246	163	180	99	29	230	110
Region 4	12.7	0.02	1.80	355	107	39	251	167	180	99	29	230	110
WEST EUROPE													
Belgium	30.5	0.14	9.86	920	520	540	750	440	180	65	55	150	40
France													
Region 1	148.0	0.15	10.50	4150	1500	1300	1700	2100	130	84	84	132	73
Region 2	270.0	0.41	28.90	2520	5800	2900	6200	1300	130	84	84	132	73
Region 3	133.0	0.20	14.20	1330	600	1850	700	500	130	84	84	132	73
Ireland	70.3	0.08	3.54	100	220	270	390	220	163	68	40	69	50
Luxembourg	2.6	0.005	0.37	80	40	12	60	30	110	95	33	150	88
Netherlands	37.3	0.22	14.49	2100	310	2800	490	2200	145	65	65	135	70
United Kingdom													
Region 1	138.1	0.72	48.06	11000	5000	1200	4500	530	163	68	40	100	71
Region 2	86.5	0.10	6.56	730	780	88	1200	210	163	68	40	100	71
Region 3	19.5	0.02	1.25	730	220	13	180	96	163	68	40	100	71
SOUTH EUROPE													
Bulgaria													
Region 1	48.6	0.06	3.71	630	460	280	580	280	123	179	20	76	64
Region 2	62.3	0.08	5.18	760	550	340	700	340	123	179	20	76	64
Greece													
Region 1	56.8	0.04	4.22	300	400	170	800	200	80	100	30	250	60
Region 2	75.2	0.10	5.61	500	500	220	2000	300	80	100	30	250	60
Italy													
Region 1	119.8	0.23	26.03	10000	3000	1000	10000	1000	90	110	50	150	60
Region 2	181.5	0.39	30.88	2000	5000	2000	10000	600	90	110	50	150	60
Portugal	91.7	0.14	9.94	530	615	1630	953	425	45	125	113	105	42
Spain													
Region 1	100.9	0.10	7.50	820	828	1370	2100	478	104	88	124	132	62
Region 2	403.8	0.40	29.80	3280	3310	5490	8400	1910	104	88	124	132	62
Yugoslavia													
Region 1	89.5	0.13	7.87	2200	3400	230	4200	880	146	146	55	128	55
Region 2	153.5	0.22	13.5	3700	5700	380	7200	150	146	146	55	128	55
Region 3	12.8	0.02	1.12	300	400	40	60	70	146	146	55	128	55

Table 8, continued

Country	Area (10^3 km^2)	Population (10^6)		Food production (10^6 kg/a)					Food consumption per caput (kg/a)				
		Infants	Total	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat
USSR													
Region 1	207.6	0.17	10.01	6360	1300	217	754	1040	332	133	37	118	63
Region 2	269.4	0.35	21.94	10800	3050	785	2270	1800	332	133	37	118	63
Region 3	485.1	0.51	29.80	8270	1780	474	1510	1300	332	133	37	118	63
Region 4	4532.6	2.47	139.70	49300	20900	4630	14800	9400	332	133	37	118	63
Region 5	16696.3	2.12	77.37	17900	10000	1780	5670	3600	332	133	37	118	63
WEST ASIA													
Cyprus	9.3	0.01	0.64	50	20	100	100	50	83	94	87	315	83
Israel	20.7	0.08	3.87	470	200	660	950	220	120	130	140	190	60
Syria	185.2	0.18	8.98	1100	1900	300	1500	200	70	190	30	340	22
Turkey	774.8	1.00	52.00	5000	20000	5000	20000	2000	125	200	100	150	40
EAST ASIA													
China	9571.3	18.63	1046.39	5380	216000	28400	170000	30000	5	229	29	173	30
India	3287.3	25.38	750.90	26300	120000	18900	61000	3060	39	183	28	89	5
Japan	358.8	1.43	121.01	4660	13500	5600	16800	16900	50	193	30	180	120
NORTH AMERICA													
Canada	9215.4	0.38	25.36	4550	10600	281	5080	3980	181	93	21	301	130
United States	9372.6	3.75	238.74	39400	71800	5970	61600	31700	174	91	25	260	146

Table 9

Time-integrated concentrations of radionuclides in ground level air
(Bq d/m³)

Country	Sr-90	Zr-95	Mo-99	Ru-103	Ru-106	I-131	Te-132	Cs-134	Cs-136	Cs-137	Ba-140	Ce-141	Ce-144	Np-239
NORTH EUROPE														
Denmark	0.012	0.15		1.1	0.33	6.7	2.1	0.26	0.10	0.49	0.31	0.15	0.10	
Finland		0.43	1.4	1.8	0.53	210	14	3.8	1.5	6.5	3.4	0.47	0.36	2.1
Norway				(11)	(2.4)	(85)	(14)	(2.8)	(1.2)	(5.3)	(2.7)	(0.37)		
Sweden														
Region 1		0.22		1.0	0.37	23	0.83	0.44	0.14	0.78	0.47		0.14	
Region 2		0.002		0.33	0.096	4.6		0.13	0.032	0.23	0.07		0.003	
Region 3		0.39		2.1	0.60	28	1.2	0.60	0.20	1.10	0.76		0.25	
CENTRAL EUROPE														
Austria	(0.58)		(12)	(28)	5.8	120	69	8.14	(1.6)	14	(6.9)	(0.32)	(0.14)	(0.14)
Czechoslovakia														
Region 1				16	(3.6)	140	43	6.0	(1.8)	12				
Region 2				23	1.1	170	94	6.8	(2.1)	14				
Region 3				26	4.0	140	85	5.4	(1.7)	11				
German Dem. Rep.														
Region 1		0.21		14	(6.9)	160	72	11	2.8	23		0.60	0.21	
Region 2		0.22		6.1	(2.6)	66	27	4.1	0.58	8.5		0.045	0.026	
Region 3		(0.22)		(6.1)	(2.6)	(66)	(27)	(4.1)	(0.58)	(8.5)		(0.045)	(0.026)	
Germany, Fed.Rep.of														
Region 1				3.3	0.78	16	20	1.4	0.52	2.6	1.3		0.052	
Region 2				9.7	2.3	52	64	4.2	1.5	7.7	3.9		0.15	
Region 3				16	3.9	84	100	7.0	2.6	13	6.5		0.26	
Hungary														
Region 1			1.3	14	(1.8)	34	33	1.9	(0.54)	3.5	0.72	0.08	0.0006	
Region 2		0.04	1.3	17	2.2	25	20	2.3	0.74	4.8	2.6	0.17	0.13	
Poland		0.31	7.9	20	2.2	72	69	4.1	1.6	8.2	1.4	0.24	0.14	
Romania														
Region 1	(1.2)	(8.4)		14	3.6	43	(70)	5.1	(1.2)	11	(5.2)			
Region 2	(1.9)	(13)		84	21	340	(110)	8.0	(1.9)	17	(8.1)			
Region 3	(1.5)	(11)		(49)	(12)	(190)	(89)	(6.6)	(1.6)	(14)	(6.6)			
Switzerland														
Region 1			(2.3)	(7.8)	(2.6)	33	(22)	(2.1)	(0.43)	4.2	(2.0)	(0.073)		(0.27)
Region 2			(1.9)	(6.2)	(2.1)	30	(18)	(1.7)	(0.34)	3.3	(1.6)	(0.058)		(0.22)
Region 3			(2.6)	(8.5)	(2.9)	28	(24)	(2.3)	(0.47)	4.6	(2.2)	(0.080)		(0.30)
Region 4			(1.6)	(5.4)	(1.8)	28	(15)	(1.5)	(0.30)	2.9	(1.4)	(0.051)		(0.19)
WEST EUROPE														
Belgium	0.05			6.5	1.5	30	20	2.0	1.0	5.0	2.0			
France														
Region 1				(0.09)	(0.018)	0.30	(0.29)	0.03	(0.009)	0.06	(0.024)	(0.002)	(0.002)	
Region 2				0.81	0.16	2.7	2.7	0.27	0.081	0.54	0.22	0.016	0.016	
Region 3				(4.8)	(0.96)	24	(16)	1.4	(0.48)	3.2	(1.3)	(0.096)	(0.096)	
Ireland				0.16	0.044	1.0	0.37	0.058	0.022	0.11	0.086			
Luxembourg	0.05			6.5	1.5	30	20	2.0	1.0	5.0	2.0			
Netherlands			0.69	2.7	0.67	19	6.9	0.92	0.35	2.1	0.92			
United Kingdom														
Region 1,2				1.4	0.75	5.0	10	0.38	0.17	0.75	0.39			
Region 3				5.6	3.0	12	23	1.5	0.58	3.0	1.5			
SOUTH EUROPE														
Bulgaria														
Region 1,2		0.39	2.5	28	6.9	41	38	4.5		9.1	22	1.2	1.3	
Greece														
Region 1	1.0	2.0		40	8.0	40	30	7.5		10	2.5	2.3	1.9	
Region 2	1.0	2.0		40	7.0	40	30	7.5		10	2.5	2.3	1.9	
Italy														
Region 1				11	4.7	46	41	2.9	0.59	5.9	3.0			
Region 2				4.9	2.2	26	23	1.6	0.27	2.7	1.4			
Portugal				(0.04)	(0.012)	(0.07)	(0.004)	(0.01)	(0.004)	(0.02)	(0.001)		(0.006)	
Spain														
Region 1				(0.11)	(0.021)	(0.40)		(0.035)	(0.011)	(0.07)	(0.028)			
Region 2				(0.03)	(0.006)	(0.07)		(0.010)	(0.003)	(0.02)	(0.008)			
Yugoslavia														
Region 1			3.6	14	2.7	72	53	3.4		7.4	3.7			
Region 2			3.0	8.7	1.9	57	45	2.5		5.9	2.6			
Region 3			3.6	14	2.7	72	53	3.4		7.4	3.7			
USSR														
Region 1	3.7	15	6	58	12	490	110	29		54	56	12	7.6	190
Region 2	2.4	8.6	11	26	4	160	43	13		21	42	3.7	2.7	63
Region 3	0.7	2.7	8.7	18	4.5	120	34	6.9		13	11	2.7	1.8	28
Region 4	(0.5)	(0.7)	(1.3)	5.7	1.7	33	7.2	2.1		4.1	(3.1)	(0.68)	(0.47)	(4.6)
Region 5	(0.02)	(0.04)	(0.02)	(0.4)	(0.1)	(2.7)	(0.4)	(0.2)		(0.3)	(0.1)	(0.033)	(0.025)	(0.07)
WEST ASIA														
Cyprus						(20)		(3.5)		(7.0)				
Israel				14		20	14	3.4		6.5	15			
Syrian Arab Rep.						(0.16)		(0.015)		(0.03)				
Turkey				24		17	40	3.8	5.8	7.4	36	5.1	36	
EAST ASIA														
China			(0.22)	(0.92)	(0.20)	4.4	(2.6)	(0.34)	(0.099)	(0.66)	(0.39)			
India				(0.074)	(0.010)	0.17	0.016	0.010	0.003	0.035	0.010			
Japan		(0.0006)	(0.01)	(0.70)	(0.14)	3.4	(1.1)	0.14	(0.033)	0.28	(0.020)			
NORTH AMERICA														
Canada				(0.073)	0.029	0.14	(0.22)	(0.028)	(0.011)	0.055	(0.028)			
United States				(0.063)	(0.008)	0.27	(0.046)	(0.013)		(0.027)				

Numbers in parentheses are inferred values.

Table 10

Ratios of integrated concentrations of radionuclides in air to caesium-137

Country	Ru-103	Ru-106	I-131	Te-132	Cs-134	Cs-136	Ba-140	Ce-141
NORTH EUROPE								
Denmark	2.2	0.67	14	4.2	0.53	0.21	0.63	0.30
Finland	0.28	0.08	32	2.2	0.58	0.23	0.52	0.07
Norway	(2.0)	(0.45)	(16)	(2.6)	(0.53)	(0.23)	(0.51)	(0.07)
Sweden								
Region 1	1.3	0.47	29	1.1	0.56	0.18	0.59	
Region 2	1.4	0.42	20		0.57	0.14	0.30	
Region 3	1.9	0.55	25	1.1	0.55	0.18	0.69	
CENTRAL EUROPE								
Austria	(2.0)	0.40	8.2	4.8	0.57	(0.11)	(0.48)	(0.02)
Czechoslovakia								
Region 1	1.3	(0.30)	12	3.6	0.50			
Region 2	1.6	0.30	12	6.8	0.49			
Region 3	2.4	0.37	13	7.7	0.49			
German Dem. Rep.								
Region 1	0.61	(0.30)	6.9	3.2	0.50	0.12		0.03
Region 2,3	0.72	(0.30)	7.7	3.1	0.48	0.07		0.01
Germany, Fed.Rep.of								
Region 1	1.3	0.30	6.2	7.6	0.54	0.20	0.50	
Region 2	1.3	0.30	6.8	8.3	0.55	0.20	0.50	
Region 3	1.3	0.30	6.5	8.0	0.54	0.20	0.50	
Hungary								
Region 1	4.0	(0.52)	9.7	9.3	0.54	(0.16)		0.01
Region 2	3.5	(0.46)	5.3	4.3	0.48	0.16		0.04
Poland	2.5	0.26	8.8	8.4	0.50	0.19	0.16	0.03
Romania								
Region 1	1.3	0.33	4.0	(6.4)	0.48	(0.11)	(0.48)	
Region 2	5.0	1.3	20	(6.4)	0.48	(0.11)	(0.48)	
Region 3	(3.6)	(0.89)	(14)	(6.4)	(0.48)	(0.11)	(0.48)	
Switzerland								
Region 1	(1.9)	(0.63)	8.0	(5.3)	(0.50)	(0.10)	(0.48)	(0.02)
Region 2	(1.9)	(0.63)	9.0	(5.3)	(0.50)	(0.10)	(0.48)	(0.02)
Region 3	(1.9)	(0.63)	6.2	(5.3)	(0.50)	(0.10)	(0.48)	(0.02)
Region 4	(1.9)	(0.63)	9.7	(5.3)	(0.50)	(0.10)	(0.48)	(0.02)
WEST EUROPE								
Belgium	1.3	0.30	6.0	4.0	0.40	0.20	0.40	
France								
Region 1,2,3	1.5	0.30	5.0	(4.8)	0.50	0.15	0.40	0.03
Ireland	1.5	0.40	9.1	3.4	0.53	0.20	0.78	
Luxembourg	1.3	0.30	6.0	4.0	0.40	0.20	0.40	
Netherlands	1.3	0.32	8.9	3.3	0.44	0.17	0.44	
United Kingdom								
Region 1,2	1.9	1.0	6.7	13	0.51	0.23	0.52	
Region 3	1.9	1.0	3.9	7.7	0.50	0.19	0.50	
SOUTH EUROPE								
Bulgaria								
Region 1,2	3.1	0.76	4.5	4.2	0.49		2.4	0.13
Greece								
Region 1	4.0	0.80	4.0	7.0	0.75		0.25	0.23
Region 2	4.0	0.70	4.0	7.0	0.75		0.25	0.23
Italy								
Region 1	1.8	0.80	7.8	7.0	0.49	0.10	0.50	
Region 2	1.8	0.81	9.6	8.5	0.59	0.10	0.50	
Portugal	(2.0)	(0.60)	(3.5)	(0.2)	(0.50)	(0.20)	(0.06)	
Spain								
Region 1	(1.5)	(0.30)	(5.7)		(0.50)	(0.15)	(0.40)	
Region 2	(1.5)	(0.30)	(3.5)		(0.50)	(0.15)	(0.40)	
Yugoslavia								
Region 1	1.8	0.36	9.7	7.2	0.46		0.50	
Region 2	1.5	0.32	9.7	7.6	0.42		0.44	
Region 3	1.8	0.36	9.7	7.2	0.46		0.50	
USSR								
Region 1	1.1	0.23	9.1	2.1	0.55		1.0	0.22
Region 2	1.2	0.19	7.6	2.0	0.62		2.0	0.17
Region 3	1.4	0.34	8.7	2.6	0.52		0.82	0.20
Region 4	1.4	0.40	8.1	1.8	0.50		(0.75)	(0.16)
Region 5	(1.3)	(0.33)	(9.0)	(1.3)	(0.67)		(0.33)	(0.11)

Table 10, continued

Country	Ru-103	Ru-106	I-131	Te-132	Cs-134	Cs-136	Ba-140	Ce-141
WEST ASIA								
Cyprus			(2.9)		(0.50)			
Israel	2.2		3.1	2.2	0.52		2.3	
Syrian Arab Rep.			(5.3)		(0.50)			
Turkey	3.3		2.4	5.4	0.51	0.78	4.8	0.69
EAST ASIA								
China	(1.4)	(0.30)	(6.7)	(3.9)	(0.51)	(0.15)	(0.59)	
India	(2.1)	(0.30)	5.0	(0.47)	0.29	0.10	0.29	
Japan	(2.5)	(0.50)	12	(3.9)	0.50	(0.12)	(0.07)	
NORTH AMERICA								
Canada	(1.3)	0.52	2.5	(4.0)	(0.50)	(0.20)	(0.50)	
United States	(2.4)	(0.30)	(10)	(1.7)	(0.50)			
MEDIAN VALUES	1.5	0.37	8.2	4.2	0.50	0.19	0.50	0.13

Numbers in parentheses are inferred values.

Table 11

Deposition of radionuclides

Country	Deposition density (kBq/m^2)					Ratios of deposition densities to caesium-137			
	Ru-103	Ru-106	I-131	Cs-134	Cs-137	Ru-103	Ru-106	I-131	Cs-134
NORTH EUROPE									
Denmark	1.9	0.59	(6.1)	0.65	1.29	1.5	0.5	(4.7)	0.5
Finland	19	12	100	7.6	15	1.3	0.8	7.0	0.5
Norway	(11)	2.4	(85)	2.8	5.3	(2.0)	0.5	(16)	0.5
Sweden									
Region 1	9.9	3.7	160	17	31	0.3	0.1	5.2	0.6
Region 2	2.3	0.85	13	0.45	0.81	2.8	1.0	16	0.6
Region 3	5.3	2.0	41	1.2	2.3	2.3	0.9	18	0.5
CENTRAL EUROPE									
Austria	31	(6.3)	120	(12)	23	1.3	(0.3)	5.0	(0.5)
Czechoslovakia									
Region 1	4.0	(0.72)	(26)	1.3	2.3	1.7	(0.3)	(11)	0.6
Region 2	6.3	(1.6)	(58)	2.7	5.3	1.2	(0.3)	(11)	0.5
Region 3	6.1	(0.85)	(30)	1.3	2.8	2.2	(0.3)	(11)	0.5
German Dem. Rep.									
Region 1	14	(1.8)	(45)	2.9	6.1	2.4	(0.3)	(7.4)	0.5
Region 2	23	(3.2)	42	5.7	11	2.1	(0.3)	3.9	0.5
Region 3	(14)	(1.8)	(19)	(2.9)	(6.1)	(2.4)	(0.3)	(3.1)	(0.5)
Germany, Fed. Rep. of									
Region 1	2.5	0.6	12	1.0	2.0	1.3	0.3	6.2	0.5
Region 2	5.0	1.2	27	2.0	4.0	1.3	0.3	6.8	0.5
Region 3	20	4.8	100	8.0	16	1.3	0.3	6.5	0.5
Hungary									
Region 1	12	(2.9)	30	2.4	4.8	2.5	(0.6)	6.3	0.5
Region 2	3.8	(0.90)	9.3	0.75	1.5	2.5	(0.6)	6.2	0.5
Poland	(13)	(1.6)	38	2.6	5.2	(2.5)	(0.3)	7.3	0.5
Romania									
Region 1	(13)	(3.3)	(24)	(2.1)	(4.5)	(2.9)	(0.7)	(5.2)	(0.5)
Region 2	(52)	(13)	(94)	(8.6)	(18)	(2.9)	(0.7)	(5.2)	(0.5)
Region 3	26	(6.5)	47	(4.3)	9.0	2.9	(0.7)	5.2	(0.5)
Switzerland									
Region 1	(28)	(9.3)	(30)	8.9	15	(1.9)	(0.6)	(2.0)	0.6
Region 2	(6.5)	(2.2)	25	2.1	3.5	(1.9)	(0.6)	7.2	0.6
Region 3	(3.8)	(1.3)	15	1.2	2.0	(1.9)	(0.6)	7.2	0.6
Region 4	(2.4)	(0.82)	9.4	0.78	1.3	(1.9)	(0.6)	7.2	0.6

Table 11, continued

Country	Deposition density (kBq/m²)					Ratios of deposition densities to caesium-137			
	Ru-103	Ru-106	I-131	Cs-134	Cs-137	Ru-103	Ru-106	I-131	Cs-134
WEST EUROPE									
Belgium	(1.4)	0.4	5.2	0.4	0.84	(1.7)	0.5	6.2	0.5
France									
Region 1	(0.27)	(0.054)	(0.9)	0.09	(0.18)	(1.5)	(0.3)	(5.0)	(0.5)
Region 2	(0.99)	(0.2)	(5.3)	0.33	(0.66)	(1.5)	(0.3)	(8.0)	(0.5)
Region 3	(3.2)	(0.96)	(24)	1.6	(3.2)	(1.0)	(0.3)	(7.5)	(0.5)
Ireland	4.9	1.3	10	1.7	3.4	1.5	0.4	3.1	0.5
Luxembourg	(4.5)	(1.3)	19	1.3	2.7	(1.7)	(0.5)	7.0	0.5
Netherlands	3.4	0.85	11	0.92	1.8	1.9	0.5	6.3	0.5
United Kingdom									
Region 1	0.18	0.06	0.8	0.05	0.1	1.8	0.6	8.0	0.5
Region 2	3.1	0.8	2.0	0.85	1.7	1.8	0.5	1.2	0.5
Region 3	5.5	1.4	6.0	1.5	3.0	1.8	0.5	2.0	0.5
SOUTH EUROPE									
Bulgaria									
Region 1	9.9	2.6	4.2	2.0	3.9	2.5	0.7	1.1	0.5
Region 2	30	7.9	13	6.2	12	2.5	0.7	1.1	0.5
Greece									
Region 1	33	3.0	36	4.0	8.0	4.1	0.4	4.5	0.5
Region 2	3.0	0.7	14	1.3	2.4	1.3	0.3	5.8	0.5
Italy									
Region 1	14	3.8	25	3.0	6.0	2.3	0.6	4.2	0.5
Region 2	7.0	2.0	15	2.0	4.0	1.8	0.5	3.8	0.5
Portugal	(0.04)	(0.012)	0.07	(0.01)	0.02	(2.0)	(0.6)	3.5	(0.5)
Spain									
Region 1	(0.11)	(0.021)	0.4	(0.035)	0.07	(1.5)	(0.3)	5.7	(0.5)
Region 2	(0.03)	(0.006)	(0.07)	(0.01)	(0.02)	(1.5)	(0.3)	(3.5)	(0.5)
Yugoslavia									
Region 1	33	7.0	140	9.0	23	1.4	0.3	5.9	0.4
Region 2	15	3.0	60	4.0	10	1.5	0.3	6.0	0.4
Region 3	6.0	1.3	24	1.7	4.0	1.5	0.3	6.0	0.4
USSR									
Region 1	41	8.8	590	21	39	1.1	0.2	15	0.6
Region 2	17	2.6	480	8.7	15	1.2	0.2	33	0.6
Region 3	13	3.2	160	5.2	10	1.3	0.3	16	0.5
Region 4	(3.7)	(1.1)	20	1.4	2.8	(1.4)	(0.4)	7.2	0.5
Region 5	(0.1)	(0.04)	(0.4)	(0.05)	0.09	(1.1)	(0.4)	(4.3)	(0.5)
WEST ASIA									
Cyprus			(2.0)	(0.3)	(0.6)	-	-	(3.3)	(0.5)
Israel	(1.6)		(0.7)	(0.2)	(0.4)	(4.0)	-	(1.8)	(0.5)
Syrian Arab Rep.				(0.06)	(0.13)	-	-	-	(0.5)
Turkey				2.0	4.0	-	-	-	(0.5)
EAST ASIA									
China	(0.21)	(0.044)	0.29	(0.075)	(0.15)	(1.4)	(0.3)	(2.0)	(0.5)
India	(0.073)	(0.011)	(0.044)	(0.010)	0.035	(2.1)	(0.3)	(1.3)	(0.3
Japan	(0.45)	(0.090)	1.6	0.087	0.18	(2.5)	(0.5)	9.0	0.5
NORTH AMERICA									
Canada	(0.04)	(0.016)	0.10	0.015	0.030	(1.3)	(0.5)	3.4	0.5
United States	(0.062)	(0.0079)	0.15	0.013	0.026	(2.4)	(0.3)	5.7	0.5
Median values						1.6	0.5	6.2	0.5

Numbers in parentheses are inferred values.

Table 12

Quotients of deposition density and time-integrated concentration in air for caesium-137

Country	Deposition density a/ (kBq/m^2)	Integrated concentration in air a/ (Bq d/m^3)	Quotient (cm/s)
NORTH EUROPE			
Denmark	1.3	0.49	3.1
Finland	11	6.5	1.9
Norway	7.1	(5.3)	(1.6)
Sweden	9.5	0.8	14
CENTRAL EUROPE			
Austria	23	14	1.9
Czechoslovakia	4.2	13	0.4
German Dem. Rep.	7.2	11	0.8
Germany, Fed. Rep. of	5.1	5.8	1.0
Hungary	2.7	4.3	0.7
Poland	5.2	8.2	0.7
Romania	(9.4)	(13)	(0.8)
Switzerland	3.4	3.7	1.1
WEST EUROPE			
Belgium	0.84	5	0.2
France	1.1	1.1	1.2
Ireland	3.3	0.11	35
Luxembourg	2.7	5	0.6
Netherlands	1.8	2.1	1.0
United Kingdom	0.9	0.9	1.2
SOUTH EUROPE			
Bulgaria	8.5	9.1	1.1
Greece	4.8	10	0.6
Italy	4.8	4.0	1.4
Portugal	0.02	(0.02)	(1.2)
Spain	0.03	(0.03)	(1.2)
Yugoslavia	14	7.0	2.4
USSR	1.4	2.1	0.8
WEST ASIA			
Cyprus	(0.6)	(7.0)	(0.1)
Israel	(0.4)	(6.5)	(0.07)
Syrian Arab Rep.	(0.13)	(0.03)	(5.0)
Turkey	4.0	7.4	0.6
EAST ASIA			
China	(0.15)	(0.66)	(0.3)
India	0.035	0.035	1.2
Japan	0.18	0.28	0.7
NORTH AMERICA			
Canada	0.030	0.055	0.6
United States	0.026	(0.027)	(1.1)

a/ Area-weighted average values.

Table 13

Outdoor effective dose equivalent in the first month from external irradiation per unit caesium-137 deposition

Country	Effective dose equivalent in first month (μSv)	Population-weighted deposition density of caesium-137 (kBq/m^2)	Effective dose equivalent per unit caesium-137 deposition (μSv per kBq/m^2)
NORTH EUROPE			
Denmark	(17)	1.3	(13)
Finland	(210)	15	(14)
Norway	(74)	5.3	(14)
Sweden			
Region 1	25	31	0.8
Region 2	6.6	0.8	8
Region 3	18	2.3	8
CENTRAL EUROPE			
Austria	(220)	22	(10)
Czechoslovakia			
Region 1	93	2.3	40
Region 2	180	5.3	34
Region 3	140	2.8	52
German Dem. Rep.			
Region 1	200	6.1	33
Region 2	200	11	19
Region 3	100	(6.1)	(17)
Germany, Fed. Rep. of			
Region 1	(26)	2.0	(13)
Region 2	(51)	4.0	(13)
Region 3	210	16	13
Hungary			
Region 1	100	4.8	21
Region 2	42	1.5	28
Poland	14	5.2	3
Romania			
Region 1	(70)	(4.5)	(16)
Region 2	(280)	(18)	(16)
Region 3	(140)	9.0	(16)
Switzerland			
Region 1	(200)	15	(14)
Region 2	(120)	3.5	(34)
Region 3	(64)	2.0	(32)
Region 4	(48)	1.3	(37)
WEST EUROPE			
Belgium	(11)	0.8	(13)
France			
Region 1	(2.5)	0.2	(14)
Region 2	(9.2)	0.7	(14)
Region 3	(45)	3.2	(14)
Ireland	(40)	3.4	(12)
Luxembourg	(35)	2.7	(13)
Netherlands	(22)	1.8	(12)
United Kingdom			
Region 1	(1.2)	0.1	(12)
Region 2	(21)	1.7	(12)
Region 3	(36)	3.0	(12)
SOUTH EUROPE			
Bulgaria			
Region 1	120	3.9	32
Region 2	210	12	18
Greece			
Region 1	31	8.0	4
Region 2	12	2.4	5
Italy			
Region 1	67	6.0	11
Region 2	11	4.0	3
Portugal	(0.3)	0.02	(14)
Spain			
Region 1	(1.0)	0.07	(14)
Region 2	(0.3)	(0.02)	(15)
Yugoslavia			
Region 1	5.3	23	0.2
Region 2	0.6	10	0.06
Region 3	0.9	4	0.2

Table 13, continued

Country	Effective dose equivalent in first month (μSv)	Population-weighted deposition density of caesium-137 (kBq/m^2)	Effective dose equivalent per unit caesium-137 deposition (μSv per kBq/m^2)
USSR			
Region 1	1200	39	31
Region 2	860	15	59
Region 3	190	10	19
Region 4	86	2.8	31
Region 5	(1.3)	0.09	(14)
WEST ASIA			
Cyprus	(5.6)	(0.6)	(9)
Israel	(5.6)	(0.4)	(14)
Syrian Arab Rep.	(3.9)	(0.1)	(39)
Turkey	5.6	4.0	1
EAST ASIA			
China	(1.3)	(0.15)	(9)
India	(0.2)	0.04	(5)
Japan	(1.7)	0.18	(9)
NORTH AMERICA			
Canada	(0.13)	0.03	(4)
United States	(0.13)	0.026	(5)

Numbers in parentheses are inferred values.

Table 14

Iodine-131 in foods

Country	Latitude (degrees north)	Integrated concentration (Bq a/kg)		Normalized integrated concentration (Bq a/kg per kBq/m^2)		Ratio of integrated concentrations
		Milk products	Leafy vegetables	Milk products	Leafy vegetables	Leafy vegetables / milk products
NORTH EUROPE						
Denmark	56	0.14	(0.6)	0.02	0.1	(4.3)
Finland	63	0.9	3.5	0.01	0.03	3.9
Norway	61	(0.8)	(3.0)	(0.01)	(0.04)	(3.8)
Sweden						
Region 1	62	2	(2)	0.01	(0.01)	(1.0)
Region 2	66	(0)	(0)	(0)	(0)	(1.0)
Region 3	59	1	(1)	0.02	(0.02)	(1.0)
CENTRAL EUROPE						
Austria	48	12	(0)	0.1	(0)	-
Czechoslovakia						
Region 1	50	14	68	0.5	2.7	5.1
Region 2	50	15	68	0.3	1.2	4.6
Region 3	49	28	68	0.9	2.3	2.5
German Dem. Rep.						
Region 1	52	15	32	0.3	0.7	2.1
Region 2	53	10	15	0.2	0.4	1.5
Region 3	52	3.8	8.0	0.2	0.4	2.1
Germany, Fed. Rep. of						
Region 1	52	0.7	5.5	0.05	0.4	8.5
Region 2	49	2.6	12	0.1	0.4	4.6
Region 3	48	6.8	46	0.07	0.4	6.8
Hungary						
Region 1	47	10	15	0.3	0.5	1.5
Region 2	47	6	6	0.2	0.6	1.0
Poland	52	11	4	0.3	0.1	0.4

Table 14, continued

Country	Latitude (degrees north)	Integrated concentration (Bq a/kg)		Normalized integrated concentration (Bq a/kg per kBq/m^2)		Ratio of integrated concentrations
		Milk products	Leafy vegetables	Milk products	Leafy vegetables	Leafy vegetables / milk products
Romania						
Region 1	46	(11)	(9.1)	(0.5)	(0.4)	(0.8)
Region 2	46	(44)	(36)	(0.5)	(0.4)	(0.8)
Region 3	45	22	18	0.5	0.4	0.8
Switzerland						
Region 1	46	37	55	(1.2)	(1.9)	1.5
Region 2	47	26	54	1.0	2.1	2.0
Region 3	47	23	30	1.6	2.0	1.3
Region 4	47	7.3	32	0.8	3.4	4.4
WEST EUROPE						
Belgium	51	2.9	5.0	0.6	1.0	1.7
France						
Region 1	47	0.6	1.4	0.7	1.6	2.3
Region 2	47	1.5	3.3	0.3	0.6	2.2
Region 3	47	4.4	10	0.2	0.4	2.3
Ireland	53	3.1	12	0.3	1.2	4.0
Luxembourg	50	3.1	18	0.2	1.0	5.8
Netherlands	52	1.0	8.0	0.09	0.7	8.0
United Kingdom						
Region 1	53	0.8	(0.8)	1.0	(1.0)	(1.0)
Region 2	56	1.7	(1.7)	0.9	(0.9)	(1.0)
Region 3	55	2.1	(2.1)	0.4	(0.4)	(1.0)
SOUTH EUROPE						
Bulgaria						
Region 1	43	34	(34)	8.1	8.2	(1.0)
Region 2	42	34	(34)	2.6	2.7	(1.0)
Greece						
Region 1	41	36	150	1.0	4.1	4.1
Region 2	39	14	56	1.0	4.0	4.0
Italy						
Region 1	45	11	65	0.4	2.6	5.9
Region 2	42	11	12	0.7	0.8	1.1
Portugal	40	0.01	0.04	0.1	0.6	4.0
Spain						
Region 1	40	0.7	1.2	1.8	3.0	1.7
Region 2	40	(0.01)	(0.04)	(0.1)	(0.6)	(4.0)
Yugoslavia						
Region 1	45	24	210	0.2	1.6	8.8
Region 2	43	11	90	0.2	1.5	8.2
Region 3	43	3.6	31	0.15	1.3	8.6
USSR						
Region 1	55	25	13	0.04	0.02	0.5
Region 2	54	31	42	0.06	0.09	1.4
Region 3	52	4.4	2.2	0.03	0.01	0.5
Region 4	51	4.9	(0)	0.3	(0)	-
Region 5	53	(0.1)	(0)	(0.3)	(0)	-
WEST ASIA						
Cyprus	35	(6.0)	(24)	(3.0)	(12)	(4.0)
Israel	32	(1.6)	(15)	(2.3)	(21)	(9.4)
Syrian Arab Rep.	35	(2.0)	(0.6)	-	-	(0.3)
Turkey	40	3.0	2.5	-	-	0.8
EAST ASIA						
China	32	0.48	2.2	1.6	7.4	4.5
India	23	0.094	0.095	(2.2)	(2.2)	(1.0)
Japan	36	0.14	4.6	0.09	2.9	33
NORTH AMERICA						
Canada	55	(0.10)	(0.10)	(1.0)	(1.0)	(1.0)
United States	36	(0.15)	(0.15)	(1.0)	(1.0)	(1.0)

Numbers in parentheses are inferred values.

Table 15

Caesium-137 in foods during the first year

Country	Integrated concentration (Bq a/kg)					Normalized integrated concentration (Bq a/kg per kBq/m²)					Ratio of integrated concentrations	
	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Leafy veg. / milk	Meat/ milk
NORTH EUROPE												
Denmark	1.6	2.1	0.5	0.8	1.3	1.2	1.6	0.4	0.6	1.0	0.3	0.8
Finland	21	3.8	2.9	9.4	66	1.4	0.3	0.2	0.6	4.5	0.1	3.1
Norway	14	1.2	7.0	4.3	44	2.6	0.2	1.3	0.8	8.3	0.5	3.1
Sweden												
Region 1	18	3	10	20	33	0.6	0.1	0.3	0.7	1.1	0.6	1.8
Region 2	(6)	(3)	(5)	(7)	(11)	(7.4)	(3.7)	(6.2)	(8.6)	(14)	(0.8)	(1.8)
Region 3	6	3	5	7	11	2.6	1.3	2.2	3.0	4.8	0.8	1.8
CENTRAL EUROPE												
Austria	44	15	18	26	57	1.9	0.7	0.8	1.1	2.5	0.4	1.3
Czechoslovakia												
Region 1	7.4	4.9	8	33	15	3.2	2.1	3.4	14	6.5	1.1	2.0
Region 2	7.8	13	8	33	15	1.5	2.5	1.5	6.3	2.9	1.0	1.9
Region 3	9.6	8.1	8	33	15	3.5	2.9	2.9	12	5.5	0.8	1.6
German Dem. Rep.												
Region 1	6.0	15	0.3	2.6	8.4	1.0	2.5	0.05	0.4	1.4	0.05	1.4
Region 2	17	23	0.8	5.1	20	1.6	2.1	0.07	0.5	1.9	0.05	1.2
Region 3	4.2	12	0.3	3.3	12	(0.7)	(2.0)	(0.05)	(0.5)	(2.0)	0.07	2.9
Germany, Fed. Rep. of												
Region 1	4.6	4.5	2.3	3	8	2.3	2.3	1.2	1.5	4.0	0.5	1.7
Region 2	6.7	9.0	4.5	6	16	1.7	2.3	1.2	1.5	4.0	0.7	2.4
Region 3	24	36	18	24	64	1.5	2.3	1.2	1.5	4.0	0.8	2.7
Hungary												
Region 1	13	9	12	10	25	2.7	1.9	2.5	2.1	(5.2)	0.9	1.9
Region 2	8	5	7	5	25	5.3	3.3	4.7	3.3	(17)	0.9	3.1
Poland	25	2	10	10	34	4.8	0.4	2.0	1.9	6.4	0.4	1.3
Romania												
Region 1	(9.7)	(12)	(4.8)	(3.6)	(28)	(2.2)	(2.7)	(1.1)	(0.8)	(6.3)	(0.5)	(2.9)
Region 2	(39)	(49)	(19)	(14)	(110)	(2.2)	(2.7)	(1.1)	(0.8)	(6.3)	(0.5)	(2.9)
Region 3	19	(24)	9.5	(7.2)	56	2.2	(2.7)	1.1	(0.8)	6.3	0.5	2.9
Switzerland												
Region 1	48	(30)	54	(12)	200	3.2	(2.0)	3.7	(0.8)	14	1.1	4.3
Region 2	11	(7.0)	16	(2.8)	24	3.2	(2.0)	4.5	(0.8)	6.9	1.4	2.1
Region 3	8.9	(4.1)	9.1	(1.6)	12	4.4	(2.0)	4.5	(0.8)	5.8	1.0	1.3
Region 4	2.7	(2.6)	4.8	(1.0)	12	2.1	(2.0)	3.7	(0.8)	9.0	1.8	4.4
WEST EUROPE												
Belgium	1.5	(1.7)	1.1	(0.7)	(2.6)	1.8	(2.0)	1.3	(0.8)	(3.1)	0.7	(1.7)
France												
Region 1	0.18	0.37	0.13	0.08	0.05	1.0	2.1	0.7	0.4	0.3	0.7	0.3
Region 2	2.7	1.2	4.4	1.2	0.8	4.1	1.8	6.7	1.8	1.2	1.6	0.3
Region 3	13	6.2	9.6	5.6	4.0	4.1	1.9	3.0	1.8	1.3	0.7	0.3
Ireland	10	(6.7)	3.0	(2.7)	18	3.0	(2.0)	0.9	(0.8)	5.4	0.3	1.8
Luxembourg	4.9	(5.4)	(3.5)	(2.2)	(8.1)	1.8	(2.0)	(1.3)	(0.8)	(3.0)	(0.7)	(1.7)
Netherlands	1.5	(3.6)	(2.3)	(1.4)	(5.4)	0.9	(2.0)	(1.3)	(0.8)	(3.0)	(1.5)	(3.5)
United Kingdom												
Region 1	0.9	(0.2)	(0.2)	(0.08)	(1.8)	9	(2.0)	(2.0)	(0.8)	(18)	(0.2)	(2.0)
Region 2	10	(3.4)	(2)	(1.4)	(20)	5.9	(2.0)	(1.2)	(0.8)	(12)	(0.2)	(2.0)
Region 3	18	(6.0)	(4)	(2.4)	(36)	6	(2.0)	(1.3)	(0.8)	(12)	(0.2)	(2.0)
SOUTH EUROPE												
Bulgaria												
Region 1	38	13	(27)	41	200	9.7	3.3	(6.9)	10	51	(0.7)	5.3
Region 2	38	13	(27)	41	200	3.1	1.1	(2.3)	3.4	17	(0.7)	5.3
Greece												
Region 1	76	60	46	46	61	9.5	7.5	5.8	5.8	7.6	0.6	0.8
Region 2	23	20	19	14	18	9.6	8.3	7.9	5.8	7.5	0.8	0.8
Italy												
Region 1	22	8	27	15	70	3.7	1.3	4.5	2.5	12	1.2	3.2
Region 2	13	12	3.5	10	60	3.3	3.0	0.9	2.5	15	0.3	4.6
Portugal	0.16	(0.2)	(0.1)	(0.04)	(0.4)	8.0	(10)	(5.0)	(2.0)	(20)	(0.6)	(2.6)
Spain												
Region 1	0.8	(0.7)	(0.5)	(0.2)	(2)	11	(10)	(7.1)	(2.9)	(29)	(0.6)	(2.6)
Region 2	(0.16)	(0.2)	(0.1)	(0.04)	(0.4)	(8.0)	(10)	(5.0)	(2.0)	(20)	(0.6)	(2.6)
Yugoslavia												
Region 1	49	2.6	10	1.	77	2.1	0.1	0.4	0.04	3.3	0.2	1.6
Region 2	20	1.1	4.3	0.5	33	2.0	0.1	0.4	0.05	3.3	0.2	1.6
Region 3	7	0.4	1.5	0.15	11	1.8	0.1	0.4	0.04	2.8	0.2	1.6
USSR												
Region 1	90	23	25	33	200	2.3	0.6	0.6	0.9	5.2	0.3	2.2
Region 2	26	6.0	17	8.0	55	1.8	0.4	1.2	0.6	3.8	0.7	2.1
Region 3	21	6.0	10	10	50	2.1	0.6	1.0	1.0	5.0	0.5	2.4
Region 4	4.2	(1.7)	(2.1)	(2.6)	14	1.5	(0.6)	(0.8)	(0.9)	5.1	(0.5)	3.3
Region 5	(0.16)	(0.05)	(0.04)	(0.06)	(0.50)	(1.7)	(0.5)	(0.4)	(0.6)	(5.3)	(0.3)	(3.1)

Table 15, continued

Country	Integrated concentration (Bq a/kg)					Normalized integrated concentration (Bq a/kg per kBq/m^2)					Ratio of integrated concentrations	
	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Leafy veg. / milk	Meat/ milk
WEST ASIA												
Cyprus	(3.0)	(2.0)	(3.0)	(0.6)	(2.0)	(5.0)	(3.3)	(5.0)	(1.0)	(3.3)	(1.0)	(0.7)
Israel	(0.8)	(4.0)	(5.0)	(2.0)	(10)	(2.0)	(10)	(13)	(5.0)	(25)	(6.3)	(13)
Syrian Arab Rep.	(1.0)	(0.3)	(0.05)	(0.02)	(0.3)	(7.7)	(2.3)	(0.4)	(0.2)	(2.3)	(0.05)	(0.3)
Turkey	21	2.0	6.5	3.5	17	5.3	0.5	1.6	0.9	4.3	0.3	0.8
EAST ASIA												
China	(0.18)	(0.47)	(1.1)	(0.19)	(0.78)	(1.2)	(3.2)	(7.4)	(1.3)	(5.4)	(6.2)	(4.5)
India	(0.16)	(0.39)	0.11	(0.046)	(0.18)	(4.7)	(11.2)	3.2	(1.3)	(5.1)	(0.7)	(1.1)
Japan	0.22	0.056	0.29	(0.28)	0.29	1.2	0.3	1.6	(1.6)	1.6	1.3	1.4
NORTH AMERICA												
Canada	(0.05)	(0.08)	(0.03)	(0.02)	(0.09)	(1.5)	(2.5)	(1.0)	(0.8)	(3.0)	(0.7)	(2.0)
United States	(0.04)	(0.13)	(0.03)	(0.02)	(0.08)	(1.5)	(5)	(1.0)	(0.8)	(3.0)	(0.7)	(2.0)

Numbers in parentheses are inferred values.

Table 16

Comparison of body burdens of caesium-137 derived from measurements in man and expected from foodstuff concentrations (first-year intakes)

Country	Number of persons	Caesium-137 deposition density (kBq/m^2)	Time-integrated body burdens (Bq a)		Body burden ratio (measured/ expected)	Ref.
			Measured in man	Expected from diet		
Austria						
Vienna	4	4	2500	1200	2.1	[01]
Country average	200	23	2800	7000	0.4	[S19]
Bulgaria						
Country average	308	8.6	2600	9200	0.3	[C4]
Czechoslovakia						
Country average	404	4.4	960	3000	0.3	[M7]
Finland						
Region 1	102	2	1500	610	2.5	[R14]
Region 2	27	6	1650	1800	0.9	[R14]
Region 3	31	15	3300	4600	0.7	[R14]
Region 4	41	34	4500	10000	0.4	[R14]
Region 5	16	52	5400	16000	0.3	[R14]
Country average		15	2730	4500	0.6	[R14]
France						
Region 1		0.18	130	40	3.3	[L5]
Region 2		0.66	270	430	0.6 a/	[L5]
Region 3		3.2	540	1700	0.3 a/	[L5]
German Dem. Rep.						
Country average	300	6.8	1000	1700	0.6	[L1]
Germany, Fed. Rep. of						
Region 1		2	490	670	0.7	[S16]
Regions 2,3		8.6	1200	2600	0.5	[S16]
Hungary						
Country average	39	3.1	770	2300	0.3	[H4]
Italy						
Region 1	43	6	3500	4200	0.8	[M6]
Region 2	67	4	2600	3100	0.8	[M6]
Japan						
Country average	19	0.18	34	43	0.8	[N4]
Netherlands						
Country average	20	1.8	250	480	0.5	[C26]
Norway						
Oslo	38	1.0	1400	550	2.5	[B11]
Oppland	151	27.8	3100	15000	0.2	[B11]
N. Tr.	78 b/	18.7	21000	10000	2.1	[B11]
Finmark	45 c/	0.4	5600	210	27	[B11]
Poland						
Country average	535	5.2	1700	3100	0.6	[C2]
Sweden						
Region 1	50	31	1900	3300	0.6	[F6]
Country average	218	6.8	820	1200	0.7	[F6]
Switzerland						
Mitteland		2.0	750	1500	0.5	[P2]
Turkey						
Country average	30	4	1700	1900	0.9	[T2]
United Kingdom						
Region 1	30	0.1	190	120	1.6	[F12]
Region 3	300	3	710	2500	0.3	[F12]

a/ Measured composited diet samples give relative results of 0.8 and 0.7 for regions 2 and 3, respectively [S21].
b/ Southern Lapps.
c/ Northern Lapps.

Table 17

First-year dose equivalents
(μSv)

Country	Thyroid dose equivalent		Effective dose equivalent	
	Infants	Adults	Rural	Urban
NORTH EUROPE				
Denmark	160	64	33	28
Finland	1800	1200	490	440
Norway	1000	570	240	220
Sweden				
Region 1	1800	700	440	340
Region 2	47	92	87	83
Region 3	870	280	110	99
CENTRAL EUROPE				
Austria	9400	1800	710	630
Czechoslovakia				
Region 1	2000	2300	280	270
Region 2	2200	2600	370	350
Region 3	2100	3200	340	340
German Dem. Rep.				
Region 1	12000	2000	270	250
Region 2	7700	1300	360	320
Region 3	3100	690	180	160
Germany, Fed. Rep. of				
Region 1	660	200	70	63
Region 2	2300	530	140	120
Region 3	6200	1500	510	460
Hungary				
Region 1	7500	1300	290	270
Region 2	4500	770	180	170
Poland	8100	1400	280	260
Romania				
Region 1	8200	1200	270	250
Region 2	33000	5300	1100	1000
Region 3	17000	2700	550	520
Switzerland				
Region 1	27000	4600	1300	1200
Region 2	20000	3000	320	310
Region 3	17000	2300	210	200
Region 4	5800	1100	120	120
WEST EUROPE				
Belgium	2300	460	42	39
France				
Region 1	450	90	6.7	6.1
Region 2	1100	240	40	37
Region 3	3400	810	160	150
Ireland	2500	540	130	120
Luxembourg	2700	580	100	93
Netherlands	940	390	61	54
United Kingdom				
Region 1	600	97	12	12
Region 2	1300	260	110	100
Region 3	1700	400	200	190
SOUTH EUROPE				
Bulgaria				
Region 1	25000	2800	720	700
Region 2	25000	2900	810	770
Greece				
Region 1	30000	7600	960	930
Region 2	12000	3000	330	320
Italy				
Region 1	4400	2300	380	360
Region 2	2700	970	240	230
Portugal	9	4	1.9	1.8
Spain				
Region 1	520	100	12	12
Region 2	9	5	2.2	2.1
Yugoslavia				
Region 1	22000	8500	660	590
Region 2	10000	4000	290	260
Region 3	3600	1500	110	99

Table 17. continued

Country	Thyroid dose equivalent		Effective dose equivalent	
	Infants	Adults	Rural	Urban
USSR				
Region 1	21000	6900	2000	1900
Region 2	24000	6300	930	880
Region 3	3800	1400	460	420
Region 4	3600	910	140	130
Region 5	82	25	4.3	3.9
WEST ASIA				
Cyprus	4700	1200	67	66
Israel	1500	1100	94	92
Syrian Arab Rep.	1400	74	7.7	7.3
Turkey	2300	480	200	180
EAST ASIA				
China	390	47	7.9	7.4
India	69	5	2.1	2.0
Japan	210	100	7.9	7.2
NORTH AMERICA				
Canada	75	11	1.4	1.3
United States	110	15	1.5	1.4

Table 18

Country average of first-year dose equivalents

Country	Thyroid dose equivalent		Effective dose equivalent	Ratio to result reported from country [N5]		
				Thyroid dose		Effective dose
	Infant	Adult		Infant	Adult	
	(μSv)		(μSv)			
EUROPE						
Bulgaria	25000	2900	760			
Austria	9400	1800	670	1.2	1.0	1.0
Greece	20000	5000	590	3.6	2.6	1.6
Romania	18000	2800	570			
Finland	1800	1200	460	1.0	1.7	0.9
Yugoslavia	14000	5500	390			
Czechoslovakia	2200	2700	350			
Italy	3400	1500	300	0.5	0.5	0.6
Poland	8100	1400	270			
Switzerland	15000	2300	270	9.3	2.1	1.2
Hungary	6000	1000	230			
Norway	1000	570	230	0.8	1.5	1.4
German Dem. Rep.	5100	970	210			
Sweden	1000	340	150	2.0	0.9	0.7
Germany, Fed. Rep. of	1700	440	130	0.6	0.5	0.4
Ireland	2500	540	120	0.2	2.3	1.1
Luxembourg	2700	580	98	3.5	1.7	0.8
France	1600	360	63	1.8	4.1	2.6
Netherlands	940	390	58	0.6	1.3	0.8
Belgium	2300	460	41	1.7	2.2	1.0
Denmark	160	64	30	0.6	1.3	1.1
United Kindom	710	130	27	0.3	0.8	0.7
Spain	110	24	4.2			
Portugal	9	4	1.8	0.1	0.4	0.3
USSR	5000	1400	260			
ASIA						
Turkey	2300	480	190	0.7	1.2	2.2
Israel	1500	1100	92			
Cyprus	4700	1200	68			
Syrian Arab Rep.	1400	74	8.3			
China	390	47	7.8			
Japan	210	100	7.6	1.4	2.1	1.2
India	69	5	2.1			
NORTH AMERICA						
Canada	75	11	1.4	4.2	6.5	0.6
United States	110	15	1.5			

Table 19

Transfer factor from deposition to thyroid dose for iodine-131

Country	Iodine-131 deposition density (kBq/m^2)	Thyroid dose equivalent in first year from iodine-131 (μSv)		Transfer factor (P_{25}) deposition to thyroid dose for iodine-131 (μSv per kBq/m^2)	
		Infants	Adults	Infants	Adults
NORTH EUROPE					
Denmark	6.1	130	33	21	5.4
Finland	100	1500	690	15	6.7
Norway	85	930	350	11	4.1
Sweden					
Region 1	160	1500	280	9.4	1.8
Region 2	13	17	13	1.3	1.0
Region 3	41	820	190	20	4.6
CENTRAL EUROPE					
Austria	120	9000	1100	78	9.5
Czechoslovakia					
Region 1	26	1900	2100	74	82
Region 2	58	2000	2200	34	38
Region 3	30	1900	2900	63	96
German Dem.Rep.					
Region 1	45	12000	1800	260	40
Region 2	42	7500	970	180	23
Region 3	19	3000	520	160	28
Germany, Fed. Rep. of					
Region 1	12	610	130	50	11
Region 2	27	2200	380	81	14
Region 3	100	5900	1000	57	9.7
Hungary					
Region 1	30	7400	1000	250	33
Region 2	9.3	4400	610	470	66
Poland	38	8000	1100	210	29
Romania					
Region 1	24	8100	980	340	42
Region 2	94	33000	4400	350	47
Region 3	47	17000	2200	360	47
Switzerland					
Region 1	30	27000	3600	910	120
Region 2	25	20000	2800	790	110
Region 3	15	17000	2200	1200	150
Region 4	9.4	5800	1000	620	110
WEST EUROPE					
Belgium	5.2	2200	420	420	81
France					
Region 1	0.9	450	85	500	94
Region 2	5.3	1100	210	210	40
Region 3	24	3300	670	140	28
Ireland	10	2400	430	230	41
Luxembourg	19	2600	480	140	25
Netherlands	11	910	340	80	30
United Kingdom					
Region 1	0.8	590	84	740	110
Region 2	2.0	1200	160	600	80
Region 3	6.0	1600	220	270	37
SOUTH EUROPE					
Bulgaria					
Region 1	4.2	24000	2200	5700	520
Region 2	13	24000	2200	1900	170
Greece					
Region 1	36	30000	6900	830	190
Region 2	14	12000	2700	860	190
Italy					
Region 1	25	4400	1900	180	76
Region 2	15	2700	750	180	50
Portugal	0.07	8.0	2.3	110	33
Spain					
Region 1	0.4	510	96	1280	240
Region 2	0.07	8.0	2.8	110	40
Yugoslavia					
Region 1	140	22000	8100	160	60
Region 2	60	10000	3800	170	63
Region 3	24	3500	1400	150	58

Table 19, continued

Country	Iodine-131 deposition density (kBq/m^2)	Thyroid dose equivalent in first year from iodine-131 (μSv)		Transfer factor (P_{25}) deposition to thyroid dose for iodine-131 (μSv per kBq/m^2)	
		Infants	Adults	Infants	Adults
USSR					
Region 1	590	20000	5100	34	8.6
Region 2	480	23000	5500	48	11
Region 3	160	3500	980	22	6.0
Region 4	20	3600	790	180	40
Region 5	0.4	80	22	200	55
WEST ASIA					
Cyprus	2.0	4700	1200	2400	600
Israel	0.7	1500	1000	2100	1400
Syrian Arab Rep.	-	1400	69		
Turkey	-	2200	320		
EAST ASIA					
China	0.3	390	40	1300	130
India	0.04	68	3.2	1500	73
Japan	1.6	210	96	130	60
NORTH AMERICA					
Canada	0.1	75	9.4	750	94
United States	0.15	110	14	740	94

Table 20

Transfer factor from deposition to first-year effective dose equivalent from ingestion of caesium-137

Country	Deposition density (kBq/m^2)	First-year diet		Body burden integrated concentration (Bq a/kg)	Effective dose equivalent (μSv)	Transfer factors		
		Integrated concentration (Bq a/kg)	Intake (Bq)			b_1 a/	P_{34} b/	$P_{25,1}$ c/
NORTH								
Denmark	1.3	1.35	660	3.7	9.2	1.0	2.7	7.2
Finland	14.7	20.8	12100	68	170	1.4	3.3	12
Norway	5.3	14.1	7030	39	98	2.7	2.8	19
Sweden								
Region 1	31	17.3	8860	50	120	0.6	2.9	4.0
Region 2	0.81	(6.3)	(3210)	(18)	(45)	(7.7)	2.9	(55)
Region 3	2.3	6.3	3210	18	45	2.7	2.9	20
TEMPERATE								
Austria	23	34.5	17800	100	250	1.5	2.9	11
Belgium	0.84	1.3	650	3.6	9.1	1.6	2.7	11
Bulgaria								
Region 1	3.9	50.8	23500	130	330	13	2.6	84
Region 2	12	50.8	23500	130	330	4.2	2.6	27
Canada	0.03	(0.05)	(35)	(0.2)	(0.5)	(1.6)	4.1	(16)
Czechoslovakia								
Region 1	2.3	13.8	6660	37	93	5.9	2.7	40
Region 2	5.3	16.1	7780	44	110	3.1	2.7	21
Region 3	2.8	15.2	7380	41	100	5.5	2.7	38
France								
Region 1	0.18	0.16	80	0.45	1.1	0.9	2.8	6.2
Region 2	0.66	2.1	1040	5.8	15	3.1	2.8	22
Region 3	3.2	8.0	4050	23	57	2.5	2.8	18
German Dem.Rep.								
Region 1,3	6.1	6.0	3660	20	51	1.0	3.4	8.4
Region 2	11	12.1	7470	42	100	1.1	3.4	9.7

Table 20, continued

Country	Deposition density (kBq/m²)	First-year diet: Integrated concentration (Bq a/kg)	First-year diet: Intake (Bq)	Body burden integrated concentration (Bq a/kg)	Effective dose equivalent (μSv)	Transfer factors: b_1 a/	Transfer factors: P_{34} b/	Transfer factors: $P_{25,1}$ c/
Germany, Fed. Rep. of								
Region 1	2	4.5	1690	9.4	24	2.2	2.1	12
Region 2	4	8.2	3100	17	43	2.0	2.1	11
Region 3	16	31.9	12000	67	170	2.0	2.1	11
Hungary								
Region 1	4.8	13.0	7300	41	100	2.7	3.1	21
Region 2	1.5	8.9	5000	28	70	6.0	3.1	47
Ireland	3.4	8.4	3280	18	46	2.5	2.2	14
Italy								
Region 1	6	23.2	10700	60	150	3.9	2.6	25
Region 2	4	16.9	7770	43	110	4.2	2.6	27
Luxembourg	2.7	4.6	2210	12	31	1.7	2.7	11
Netherlands	1.8	2.4	1170	6.6	16	1.4	2.7	9.1
Poland	5.2	14.6	8160	46	110	2.8	3.1	22
Romania								
Region 1	4.5	(10.3)	(7250)	(41)	(100)	(2.3)	4.0	(23)
Region 2	18	(41.0)	(29000)	(160)	(410)	(2.3)	4.0	(23)
Region 3	9	20.5	14500	81	200	2.3	4.0	23
Switzerland								
Region 1	14.8	59.1	37900	210	530	4.0	3.6	36
Region 2	3.5	10.0	6430	36	90	2.9	3.6	26
Region 3	2.0	6.1	3890	22	54	3.0	3.6	27
Region 4	1.3	3.7	2390	13	33	2.9	3.6	26
United Kingdom								
Region 1	0.1	0.69	300	1.7	4.3	6.9	2.5	43
Region 2	1.7	7.9	3500	20	49	4.7	2.5	29
Region 3	3.0	14.2	6300	35	88	4.7	2.5	29
USSR								
Region 1	38.8	73.7	50400	280	700	1.9	3.8	18
Region 2	14.5	21.2	14500	81	200	1.5	3.8	14
Region 3	10.0	18.3	12500	70	170	1.8	3.8	18
Region 4	2.8	4.2	2890	16	40	1.5	3.8	15
Region 5	0.094	(0.15)	(100)	(0.6)	(1.4)	(1.6)	3.8	(15)
Yugoslavia								
Region 1	23	23.5	12400	70	170	1.0	3.0	7.6
Region 2	10	9.8	5200	29	73	1.0	3.0	7.3
Region 3	4	3.4	1780	10	25	0.8	3.0	6.2
SOUTH								
China	0.15	(0.42)	(200)	(1.1)	(2.8)	(2.8)	2.6	(18)
Cyprus	0.6	(1.6)	(1050)	(5.9)	(15)	(2.7)	3.7	(25)
Greece								
Region 1	8	55.0	28600	160	400	6.9	2.9	50
Region 2	2.4	17.3	8990	50	130	7.2	2.9	52
India	0.035	(0.25)	(86)	(0.5)	(1.2)	(7.1)	1.9	(34)
Israel	0.4	(3.6)	(2300)	(13)	(32)	(9.0)	3.6	(80)
Japan	0.18	0.20	120	0.6	1.6	1.1	3.2	9.0
Portugal	0.02	0.15	65	0.4	0.9	7.5	2.4	45
Spain								
Region 1	0.07	0.70	360	2.0	5.0	10	2.9	71
Region 2	0.02	(0.15)	(77)	(0.4)	(1.1)	(7.5)	2.9	(54)
Syrian Arab Rep.	0.13	(0.22)	(140)	(0.8)	(2.0)	(1.7)	3.6	(15)
Turkey	4	7.9	4880	27	68	2.0	3.4	17
United States	0.026	(0.05)	(36)	(0.2)	(0.5)	(2.0)	3.9	(20)

a/ Deposition to first-year total diet; units: Bq a/kg per kBq/m².
b/ Diet to body; units: Bq a/kg per Bq a/kg.
c/ Deposition to first-year committed effective dose equivalent; units: μSv per kBq/m².

Table 21

Transfer factor from deposition to effective dose equivalent from ingestion of caesium-137 after the first year

Region a/	Food consumption (kg/a)						Transfer factors			
	Milk prod.	Grain prod.	Leafy veg.	Veg./ fruit	Meat	Total	$P_{23,2+}$ b/ Grain	$P_{23,2+}$ b/ Total diet	P_{34} c/	$P_{25,2+}$ d/
North Europe	220	75	25	140	65	525	0.9	2.6	2.9	19
Central Europe	140	120	30	150	70	510	1.9	2.8	2.8	20
West Europe	150	75	60	120	70	475	2.0	2.8	2.6	19
Southeast Europe	105	125	45	150	60	485	2.3	3.0	2.7	20
Southwest Europe	90	95	120	125	60	490	10	4.3	2.8	30
USSR	330	130	35	120	65	680	0.6	2.3	3.8	22
West Asia	115	190	95	180	40	620	1.3	2.3	3.5	20
East Asia	20	210	30	140	25	425	6.0	4.4	2.4	26
North America	175	90	25	265	145	700	4.8	3.9	3.9	38

a/ North Europe: Denmark, Finland, Norway, Sweden.
Central Europe: Austria, Czechoslovakia, German Democratic Republic, Federal Republic of Germany, Hungary, Poland, Romania, Switzerland.
West Europe: Belgium, France, Ireland, Luxembourg, Netherlands, United Kingdom.
Southeast Europe: Bulgaria, Greece, Italy, Yugoslavia.
Southwest Europe: Portugal, Spain.
West Asia: Cyprus, Israel, Syrian Arab Rep., Turkey.
East Asia: China, India, Japan.
North America: Canada, United States.

b/ Deposition to total diet after first year; units: Bq a/kg per kBq/m^2.

c/ Diet to body; units: Bq a/kg per Bq a/kg.

d/ Deposition to effective dose equivalent commitment after first year; units: μSv per kBq/m^2.

Table 22

Regional transfer factors applicable after first year and components of the effective dose equivalent commitment

Region a/	Population-weighted caesium-137 deposition density (kBq/m^2)	Transfer factor related to caesium-137 deposition				Effective dose equivalent commitment (μSv)		
		$P_{25,2+}$ External gamma	$P_{25,2+}$ ingestion Cs-137	$P_{25,2+}$ ingestion Cs-134	$P_{25,2+}$ Total	First year	After first year	Total
North Europe	7.0	76	20	12	110	210	760	970
Central Europe	6.1	76	20	12	110	270	670	940
West Europe	1.0	76	20	12	110	48	110	160
Southeast Europe	7.4	76	20	12	110	390	810	1200
Southwest Europe	0.03	76	30	18	120	3.7	3.4	7
USSR	5.1	76	20	12	110	260	560	820
West Asia	3.2	76	20	12	110	160	350	510
East Asia	0.1	76	30	18	120	5.6	13	19
North America	0.03	76	30	18	120	1.5	3.2	5

a/ North Europe: Denmark, Finland, Norway, Sweden.
Central Europe: Austria, Czechoslovakia, German Democratic Republic, Federal Republic of Germany, Hungary, Poland, Romania, Switzerland.
West Europe: Belgium, France, Ireland, Luxembourg, Netherlands, United Kingdom.
Southeast Europe: Bulgaria, Greece, Italy, Yugoslavia.
Southwest Europe: Portugal, Spain.
West Asia: Cyprus, Israel, Syrian Arab Rep., Turkey.
East Asia: China, India, Japan.
North America: Canada, United States.

Table 23

Total transfer factor for effective dose equivalent based on caesium-137 deposition
(μSv per kBq/m^2)

Pathway/ radionuclide	North			Temperate			South		
	First year	After first year	Total	First year	After first year	Total	First year	After first year	Total
External gamma									
Cs-137	2.2	71	73	2.2	71	73	2.2	71	73
Cs-134	2.5	4.9	7	2.5	4.9	7	2.5	4.9	7
Other	5.6	0.2	6	5.6	0.2	6	5.6	0.2	6
Subtotal	10	76	86	10	76	86	10	76	86
Ingestion									
Cs-137	15	20	35	20	20	40	25	25	50
Cs-134	11	12	23	14	12	26	18	15	33
I-131	1	-	1	10	-	10	20	-	20
Subtotal	27	32	59	44	32	76	63	40	103
Total (rounded)	40	110	150	50	110	160	70	120	190

Table 24

Total caesium-137 deposit and dose commitments in the northern hemisphere

Region	Area	Population	Distance from Chernobyl	Caesium-137 deposition (kBq/m^2) weighted by		Cs-137 deposit	Effective dose equivalent commitment			
							Per caput (μSv)		Collective (man Sv)	
	(10^3 km^2)	(10^6)	(km)	Area	Population	(PBq)	First year	Total	First year	Total
EUROPE										
North a/	1249	22.8	1300	8.2	7.0	10.2	210	970	4700	22000
Central b/	1253	178.0	1200	7.0	6.0	8.8	280	930	49000	166000
West c/	936	137.7	2000	1.3	1.0	1.2	48	150	6600	21000
Southeast d/	829	101.6	1500	8.2	7.2	6.8	380	1200	39000	121000
Southwest e/	596	47.2	2900	0.03	0.03	0.02	4	7	180	340
USSR	22190	279.1	-	1.4	5.0	30.9	260	810	72000	226000
ASIA										
Southwest f/	4611	114.9	2200	1.0	1.0	4.6	70	190	8000	22000
South g/	6786	1082	5400	0.08	0.08	0.5	6	15	6100	16000
Southeast h	2575	240.6	7800	0.03	0.03	0.08	2	6	510	1400
East i/	11720	1268	6600	0.04	0.04	0.5	3	8	3600	9600
AMERICA										
North j/	20560	347.0	9000	0.02	0.02	0.4	1	4	490	1300
Caribbean k/	216	30.1	9200	0.018	0.018	0.004	1	3	40	100
Central l/	517	26.9	10700	0.012	0.012	0.006	0.7	2	20	60
South m/	2520	49.7	10100	0.013	0.013	0.03	1	2	50	120
AFRICA										
North n/	8438	128.4	3000	0.4	0.4	3.4	28	76	3600	9800
West o/	6118	172.3	5600	0.08	0.08	0.5	6	15	970	2600
Central p/	2415	18.3	5300	0.08	0.08	0.2	5	15	100	280
East q/	2117	59.5	5100	0.09	0.09	0.2	6	17	380	1000
Greenland	2176	0.06	4000	0.18	0.18	0.4	7	30	0.4	2
North Atlantic Ocean	53000	-	5700	0.07	-	3.7				
North Pacific Ocean	102000	-	10900	0.01	-	1.0				
Northern hemisphere Total (rounded)	252800	4304	5700	0.3	0.9	70	45	140	200000	600000

a/ Denmark, Finland, Iceland, Norway, Sweden.
b/ Austria, Czechoslovakia, German Dem.Rep., Germany, Fed.Rep., Hungary, Poland, Romania, Switzerland.
c/ Belgium, France, Ireland, Luxembourg, Netherlands, United Kingdom.
d/ Albania, Bulgaria, Greece, Italy, Malta, Yugoslavia.
e/ Portugal, Spain.
f/ Bahrain, Cyprus, Dem. Yemen, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Qatar, Saudi Arabia, Syrian Arab Rep., Turkey, United Arab Emirates, Yemen.
g/ Afghanistan, Bangladesh, Bhutan, India, Iran, Nepal, Pakistan, Sri Lanka.
h/ Burma, Dem. Kampuchea, Laos Dem. Rep., Malaysia, Philippines, Singapore, Thailand, Viet Nam.
i/ China, Dem. Korea, Hong Kong, Japan, Korea, Rep., Mongolia.
j/ Canada, United States, Mexico.
k/ Cuba, Dominican Rep., Haiti, Jamaica, Puerto Rico, Trinidad/Tobago.
l/ Costa Rica, El Salvador, Guatemala, Honduras, Nicaragua, Panama.
m/ Colombia, Guyana, Suriname, Venezuela, French Guiana.
n/ Algeria, Egypt, Libya, Morocco, Sudan, Tunisia.
o/ Benin, Burkina Faso, Cape Verde, Cote d'Ivoire, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Mali, Mauritania, Niger, Nigeria, Senegal, Sierra Leone, Togo.
p/ Cameroon, Central Afr.Rep., Chad, Equatorial Guinea.
q/ Ethiopia, Somalia, Uganda, Djibouti.

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back to first page

ANNEX E

Genetic hazards

CONTENTS

Introduction

1. The evaluation of genetic hazards associated with the exposure of human populations to ionizing radiation is one of the major areas in which the Committee has been active since its inception. Its first comprehensive Report on this topic was published in 1958 [U1], and this was followed by six Reports of a similar nature published in 1962 [U2], 1966 [U3], 1972 [U4], 1977 [U5], 1982 [U6] and 1986 [U7]. This time span of over a quarter of a century has witnessed a number of major advances in radiation genetics, human genetics, cytogenetics and epidemiology; it also represents a period during which, from the standpoint of genetic risk evaluation, there have been changes in concepts and methods and shifts in emphasis necessitating revision of views and quantitative estimates of genetic risks. The paucity of direct data that bear on the induction by radiation of genetic effects leading to disease states in man continues to remain a major drawback; however, the prodigious amount of literature on such effects in other species makes it prudent and reasonable to believe that exposure of human germ cells to ionizing radiation will cause mutations and chromosomal aberrations, which in turn may lead to diseases. So far, there has been no alternative but to use data generated from experimental organisms as the main basis for predicting expected effects in man; in fact, the estimation of genetic risks to man from exposure to ionizing radiation remains a major exercise in extrapolation.

2. The aim of this document is threefold: (a) to provide a general background of the principles and methods that are used in the evaluation of genetic radiation hazards in man; (b) to trace the evolution of the conceptual framework, the data-base, the assumptions and the extrapolations involved in genetic risk estimation, from about the mid-1950s to the present; and (c) to indicate in which areas research is in progress or needed in the years to come. While the emphasis will be on the Committee's continuing efforts in this direction, the work of other scientific bodies will also be discussed where appropriate.

A. GERM CELL STAGES AND RADIATION CONDITIONS RELEVANT TO GENETIC RISK EVALUATION

3. From the standpoint of hazard evaluations, it is the effects of radiation on two particular germ cell stages that are considered important: (a) the mitotically dividing stem-cell spermatogonia in the male, which constitute a permanent germ cell population in the testes and which continue to multiply throughout the reproductive life span of the individual, and (b) the oocytes, primarily the immature ones, in the female. Female mammals are born with a finite number of oocytes, formed during embryonic development. These primordial oocytes, as they are called, grow and become surrounded by follicles but are arrested at a particular stage (diplotene or dictyate) in meiosis. This arrest lasts from late pre-natal life until shortly before ovulation in the mature female. Because oocytes are not replenished by mitosis during adult life and because they only have to complete the meiotic divisions before pronuclear fusion, these are clearly the cell stages in the female whose irradiation has great potential significance for hazard evaluations.

4. The radiation exposures received by human populations are usually delivered as small doses at high-dose rates (e.g., exposure during diagnostic radiology) or they are greatly protracted (e.g., continuous exposure to natural and man-made sources). In therapeutic radiology, doses as high as several Gy may be delivered, and at high dose rates; however, such exposures are warranted on medical grounds and are given only to selected individuals for the treatment of specific cancers. In estimating genetic hazards to the population, the relevant radiation conditions are, therefore, low doses and low-dose rates.

B. GENERAL ASSUMPTIONS

5. In using the data from mouse studies (the principal model in this context) or studies of other suitable mammals (such as non-human primates) to make quantitative estimates of genetic risks in man, three important assumptions are made unless there is evidence to the contrary: (a) the amount of genetic damage induced by a given type of radiation under a given set of conditions is the same in human germ cells and in those of the test species used as the model; (b) the biological factors (e.g., sex, germ cell stage and age) and physical factors (e.g., quality of radiation and dose rate) affect the magnitude of the damage in similar ways and to similar extents in the experimental species from which extrapolations are made and in humans; and (c) at low doses and at low-dose rates of low-LET irradiation, there is a linear relationship between dose and the frequency of genetic effects. Other more specific assumptions and considerations will be discussed in the appropriate sections of this review.

C. METHODS

6. The methods that have been used so far in quantitative genetic risk assessments can be broadly grouped under two headings: the doubling dose method (or the relative mutation risk method) and the direct methods. The aim of the doubling dose method is to provide an estimate of risks in terms of the additional number of cases of genetic disease due to radiation exposure using the natural prevalence of such diseases in the population as a frame of reference.

7. The doubling dose is the amount of radiation necessary to produce as many mutations as those that occur spontaneously in a generation; it is obtained by dividing the spontaneous rate by the rate of induction per unit dose. Thus, for instance, if the average spontaneous rate is m_1 per locus and the average rate of induction is m_2 per locus per unit dose of radiation, then the doubling dose $c = m_1/m_2$. The reciprocal of the doubling dose, $1/c$, is the relative mutation risk per unit dose. It is easy to see that the lower the

doubling dose, the higher the relative mutation risk and vice versa.

8. The doubling dose method is generally used to estimate risks to a population under continuous irradiation. The general concept is that, under normal conditions in the absence of radiation, there is an equilibrium between those mutations that arise spontaneously and those that are eliminated by selection in every generation. Under conditions of continuous irradiation (with the influx of new mutations that it entails), the population will eventually reach a new equilibrium between those mutations that enter the gene pool and those that are eliminated.

9. In practice, what is done is to: (a) estimate the doubling dose(s) from experimental data on spontaneous and induced mutations and chromosomal aberrations and (b) estimate the expected increase at equilibrium as a product of prevalence, p, of spontaneously arising diseases, relative mutation risk, 1/c, and the dose sustained by the population. The increase in the first generation is then estimated from that at equilibrium, using certain assumptions. It bears mentioning that the risk at equilibrium under conditions of continuous irradiation (say, at a rate of x mGy per generation) is numerically equal to the integrated risk over all future generations following a single (i.e., one time only) dose of x mGy to the parental generation.

10. In principle, the doubling dose method can be used to estimate risks from the induction of mutational events, irrespective of whether they are operationally classified as dominants or recessives, as well as chromosomal aberrations. The major application of the above method, however, is to simple, dominantly inherited traits whose equilibrium frequencies (i.e., those of the responsible mutant genes) can be assumed to be directly proportional to the mutation rate [B1, D1]. The assumption is almost as good for sex-linked traits [B1].

11. An increase in mutation rate of autosomal recessive genes will not lead to a corresponding increase in the frequency of recessive diseases for two reasons: (a) when recessive mutations first arise (or are induced), they are present in heterozygous condition and their fate depends strongly on the way selection acts [B1] and (b) a recessive mutation has to combine with an already existing recessive allele or become homozygous to manifest the disease, and this may take from many to hundreds of generations, depending on a number of factors.

12. Evidence for the radiation induction of numerical chromosomal anomalies resulting in live births, either in experimental mammals or in man, is insufficient and equivocal, apart from XO induction in mice. Consequently, the use of the doubling dose method to estimate risks for chromosomal diseases is subject to considerable uncertainty, although it was used by the Committee in the UNSCEAR 1977 Report [U5]. However, there is definite evidence for the induction of structural chromosomal anomalies, particularly reciprocal translocations (but not Robertsonian translocations) in mammalian and human germ cells. With certain assumptions, the doubling dose method can therefore be used to estimate risks from the induction of at least certain kinds of structural chromosomal anomalies.

13. The estimation of risks associated with the induction of congenital anomalies and other diseases of complex aetiology (whose spontaneous prevalences are much higher than those of Mendelian and chromosomal diseases) poses a different problem. For a number of well-studied conditions belonging to this group, the evidence is consistent with the assumption that their aetiology is multifactorial, depending on polygenic genetic predisposition and environmental factors that may also be multiple. In order to be able to estimate risks of induction for this group of diseases, the BEIR Committee [B1] introduced the concept of "mutation component" in its 1972 Report (see also [C1] and [C2]). In that Report [B1], the mutation component of a disease was defined as "the proportion of its incidence that is directly proportional to the mutation rate". For Mendelian diseases and chromosomal anomalies, the mutation component is 1, except if there is some selective advantage in the heterozygote; for the diseases of complex aetiology mentioned above, it was assumed that this component is less than 1 (in the range 0.05-0.5). Therefore, for the estimation of risks of inducting this group of diseases, the principle is the same as that for autosomal dominant and X-linked ones, except that the product of p, 1/c and x (see paragraphs 7-9) is to be further multiplied by the mutation component.

14. In addition to the doubling dose method discussed above, a number of other methods, called direct methods, have been used over the years for the estimation of genetic risks; these are discussed later. The advantage of these methods is that they express absolute risks in terms of effects expected in the progeny for the different kinds of genetic damage on the basis of experimental data. However, it has not always been possible to bridge satisfactorily the gap between the estimates of rates of induction and the actual effects expected in terms of genetic disease.

I. BRIEF HISTORY OF THE APPROACHES TO GENETIC RISK ASSESSMENTS

A. THE DOUBLING DOSE METHOD

1. Reports by different scientific bodies, including UNSCEAR, in the period 1956-1966

15. The general radiation genetic principles that guided the BEAR Committee [B2], the British Medical Research Council Committee [M1] and UNSCEAR [U1] in the preparation of their respective reports in the mid- and late 1950s were those that emerged from the extensive work with Drosophila (primarily mature sperm irradiation), ongoing work with experimental mammals (primarily with the mouse) and the few sparse human data. Of these principles, the following deserve mention: (a) mutations, spontaneous or induced, are

usually harmful; (b) any dose of radiation, however small, entails some genetic risk; (c) the number of mutations produced is proportional to the dose, so that linear extrapolation from high-dose data provides a valid estimate of the low-dose effects; and (d) the effect is independent of the dose rate at which the radiation is delivered and of the spacing between the exposures.

16. In its 1956 Report, the BEAR Committee [B2] estimated that the doubling dose was probably between 30 R and 80 R and that it would be reasonable to use 40 R in computations. It also assumed that about 2% of all live-born children are, or will be, seriously affected by defects of "simple genetic origin". Under the further assumption that for this fraction of human genetic defects the incidence is proportional to the mutation rate, the effect at equilibrium after a continuing exposure to the then-recommended limit of 10 R per generation was computed. The conclusion was that there would be about 5,000 new instances of "tangible inherited defects" per million births, with about one tenth of this number in the first generation after the beginning of radiation exposure. The general philosophy, the range and the best estimates of the doubling dose arrived at by the British Medical Research Council Committee [M1] and by UNSCEAR [U1] were roughly similar.

17. With increasing reliance on mouse data, attention was focused on collecting more extensive information after irradiation of spermatogonia and oocytes, the cell stages most at risk from the standpoint of genetic risks. The results that became available (after the publication of the three reports mentioned earlier) demonstrated that (a) chronic gamma-irradiation of spermatogonia was mutationally less effective (by a factor of about 3) than the same total dose of high-dose-rate x-irradiation [R1, R3]; (b) following acute x-irradiation at high doses, the mutation rate in mature and maturing oocytes was higher than in spermatogonia; and (c) the dose-rate effect in females was even more pronounced than in males [R4].

18. On the basis of these results, the UNSCEAR 1962 Report [U2] suggested that for chronic low-LET irradiation of males, the doubling dose was probably 3-4 times the value of 30 R used in the UNSCEAR 1958 Report [U1] and also noted the possibility that, for similar radiation conditions, the doubling dose for females could be higher than for males. It was pointed out that ". . . a permanent doubling of the mutation rate would ultimately double the prevalence of those serious defects determined by the unconditionally harmful genes which are estimated to affect about 1% of those born alive".

19. By the time of the UNSCEAR 1966 Report [U3], the earlier mouse results had been amply confirmed and extended and new data had been obtained in females showing that there was a dramatic effect of the interval between irradiation and conception: in the first seven weeks after irradiation, the mutation frequency was high; subsequently, however, no mutations at all were recovered [R5]. In view of the wealth of mouse data that by then had accumulated, the Committee abandoned the doubling dose approach in favour of a more direct approach, using primarily the mouse specific-locus data as a basis. This aspect will be discussed later.

2. The UNSCEAR 1972 and BEIR 1972 Reports

20. In the UNSCEAR 1972 Report, the Committee revived its interest in the doubling dose method but gave it a low profile. Part of the reason for this renewed interest was that Lüning and Searle [L1] had summarized a number of estimates of doubling doses for different kinds of genetic damage in the mouse (semi-sterility, specific locus mutations, dominant visibles, mutations affecting the skeleton and autosomal recessive lethals). They all fell within a range of 16-51 R, averaging about 30 R for spermatogonia exposed to high acute x-ray doses. On the basis of the dose-rate studies on the induction of specific locus mutations in male mice, it was inferred that the doubling dose for chronic low-LET irradiation conditions could be about three times the above value, i.e., 100 R. Lüning and Searle [L1] gave no doubling dose estimates for females, since very little information on spontaneous rates was available in the literature.

21. On the assumption, based on the Northern Ireland survey [S1], that in man about 3% of live born are affected by deleterious traits maintained by mutation (now including simple dominants, some traits of uncertain genetic origin and chromosomal anomalies) and that the doubling dose is 100 R, UNSCEAR [U4] estimated that there would be about 300 extra cases per million live births for each rad of low-dose-rate, low-LET radiation to the males of the parental generation. It argued that ". . . the great majority of these will be (the result of) gene mutations with an unknown degree of dominance . . . if, however, the range observed in Drosophila (2-5%) is used as an upper limit to the average dominance in man as expressed by the frequency of deleterious traits among liveborn, then 6-15 affected individuals per million liveborn would be expected in the first generation following irradiation, the rest of the damage being expressed in subsequent generations".

22. In 1972, the BEIR Committee published its Report [B1]. In that Report, the doubling dose was calculated using an assumed range for the rate of spontaneous mutations in man ($0.5\ 10^{-6}$ to $0.5\ 10^{-5}$ per gene) and an induction rate from mouse specific-locus data ($0.25\ 10^{-7}$ per locus per rem, an average rate taking into account both sexes and assumed to apply to low-dose-rate, low-LET irradiation conditions). The range 20-200 rem thus derived represents a "hybrid" doubling dose range. On the basis of the Northern Ireland survey [S1] (as may be recalled, this was also the basis for UNSCEAR's figures), the BEIR Committee assumed that the prevalence of genetic diseases in the human population is about 6% (including 1% dominant and X-linked diseases, 1% chromosomal and recessive diseases and 4% congenital anomalies and other diseases of complex aetiology), and they estimated the effects of 5 rem per generation on a population of 1 million live births.

23. Further considerations or assumptions used in that exercise were the following: (a) since the incidence of dominant and X-linked traits is essentially proportional to the mutation rate, their frequency will be increased by the relative mutation risk per rem multiplied by the dose; (b) the incidence of recessive diseases is only very indirectly related to the mutation rate; (c) diseases caused by chromosomal anomalies are not likely to be very much increased by low-level irradiation; (d) for diseases of complex aetiology, the mutation component is likely to be in the range of 5-50% (see paragraph 13); (e) for dominant and X-linked diseases, the expected increase in the first generation is likely to be about 20% of that at equilibrium (based on the finding, in the Northern Ireland survey, that the population incidence was only four fifths of the incidence in new-borns, and this is roughly equivalent to assuming that the average mutant persists in the population for five generations); (f) for diseases of complex aetiology, the first-generation incidence will be about one tenth of that at equilibrium.

24. The estimated effect of 5 rem per generation on a population of 1 million live births was 300-7,500 new cases of genetic disease at equilibrium and 60-1,000 cases in the first generation, relative to the assumed prevalence of 60,000 cases of spontaneous origin per million live births. To facilitate comparisons with the other estimates, the recalculated figures for 1 rem per generation are summarized in Table 1.

3. The UNSCEAR 1977 Report

25. At the time of the preparation of the UNSCEAR 1977 Report [U5], the Committee had access to (a) detailed analyses of mouse data obtained after chronic gamma-ray exposures and estimates of doubling doses therefrom [S2, S3]; (b) the results of the continuing studies of mortality rates among children born to survivors of the atomic bombs in Hiroshima and Nagasaki, published by Neel et al. [N1]; and (c) new data on the prevalence of Mendelian and chromosomal diseases, as well as on the prevalence of diseases of complex aetiology, from an extensive survey carried out in the Canadian province of British Columbia and published by Trimble and Doughty [T1].

26. The estimates of doubling doses for the mouse for different genetic end-points fell in the range 80-249 R [S2, S3]. Analysis of the Japanese mortality data by Neel and colleagues showed no significant effects of parental exposure on the mortality of children through the first 17 years of life but suggested that the doubling dose for this kind of damage is about 46 rem for fathers and about 125 rem for mothers, for the acute radiation conditions that obtained during the bombings. Neel et al. suggested that, on the basis of mouse data, the gametic doubling dose for human beings would be expected to be 3-4 times the value of 46 rem for males and as much as 1,000 rem for females.

27. UNSCEAR examined both the mouse data and the Japanese data mentioned above and concluded that it would be prudent to continue to use the doubling dose of 100 R for estimating human radiation hazards. It also appraised the British Columbia prevalence figures, taking into account, among other things, the results of the Northern Ireland survey, of several *ad hoc* surveys for specific dominant conditions and of new-born surveys for chromosomal anomalies, as well as the uncertainties involved in the aetiology of the multifactorial diseases. The following figures (expressed per 10^6) that were arrived at were used for hazard evaluation: 10,000 dominant and X-linked diseases; 1,100 autosomal recessive diseases (excluding those maintained by heterozygous advantage of the relevant genes); 4,000 chromosomal diseases (including sex-chromosomal aneuploids, autosomal trisomies and unbalanced forms of translocations, but excluding mosaics and balanced structural rearrangements); and 90,000 diseases of complex aetiology—a total of 105,100 diseases per 10^6 live births.

28. Using a doubling dose of 100 rad and the prevalence figure of 105,100 per 10^6 mentioned above, UNSCEAR [U5] estimated that, if the population were continuously exposed to low-LET irradiation at a rate of 1 R per generation, there would be a total of about 185 cases of Mendelian, chromosomal and other diseases per million live births at equilibrium, of which about one third would be expressed in the first generation (0.17% versus 0.06%, respectively, of the assumed prevalence of 10.5%) (see Table 2). These figures were arrived at using the following assumptions: (a) for dominant and X-linked diseases, the first-generation increase will be about one fifth of that at equilibrium; (b) for recessive diseases, there will be no perceptible increase; (c) for chromosomal diseases, all those due to numerical anomalies and three fifths of those due to unbalanced structural rearrangements will be expressed in the first generation; and (d) the mutation component of diseases of complex aetiology is probably about 5%, and the first-generation increment in the frequency of these disorders is probably 10% of that at equilibrium.

4. The BEIR 1980 Report

29. Subsequently, the BEIR Committee published its 1980 Report [B3]. Its summary of risk assessments is reproduced in Table 3. It should be noted that (a) prevalence figures for Mendelian, chromosomal and other diseases are essentially the same as those used in the UNSCEAR 1977 Report; (b) the calculation of risks is based on an assumed doubling dose range of 50-250 rem (instead of 20-200 rem used in the BEIR 1972 Report); (c) the risks are expressed for a population exposure of 1 rem per generation (instead of the 5 rem per generation used earlier); and (d) whereas in 1972 the doubling dose method was the preferred method of expressing risks at equilibrium and in the first generation, in the 1980 Report only the equilibrium values were obtained using the doubling dose method (the first-generation values given in Table 3 were arrived at using a direct method, to be discussed later).

30. Concerning the new doubling dose range 50-250 rem used in the BEIR 1980 Report, the

Committee stated: "... this is based mainly on our best substantiated estimates of the doubling dose; namely, 114 R for mouse spermatogonia; we approximately halve and double this to get our range of 50 to 250 rem". Moreover, although a direct method was used to obtain first-generation figures, it is stated in the Report that such figures can also be arrived at using the equilibrium values (estimated using the doubling dose method) and making assumptions similar to those made in 1972 (namely, for dominant and X-linked diseases, the first generation increase will be one fifth of that at equilibrium and for disorders of complex aetiology about one tenth of that at equilibrium).

5. The ICRP Task Group 1980 Report

31. In 1980, Oftedal and Searle [O1] published the conclusions of a Task Group of ICRP on "genetic risk estimates for radiological protection". Their risk estimates are reproduced in Table 4. While the basic data and several of the assumptions used by the Task Group were similar to those used by UNSCEAR in 1977, the numerical estimates of risk by the former were different. The important differences pertain to risk estimates for diseases of complex aetiology and for diseases stemming from unbalanced products of induced balanced reciprocal translocations. These will now be considered in turn.

32. As may be recalled (paragraph 28), it was estimated in the UNSCEAR 1977 Report that the risk of induction of diseases of complex aetiology is a product of their prevalence (taken to be 90,000 per 10^6 live births, on the basis of the results of the British Columbia study); their average mutation component (assumed to be 5%); the relative mutation risk (estimated as 1/100 on the basis of the doubling dose estimate of 100 rad); and the dose sustained (assumed to be 1 rad per generation). The figure arrived at was 45 cases per 10^6 live births (i.e., $90{,}000 \times 1/100 \times 0.05$) at equilibrium. Under the assumption that the increase in the first generation would be about 10% of the above, the Committee derived a figure of 4.5 cases per 10^6 live births in the first-generation progeny.

33. The Task Group did not, however, use any prevalence figure for the above class of diseases to make risk estimates. Instead, (a) they split up the diseases of complex aetiology into (i) dominants of incomplete penetrance and multifactorial diseases maintained by mutation (i.e., those that respond to induced mutation) and (ii) multifactorial diseases not maintained by mutation (i.e., those that do not respond to induced mutation) and (b) they assumed that the expected increase in the frequency of group (i) above (as a result of radiation exposure to the population) is unlikely to exceed the sum of expected increments in Mendelian and chromosomal diseases; in the Task Group's calculations, this amounted to 160 cases per 10^6 live births at equilibrium. Thus, the estimate of 160 cases per 10^6 live births was arrived at in a way different from that used by UNSCEAR.

34. In the UNSCEAR 1977 Report the risk of production of unbalanced gametes leading to congenitally malformed children (stemming from the induction of balanced reciprocal translocations in males) on the basis of combined marmoset and human cytogenetic data. The estimate was 2-10 affected children per 10^6 live births in the first generation per rad of paternal irradiation. The lower limit of the above range was for chronic gamma-irradiation and the upper limit was for low-dose-rate x-irradiation. The risk for the irradiation of females was considered to be low, but no quantitative estimates were given.

35. The Task Group's estimate for the above class of genetic damage was 30 cases per 10^6 live births per rad of parental (i.e., both sexes) irradiation with low-dose-rate x rays and was based on the same set of marmoset and human cytogenetic data. However, the Task Group assumed that the risk from translocation induction would be the same in both sexes (whereas UNSCEAR assumed that it would be lower in females).

6. The UNSCEAR 1982 Report

36. The risk estimates of the UNSCEAR 1982 Report [U6] are reproduced in Table 5. They are basically the same as those derived in the UNSCEAR 1977 Report [U5], except for two changes: (a) for dominant and X-linked diseases, the first-generation increment was assumed (in 1982) to be 15% of that at equilibrium (instead of the 20% assumed in 1977), based on the calculations of Childs [C3], and (b) diseases due to chromosomal anomalies were split up into those due to numerical anomalies and those due to structural anomalies; it was assumed that the increase due to the induction of numerical anomalies would probably be quite small and that the first-generation increment of those due to structural anomalies would be about three fifths of that at equilibrium. Consequently, the expected total increases at equilibrium (~150 cases per million live births) and in the first generation (~22 cases per million live births) are lower than the corresponding values estimated in 1977 (185 and 63 cases, respectively).

37. It is worth pointing out that at the time the UNSCEAR 1982 Report was prepared, the Committee had at its disposal the papers of Neel et al. [N2] and Schull et al. [S4, S5]. They contained an analysis of all the available genetic data obtained in the continuing studies of the Hiroshima and Nagasaki populations (a follow-up of the material presented by Neel and colleagues in 1974 [N2]). These new data pertained to untoward pregnancy outcomes (i.e., those resulting in children with major congenital defects, still births and deaths in the neonatal period), survival through childhood, incidence of sex-chromosomal anomalies and incidence of biochemical variants (erythrocyte and plasma protein variants, analysed using one-dimensional electrophoresis). In all the calculations, the T65D dosimetry was used and an RBE of 5 for neutrons was assumed. As Schull et al. [S5] pointed out, "... in no instance was there a statistically significant effect of parental exposure ... but for all indicators, the observed effect was in the direction suggested by the hypothesis that genetic damage resulted from the exposure."

38. Doubling dose estimates were made only for the first three indicator traits since the data on biochemical variants were considered too preliminary. The gametic doubling dose estimates presented by Schull et al. [S5] were the following: untoward pregnancy outcomes, (69 ± 93 rem); survival through childhood (F_1 mortality), (171±388 rem); and sex-chromosomal aneuploids, (535 ± 2,416 rem). The weighted average of these estimates is (139 ± 157 rem). Schull et al. [S4] considered that the doubling dose estimate for low doses and low dose rates might be higher by a factor of 3.

39. The main message from these papers is that the doubling dose for human genetic effects may be about 4 Gy, i.e., about four times the value used by UNSCEAR, which would mean that the relative risks estimated by UNSCEAR are too high by a factor of 4. The Committee examined these data and the analysis presented and concluded that, in view of the lack of statistically significant effects and the high standard deviations associated with the doubling dose estimates, it would be premature to use these results for genetic risk assessments at the present time. Therefore, the earlier doubling dose estimate of 1 Gy (based entirely on mouse data) was retained.

7. The NUREG 1985 Report

40. In 1985, a report prepared for the U.S. Nuclear Regulatory Commission entitled "Health effects model for nuclear power plant accident consequence analysis" was published [E7]. This report, hereinafter to be referred to as the NUREG Report, included (among other things) estimates of genetic risk arrived at by the doubling dose method for low-dose-rate, low-dose, low-LET irradiation conditions. Important aspects of these estimates are the following: (a) the basic data on the prevalence of autosomal dominant and X-linked diseases and of irregularly inherited diseases are the same as those used by UNSCEAR in 1982 and by the BEIR Committee in 1980; (b) the doubling dose used is 1 Gy (the same as UNSCEAR used in 1982 but different from the 50-250 rem range used by the BEIR Committee in 1980); (c) the mutation component of irregularly inherited diseases was taken to be in the range 0.05-0.5 (the same as in the BEIR 1980 Report, but different from the value of 0.05 used by UNSCEAR in 1982); (d) the risk estimates were made for a total population of 10^6 persons with 16,000 live births per year (or 480,000 in a 30-year generation), based on 1978 demographic data from the United States (UNSCEAR's estimates were for a population of 10^6 live births); and (e) estimates of risk for the first generation were derived not from the equilibrium values but by using a direct method (UNSCEAR's estimates were derived from equilibrium values as well as by using a direct method). The relevant table from the NUREG Report is reproduced here as Table 6.

8. The UNSCEAR 1986 Report

41. The risks estimated by the Committee in the UNSCEAR 1986 Report (with some additions) are given in Table 7. The additions pertain to risk from recessive diseases and for second generation effects (see footnotes c, e and f of Table 7). It can be seen that (a) the estimates of prevalence and of risk for Mendelian and chromosomal diseases are the same as those used in 1982 and (b) no risk estimates are provided for diseases of complex aetiology (i.e., for congenital anomalies and other multifactorial diseases), although they had been provided in 1982. The reasons for this departure are set out below.

42. New data on diseases of complex aetiology that became available subsequent to the UNSCEAR 1982 Report provided grounds for believing that their prevalences may need upward revision. First, the compilation and analysis of the extensive results on congenital anomalies in the Hungarian population [C5] suggested that their prevalence is about 60,000 per 10^6 live births (compared with 43,000 per 10^6, based on the British Columbia study and used in the UNSCEAR 1977 and 1982 Reports); on the basis of their aetiology, the congenital anomalies can be roughly subdivided into those due to major genes (6% of the total of 60,000 per 10^6); multifactorial causation (50% of the total); chromosomal anomalies (about 5% of the total); environmental, including maternal, factors (about 6% of the total); and aetiology as yet unknown (the remaining 30%, approximately, of the total) (see UNSCEAR 1986 Report, Annex C, paragraphs 51-58).

43. Second, in the same population, the prevalence of other irregularly inherited diseases, most of them of late onset (in middle age and later), was estimated to be about 600,000 per 10^6 of the population (see UNSCEAR 1986 Report for a discussion of these data). Relative to the prevalence used in the UNSCEAR 1977 and 1982 Reports (47,000 per 10^6 live births, based on the British Columbia study), the Hungarian prevalence for these diseases is at least an order of magnitude higher. One must hasten to add, however, that (a) the British Columbia figure relates to those multifactorial diseases with onset before the age of 21, whereas the Hungarian figure pertains to lifetime (taken as 70 years) prevalence, and (b) in Hungary, the actual frequency of affected individuals will be fewer than 600,000 per 10^6 because many of them will suffer from more than one of these diseases. One further point relates to the fact that the diseases included in the Hungarian list (at least 25 entities[a]) are by no means homogeneous, either clinically or aetiologically; the same is true of those included in the British Columbia list.

44. During the preparation of the UNSCEAR 1986 Report, the Committee discussed at length these data, the question of a new risk estimate for these diseases and the appropriateness of the assumptions used earlier (i.e., a doubling dose of 1 Gy, a mutation

[a] Thyrotoxicosis, diabetes mellitus, gout, schizophrenia, affective psychoses, multiple sclerosis, epilepsy, glaucoma, essential hypertension, acute and sub-acute myocardial infarction, varicose veins, allergic rhinitis, asthma, gastric ulcers, idiopathic proctocolitis, cholelithiasis, calculus of kidney and ureter, atopic dermatitis and related conditions, psoriasis and related conditions, systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, juvenile osteochondrosis of the spine and adolescent idiopathic scoliosis.

component of 5% and 10% expression in the first generation). If these assumptions were valid (which seem very doubtful), then, the estimate would be 330 cases of diseases of complex aetiology per 10^6 at equilibrium (i.e., $660{,}000 \times 1/100 \times 0.05 = 330$) under conditions of continuous irradiation at a rate of 0.01 Gy per generation. The expected increases in the first and second generations would then be, 33 and 30 cases, respectively, per 10^6. These estimates are about sevenfold higher relative to those arrived at in 1977 and 1982, on the basis of a prevalence figure of 90,000 per 10^6. Although there is no reason to believe that the actual risk from these diseases would be less than those arrived at in 1977 and 1982, there are at least three reasons why the reliability of the estimates based on the prevalence of 660,000 per 10^6 is open to question: (a) the prevalence figure of 600,000 per 10^6 for multifactorial diseases refers to the number of diseases per 10^6 of the population rather than to the actual number of affected individuals; it also includes conditions of less severity such as allergic rhinitis and psoriasis; (b) the applicability of the doubling dose of 1 Gy (which is based entirely on mouse results for clearly defined genetic end-points) to diseases of complex aetiology remains questionable in the absence of information on the mechanisms of maintenance of these diseases in the population and what effect, if any, radiation would have on their prevalence; and (c) the assumption of a 5% mutation component for the diseases included in the new data-set cannot be scientifically defended in the absence of a systematic analysis. These considerations led the Committee to express the view that it is at present unable to provide a reliable estimate of risk for these diseases. However, there is some hope (see paragraphs 90-91) that the difficulties will be at least partially resolved in the not too distance future.

9. The doubling dose method in retrospect

45. The rationale for the continued use of the doubling dose method for risk evaluation is that it permits one to express risks in tangible terms and that whole classes of genetic effects can be handled as a unit in the absence of information about, for instance, the number of loci involved or their individual mutation rates. The early estimates of doubling doses fell in the range 10-100 R (then, for acute, high-dose-rate irradiation), and the possible representative values lay between 30 and 40 R. With the discovery, in 1962, of dose-rate effects in the mouse, UNSCEAR adopted 1 Gy as the best estimate, and this value is still being used. However, (a) individual estimates for different kinds of genetic damage vary from about 0.8 to 2.4 Gy in the mouse and (b) the analysis of the Hiroshima and Nagasaki data suggests that the value may be higher than 1 Gy. The principal reasons for adhering to the 1 Gy estimate are that the human evidence is far from conclusive and that caution and prudence are the guiding principles in this endeavour. The changes in the estimates of relative risks from the mid-1950s to the present thus reflect increasing knowledge on the prevalence of these diseases in the human population and the evolving assumptions on their possible responses to an increase in mutation rate and not any real breakthrough in the understanding of the genetic sensitivity of human germ cells to ionizing radiation.

B. THE DIRECT METHODS

1. Direct methods used between 1956 and 1972

46. Some of the basic radiation genetic principles that guided the BEAR Committee [B2] as it prepared its 1956 Report were briefly alluded to earlier. In addition, advances in radiation genetics and in theoretical and experimental population genetics had even by that time documented the thesis that the changes due to mutated genes are seldom fully expressed in the first-generation progeny of irradiated individuals and that these mutant genes persist in the population for shorter or longer periods of time, depending on their deleterious effects on fitness, until they are eventually eliminated from the population. The concept of genetic death enunciated by Muller [M2] was current and influenced much of the thinking of geneticists. It seemed logical, therefore, to apply the concept to the estimation of genetic risks. This point of view was succinctly stated by the BEAR Committee [B2] as follows: "One way of thinking about this problem of genetic damage is to assume that all kinds of mutations on the average produced equivalent damage, whether as a drastic effect on one individual who leaves no descendants because of this damage, or a wider effect on many. Under this view, the total damage is measured by the number of mutations induced by a given increase in radiation, this number is to be multiplied in one's mind by the average damage from a typical mutation."

47. It was thought, therefore, that the total risk due to induced mutations could be obtained as the product of three factors (i.e., the number of genes at which mutations can occur, the radiation dose and the rate per gene per unit dose) and that the expression of this risk in the first and succeeding generations could be estimated by population genetic methods. Some limited data were available on mutation rates in mice; the main difficulty was the lack of reliable gene numbers for humans. To circumvent this problem, the BEAR Committee used Drosophila data, dividing the total mutation rate (for recessive lethals per gamete) by that for individual genes, multiplied this ratio by 2 to 3 to allow for mutations with less-than-lethal effects and arrived at a figure of about 10^4; this figure was then used along with the mutation rate inferred from mouse studies to estimate the "total number of mutant genes that would enter the population in the next generation if everyone in the United States of America received a dose of 10 R to the reproductive glands" ($5\ 10^6$ mutant genes) [B2]. It is thus clear that this estimate was for "a hypothetical organism whose mutation rate per gene is that of the mouse and whose gene number is that of Drosophila" (see [B1] for a discussion). The BEAR Committee concluded, however, that ". . . this kind of estimate is not a meaningful one to certain geneticists . . . their principal reservation is doubtless a feeling that, hard as it is to estimate the number of mutants, it is much harder still, at the

present state of knowledge, to translate this over into a recognizable statement of harm to individual persons" [B1]. The method was then abandoned [B1, B3].

48. In its first Report of 1968, UNSCEAR did not use any direct methods. In 1966, it used a variation of the method used by the BEAR Committee. It was argued that (a) if it is assumed that the average rate of induction of recessive mutations in the mouse (estimated as $1\ 10^{-7}$ per locus per R from data obtained for specific locus mutations, following high-dose, high-dose-rate x-irradiation of spermatogonia) is applicable to man and (b) if, for the purpose of computation, the total number of gene loci in man were assumed to be 20,000 (range: 7,000-70,000), then the total risk from the induction of point mutations will be $2\ 10^{-3}$ mutations per gamete per R. This estimate was higher than the one arrived at using the limited data on the induction of autosomal recessive lethals in mouse spermatogonia as a basis ($0.5\ 10^{-3}$ mutations per gamete per R), although not significantly so. The expression of this risk in the first and succeeding generations was then estimated using the average degree of semi-dominance of recessive lethals in Drosophila.

49. For estimating more directly the risk from the induction of dominant mutations, the Committee assumed that the rate is likely to be from 10^{-9} to 10^{-7} per locus per R and that the number of loci determining dominant diseases in man ranges from 50 to 500. The total risk was thus estimated to be from $5\ 10^{-8}$ to $5\ 10^{-5}$ mutations per gamete per R. Similar estimates of risk from the induction of chromosomal aberrations (translocations and deletions) were arrived at using the limited data on the induction of heritable semi-sterility in the mouse and data on the induction of dicentrics and deletions in human peripheral blood lymphocytes. It was pointed out that all the risks would be lower for chronic irradiation conditions.

50. In the UNSCEAR 1972 Report [U4], the Committee approached the problem of estimating gene numbers in the following way. In the mouse, there are about 20 functional units per cross-over unit [R6] and there are some 1,250 map units in the entire genome [G1]. This gives about 25,000 functional units in the mouse genome. The DNA of the mouse and human diploid genomes were estimated to contain $4.7\ 10^9$ and $5.6\ 10^9$ nucleotide pairs, respectively. Thus the estimate of the total number of functional units in the human genome was $25{,}000 \times (5.6/4.7) = 30{,}000$. By assuming that the rate of induction of specific locus mutations in mouse spermatogonia under conditions of chronic gamma-ray exposure ($0.5\ 10^{-7}$ per locus per rad; average estimate based on the 12 loci studied) was applicable to man, the total risk from the induction of point mutations in man was estimated to be 1,500 mutations per rad per million gametes (i.e., $0.5\ 10^{-7} \times 30{,}000$) (Table 8). This was considered an overestimate, because specific locus mutations may involve more than one functional unit.

51. The estimate of total risk based on the new mouse data (discussed in the 1972 Report) on the induction of autosomal recessive lethals (spermatogonial irradiation), after correction for DNA content, was 36 mutations per rad per million gametes, and this was considered a possible underestimate. The first-generation expression of these risks (computed using, as before, the degree of semi-dominance of recessive lethals (2-5%) inferred from Drosophila studies in conjunction with the total rate of 1,500) amounted to 36 mutations per rad per million gametes.

52. A modified version of the gene number approach was also used in the UNSCEAR 1972 Report for estimating the rate of induction of dominant mutations in man. For this purpose, the rate of induction of dominant visible mutations in mouse spermatogonia ($4.96\ 10^{-7}$ per rad per gamete) was used. Since this rate was for acute, high-dose-rate x-irradiation, it was divided by 3 to correct for effects at low dose rates and divided by 75 (the then-presumed number of loci in the mouse that mutated to dominant visibles), giving a rate of $2.2\ 10^{-9}$ per locus per gamete per rad.

53. The 1971 compendium of McKusick [M3] listed 415 autosomal dominant loci in man plus another 528 for which the evidence was inconclusive. Assuming that there was "good reason to predict that the number will not be less than 1,000 based on progress of research in this area", the Committee multiplied $2.2\ 10^{-9}$ by 1,000 and obtained an estimate of about two dominant mutations per rad per million gametes [U4].

54. In its 1972 Report, UNSCEAR also provided an estimate of risk from the induction of reciprocal translocations in man, using a direct method. This was done by assuming that (a) the rate of induction of balanced reciprocal translocations in man would be twice that in mouse germ cells (i.e., $2 \times 0.3\ 10^{-4}$ per gamete per rad, based on the arm number hypothesis of Brewen et al. [B4], which was then, but is not now, considered valid); (b) at low doses or low dose rates, the rates in males would be reduced by factors of 4 or 7, respectively; (c) the ratio of balanced to unbalanced products would be 1:2; and (d) only about 6% of the unbalanced products would result in liveborn children with multiple congenital anomalies. Taking all these assumptions into account, it was estimated that the risk was 1-2 congenitally malformed progeny per million live born, following irradiation of males. (It is worth mentioning that in its 1972 Report, the BEIR Committee also provided an estimate of risk from the induction of reciprocal translocations; using the same basic data and a different set of assumptions, it arrived at an estimate that was an order of magnitude higher than the estimate of UNSCEAR.)

55. In retrospect, despite gallant efforts to weld the principles of population genetics, the concept of genetic death and the principles of radiation genetics in order to arrive at risk estimates for the induction of mutations, the hopes that, until 1972, had been raised by the gene number method proved illusory. Apart from the difficulties of reliably estimating gene numbers, the conceptual difficulties of bridging the gap between the dynamics of mutant genes in the population, on the one hand, and genetic disease, on the

other, were so numerous that the method fell short of expectations and faded into oblivion. Furthermore, the degree of semi-dominance of recessive mutant genes, used to extract first-generation effects from the total risks, referred in reality to the effects of these genes on biological (reproductive) fitness and not to genetic diseases as such.

2. The UNSCEAR 1977 and BEIR 1980 Reports

56. In the UNSCEAR 1977 Report, a major conceptual change was introduced. At the time of preparation of the above Report, the Committee had at its disposal the earlier data collected by Ehling [E1, E2] on the induction of dominant mutations affecting the skeleton of the progeny of irradiated male mice and the new data on these collected by Selby and Selby [S7]. The data of the latter authors established that these skeletal mutations were in fact transmissible and also showed that they had incomplete penetrance and variable expressivity for many or all of the phenotypic effects they caused and, besides, that some of them behaved as recessive lethals when made homozygous. Doubtless, some of the properties of these mutations are similar to those of rare dominants and rare, irregularly inherited dominants in man.

57. However, in order to convert the rate of induction of mutations causing skeletal abnormalities in the mouse into an overall rate for all mutations with dominant phenotypic effects in man, information is needed on (a) the proportion of dominant conditions in man whose main effect is in the skeleton and (b) the proportion of skeletal abnormalities studied in the mouse that, at the human level, is likely to cause a serious handicap. After careful deliberation, UNSCEAR arrived at values of 10% for item (a), taking into account the proportion of clinically relevant autosomal dominants in man whose main effect is in the skeleton (which was assessed at 20%) and the ease of diagnosis of skeletal defects (possibly higher by a factor of 2) relative to defects in other bodily systems. The proportion of skeletal anomalies in the mouse that might cause a serious handicap (item (b) above) should they occur in man, was assessed as one half.

58. In risk estimation, the frequency of skeletal mutations observed after high fractionated or high acute x- or gamma-ray doses was corrected by suitable factors to arrive at a rate that would be applicable for low-dose-rate, low-LET irradiation conditions. The resultant rate of $4\ 10^{-6}$ was multiplied by 10 and divided by 2, to give $20\ 10^{-6}$, which is the probability of induction of mutations causing dominant effects in any of the bodily systems in man. In other words, following 1 R of paternal (spermatogonial) irradiation, 20 per million progeny would carry mutations causing one or another kind of dominant genetic disease, in the first generation (see Table 9).

59. In its 1980 Report, the BEIR Committee, starting from the same data-base as that used by UNSCEAR, gave a different estimate (5-65 cases per million per rem; see Table 3). There are two reasons for this difference. First, to convert the rate of induction of skeletal mutations in mice to an overall rate involving all bodily systems in human beings, UNSCEAR used a factor of 10, as mentioned in the preceding paragraph; this was divided by 2 to exclude mutations whose effects were slight.

60. The BEIR Committee, by contrast, multiplied by 5-15 to make the first conversion and multiplied by 0.25-0.75 to make the second conversion: these operations gave the range of 5-45 cases per million births. Second, to take into account the effects of irradiation of females, the BEIR Committee multiplied the upper limit by 1.44 (the UNSCEAR 1977 Report assumed that the risk for irradiated human females would be negligible and did not give any quantitative estimate). The rationale for the multiplication by 1.44 was stated as follows: "The mutational response of the resting oocytes in mice is negligible, compared with that of spermatogonia, and mature and maturing oocytes in mice have a mutation rate no greater than 0.44 times that found in spermatogonia. We do not know which of the two classes of oocytes would have a mutational response more similar to that of the arrested oocytes in women. To incorporate this range of uncertainty into our risk estimate for the combined effects of irradiation of both sexes, we have simply kept the lower limit of our estimate the same as it was (assuming a negligible mutation frequency in resting oocytes) and multiplied the upper limit by 1.44 (assuming the maximal estimate of the mutation frequency in mature and maturing oocytes). This gives an estimate of 5-65 induced serious disorders per million liveborn as the first generation expression. . . ."

61. In 1977, as may be recalled, using marmoset and human cytogenetic data [B5] as the basis for estimating the risk from the induction of reciprocal translocations, UNSCEAR arrived at an estimate of between 2 and 10 congenitally malformed children per million births per rad of low-LET irradiation of males. Briefly, the calculations were the following: $7\ 10^{-4}$ (rate of translocation induction; spermatocyte data) $\times$ 0.25 (factor to get the rate for heritable translocations in the progeny) $\times$ 0.1 or 0.5 (factors to account for dose-rate effect after chronic gamma-rays and low-dose x rays, respectively) $\times$ 2 (factor to estimate the rate of production of unbalanced products of reciprocal translocations) $\times$ 6% (the proportion of unbalanced products that was assumed to give rise to viable, but congenitally malformed, progeny). The risk for the irradiation of human females was considered to be small, but no quantitative estimate was given.

62. In its 1980 Report, the BEIR Committee used the marmoset cytogenetic data for estimating the risk associated with the induction of balanced reciprocal translocations in human males. Its estimate was $0.5\text{-}5\ 10^{-6}$ per rem (compared to that of UNSCEAR, $2\text{-}10\ 10^{-6}$ per rad, as reported in the preceding paragraph). The BEIR Committee's procedure was the following:

(a)	Basic rate	$7.7\ 10^{-4}$e/m/spermatocyte (marmoset cytogenetic data)
(b)	Rate of potentially transmissible rearrangements	$4.7\ 10^{-4}$e/m (multiply (a) by 2/3)

(c)	Correction for low total doses and for low dose rate	$2.3\ 10^{-4}$e/m (multiply (b) by 0.5)
(d)	Correction for the probability of adjacent segregations (unbalanced gametes)	$1.3\ 10^{-4}$e/m (multiply (c) by 0.55)
(e)	Correction for proportion of unbalanced gametes that would result in congenitally malformed live births	$6.5\ 10^{-6}$e/m (multiply (d) by 0.05)
(f)	Correction for the probability that only one of the four kinds of aneuploid segregation products will be viable in zygotes	$1.6\ 10^{-6}$e/m (multiply (e) by 0.25)
(g)	Correction for uncertainty	$0.5\text{-}5\ 10^{-6}$e/m (multiply (f) by 0.3-3)

63. For estimating risks for irradiated human females, the BEIR Committee made the same assumption as in its 1972 Report; namely, that the risk would be the same as that in males. Thus "the expected frequency of viable aneuploids for both sexes is assumed to range from $1\ 10^{-6}$ to $10\ 10^{-6}$ per rem". The BEIR Committee also used "an alternative and independent approach based on litter-size reduction observed after acute irradiation of mouse germ cells" and pointed out that "the upper limit of $10\ 10^{-6}$ for both sexes combined may be an overestimate, and that the true value could indeed be near to zero".

3. The UNSCEAR 1982 Report

64. The estimates of risk arrived at in the UNSCEAR 1982 Report are given in Table 10. They are basically the same as those arrived at in 1977, except for the following additions or changes: (a) an additional and independent estimate of risk from the induction of mutations having dominant effects in the F_1 progeny was presented, using the new data on the induction of dominant cataract mutations in male mice following spermatogonial irradiation; (b) the probable magnitude of risk for irradiation of females was arrived at very indirectly by assuming, on the basis of specific locus data, that the rate in females is likely to be either close to zero (on the assumption that the mutational sensitivity of the human immature oocytes may be similar to that of the mouse immature oocytes) or not more than 44% of the spermatogonial rate (see [R7]); and (c) for estimating risks from the induction of reciprocal translocations, all the then-available primate data (i.e., the marmoset data, the rhesus monkey data and the limited human data) were used.

65. The mouse cataract mutation data were obtained in experiments involving high acute or fractionated gamma-ray exposure [E3, E4, K1]. From these results, a rate applicable to chronic gamma-ray exposure was derived, using empirical correction factors derived from specific locus studies. This rate was about $2.6\ 10^{-7}$ per gamete per rad. In man, about 2.7% of all known and proven dominant mutations are associated with one or another form of cataract. The reciprocal of this (i.e., 100/2.7 = 36.8) was used as a factor by which to multiply the rate of $2.6\ 10^{-7}$, to arrive at approximately 10×10^{-6} per rad. In other words, for every rad of low-dose-rate, low-LET irradiation, about 10 individuals per million born will be affected by one or another kind of serious, clinically important, genetic disease. It is worth pointing out that these calculations did not use either the multiplier of 2 or the divisor of 2 that had been used in analogous computations with skeletal mutations to account for ease of diagnosis and severity of effects, respectively.

66. The extensive cytogenetic data on translocation induction in male rhesus monkeys show that the rate of induction, at least for high-dose-rate x-irradiation conditions, is much lower than that in marmosets ($0.86\ 10^{-4}$ versus $7.7\ 10^{-4}$ per rad per gamete) (see [B6]). Since it is not known whether human spermatogonial sensitivity is more like that of the marmoset or that of the rhesus monkey, the rates for both these species were used, one to define the probable upper limit of risk and the other, the lower limit. The correction factors used to convert the rhesus monkey acute rate into the rate applicable to human radiation conditions are the same as those used in the 1977 Report. Other correction factors and the underlying assumptions are explained in the footnotes to Table 10.

4. The NUREG 1985 Report and other publications

67. The direct estimates of risk presented in the NUREG Report [E7] (Table 6) differ in some respects from those in the BEIR 1980 Report. First, the NUREG Report assumes that both sexes have the same sensitivity to the induction of dominant mutations (the BEIR Committee assumed that the sensitivity of females is no more than 0.44 times that of males). The consequence of this assumption is that for irradiation of both sexes, the risk of induction of dominant genetic disease is twice that estimated for exposure of males; namely, $2 \times (5\text{-}45)\ 10^{-6}$ per 0.01 Gy, or $10\text{-}90\ 10^{-6}$ per 0.01 Gy. The geometric mean of this range (central estimate) is 30. As may be recalled (paragraph 60), the BEIR Committee multiplied the upper limit of the range 5-45 by 1.44 and came up with the range $5\text{-}65\ 10^{-6}$ per 0.01 Gy for irradiation of both sexes.

68. Second, the NUREG Report employed the gene number method (which is no longer used by either UNSCEAR or the BEIR Committee) to arrive at a risk estimate for X-linked genetic diseases. Assuming that the rate of specific locus mutations in male mice ($7.2\ 10^{-8}$ per locus per 0.01 Gy) is applicable for X-linked mutations as well, and that the number of loci in man at which X-linked mutations occur is 250, an estimate of $1.8\ 10^{-5}$ per 0.01 Gy was obtained for irradiation of males or females.

69. To obtain a "single central estimate" for induced translocations and the unbalanced segregation products, some of which produce viable but seriously affected live born, the NUREG Report used the following calculations:

(a)	Basic rate	$7.4\ 10^{-4}$/.01 Gy/spermatocyte (marmoset cytogenetic data)

(b)	Correction for low-dose-rate x rays	$3.7\ 10^{-4}$/.01 Gy/spermatocyte (multiply (a) by 0.5)
(c)	Correction of gamma-ray RBE	$1.48\ 10^{-4}$/.01 Gy/spermatocyte (multiply (b) by 0.4)
(d)	Correction for expected frequency of unbalanced products	$0.74\ 10^{-4}$/.01 Gy (multiply (c) by 0.5)
(e)	Correction for proportion of viable unbalanced products	$0.74\ 10^{-6}$/.01 Gy (multiply (d) by 0.01)

For irradiation of females, the rate was assumed to be the same as that in males (i.e., $1.48\ 10^{-4}$ per 0.01 Gy), and since the oocyte will contain the reciprocal translocation distributed between two tetrads of chromatids, the probability of recovering unbalanced products was estimated as nine sixteenths of the above; namely, $5.6\ 10^{-5}$ per 0.01 Gy. Assuming further that about one tenth of this would lead to viable aneuploids, it was estimated that the risk for irradiation of human females would be $5.6\ 10^{-6}$ per 0.01 Gy; for irradiation of both sexes, therefore, the total risk from induced translocations would be about 13 cases of abnormal progeny per million live births per 0.01 Gy of parental irradiation.

70. In addition to risk estimates for low-dose, low-dose-rate, low-LET irradiation conditions, the NUREG Report also provided estimates of genetic risk for the irradiation conditions that would prevail in nuclear accidents. As would be expected, the risks are higher under the latter conditions, but this case will not be further considered here.

71. The NUREG Report also sought to determine whether its risk estimates were consistent with the lack of detectable genetic effects of radiation in the genetic studies of Hiroshima and Nagasaki. In its analysis of the Japanese data, it used (a) the paper of Schull et al. as a basis [S13; specifically, Table 7, in which mortality up to age 17 for 16,173 children of exposed parents is correlated with the distribution of parental doses]; (b) the linear-quadratic model as the one applicable for the induction of gene mutations and chromosomal aberrations at high doses and high dose rates (developed independently of the Japanese data, on the basis of the experimental results discussed in the preceding paragraph); and (c) average doses for each exposure group (parents), i.e., 0.05 Gy (0.01-0.09 Gy); 0.295 Gy (0.10-0.49 Gy); 0.745 Gy (0.50-0.99 Gy); and 2 Gy (>1 Gy). These values were introduced into the equations to project the number of cases of each genetic event relative to the child sample size in each of the 32 sectors of exposure in the matrix of Schull et al. [S13].

72. The important conclusion that emerged from this analysis was that "the central estimate of prediction of cases should lead to a statistically insignificant, i.e., undetectable increase in genetic disorders among the 16,713 progeny of irradiated parents. For example, there were 1,040 deaths in this group of 16,713 progeny up to the age of 17 (6.22%); in the unexposed groups, there were 2,191 deaths among 33,976 progeny produced (6.45%) and the two frequencies are not significantly different, nor would they have been even if 50 additional cases were added to the exposed group [E7]."

73. Abrahamson [A2] and Ehling [E8] independently reached similar conclusions. In his analysis, Ehling used the estimate of the genetically significant dose (sustained by the survivors of Hiroshima and Nagasaki) of $1.1\ 10^{4}$ man Sv arrived at by WHO in its report entitled "The effects of nuclear war on health and health services" [W1]. The projected increases among the 19,000 children in the sample were less than one dominant cataract mutation and about 11 dominant skeletal mutations; the total number of expected dominant mutations was estimated to be 20-25 (on the basis of mouse cataract data) or about 56 (on the basis of mouse skeletal data). Ehling argued that, given the kinds of end-points used in the Japanese studies, (a) it is not surprising that no clear-cut positive evidence for the induction of genetic damage could be obtained and (b) the Japanese results are not inconsistent with the expectations based on the dominant cataract and skeletal data.

5. The UNSCEAR 1986 Report

74. The most recent estimates of risk, those of the UNSCEAR 1986 Report, are given in Table 11. The changes with respect to the estimates of 1982 are the following. First, on the basis of litter-size reductions observed in mouse studies involving acute, high-dose-rate x-irradiation, chronic gamma-irradiation or chronic fission neutron irradiation (in all, spermatogonial irradiation; these data are discussed in the 1986 Report), the Committee estimated that, following irradiation of male mice, between 5 and 10 per 10^{-2} Gy per million live born (in the first generation) would die between birth and weaning as a consequence of induction of dominant sub-lethal effects. In view of the uncertainty as to whether and to what extent litter-size reduction in mice can be extrapolated to mortality in humans between birth and early childhood, the above estimate has been appended to Table 11 as footnote b.

75. The second change (see footnote e in Table 11) pertains to the risk of induction of autosomal recessive genetic disease as a result of radiation exposures. In the past, the Committee's view has been that although recessive mutations are expected to be induced, no cases of individuals affected with recessive disease will occur in the first generation; it therefore made no attempt to present a quantitative estimate of risk in the subsequent generations following one-time or generation-after-generation radiation exposure. Searle and Edwards [S9] have now provided a numerical estimate of risk for autosomal recessive disorders. Basing their calculations on a combination of data from observations in human populations and in mice, these authors have estimated that (a) a genetically significant dose of 10^{-2} Gy of x- or gamma-irradiation received by each parent once in a stable population with 1 million live born would induce up to 1,200 additional recessive mutations; (b) from partnership effects (i.e., partnership with another recessive mutation

either induced or already present in the population), about one extra case of recessive disease would be expected in the following 10 generations; (c) homozygosity resulting from identity by descent would not normally occur until the fourth generation after exposure, but on certain assumptions, about 10 extra cases would be expected from this cause by the tenth generation; and (d) in the same period, about 250 recessive alleles would be eliminated as heterozygotes, on the assumption of a 2.5% heterozygous disadvantage. Such elimination through heterozygotes may occur through, for example, an increase in disease susceptibilities, malignancy or a decrease in intellect.

76. The third change pertains to risks associated with the induction of balanced reciprocal translocations. In its 1982 Report, the Committee used the cytogenetic data collected in studies with the rhesus monkey and the marmoset Saguinus fuscicollis, as well as some limited human data, as a basis for its risk estimates. The rates of unbalanced products of reciprocal translocations expected to be generated (estimated from the corresponding rates for balanced reciprocal translocations) were used in conjunction with the assumption that 6% of unbalanced zygotes might result in congenitally malformed children, giving the range 0.3-10 per 10^{-2} Gy per 10^6 live births (the lower limit was based on the rhesus monkey data and the upper one on the combined marmoset and human data). New data at low doses and low dose rates have since then become available from experiments involving the rhesus monkey and the crab-eating monkey; further, the estimate of conceptions with unbalanced products that may survive birth has been revised upwards, from 6% to 9% (discussed in the UNSCEAR 1986 Report). As a result, the estimated risk is now 1-15 cases of congenitally malformed children per 10^6 live births per 10^{-2} Gy of paternal irradiation and 0-5 cases per 10^6 live births per 10^{-2} Gy of maternal irradiation. Further details are given in footnotes f and g to Table 11.

II. RELEVANT NEW HUMAN DATA PUBLISHED AFTER THE UNSCEAR 1986 REPORT

77. The most recent results from the Hiroshima and Nagasaki genetic and cytogenetic studies again show no significant effects of radiation. The mutation work [S12] involved examining 13,052 children of proximally exposed parents and 10,609 children of control parents for rare electrophoretic variants of 30 blood proteins; three mutations were detected in each group in 725,587 and 539,170 equivalent locus tests, respectively. The mutation rates can therefore be estimated as $0.4\ 10^{-5}$ per locus (exposed group) and $0.6\ 10^{-5}$ per locus (control group). In a subset of 4,983 children of exposed parents (= 55,689 equivalent locus tests) and 5,026 control children (= 59,269 equivalent locus tests) who were studied for deficiency variants of nine erythrocyte enzymes, one mutant was encountered in the first group (rate: $1.80\ 10^{-5}$ per locus) and none in the second.

78. In cytogenetic studies [A1], among 8,322 children born to exposed survivors, 19 (0.23%) had sex-chromosomal anomalies (3 XYY, 7 XXY, 5 XXX, 3 mosaics and 1 miscellaneous), and 23 (0.28%) had structural rearrangements (10 Robertsonian translocations, 7 balanced reciprocal translocations, 1 inversion, 2 unbalanced supernumeraries and 3 miscellaneous unbalanced aberrations). Of 7,976 control children, 24 (0.30%) had sex-chromosomal anomalies (5 XYY, 9 XXY, 4 XXX, 3 mosaics and 3 miscellaneous) and 27 (0.34%) had structural rearrangements (6 Robertsonian translocations, 13 reciprocal translocations, 6 inversions and 2 miscellaneous unbalanced aberrations). Of the 11 balanced structural rearrangements for which family studies were made, one reciprocal translocation in each group was a new mutant, the rest were familial.

III. RISK COEFFICIENTS FOR GENETIC EFFECTS

79. All the numerical estimates of genetic risks discussed thus far have been obtained on the basis of genetically significant doses, i.e., on the assumption that the doses are received by individuals before or during the reproductive period. It is obvious that in the case of population exposures, the genetically significant doses are markedly less than the total doses received over a lifetime: damage sustained by germ cells of individuals who are beyond the reproductive period or who are not procreating for any other reason, poses no genetic risks. If it is assumed that the mean age at reproduction is 30 years and the average life expectancy at birth is 70-75 years, the dose received by 30 years is about 40% of the total dose.

80. To derive risk coefficients for genetic risks to a population, therefore, one needs to multiply the genetic risk estimates discussed earlier by 0.40. The following calculations make use of the most recent risk estimates presented in Table 7: (a) risk coefficient on the basis of gonadal dose in the reproductive segment of the population (from Table 7); for quantifiable damage only, over all generations, per Gy: ~12,000 per 10^6 or 1.2% ; (b) risk coefficient to the population; for quantifiable damage only, over all generations, per Sv ($1.2 \times 0.4 = 0.5$): 0.5%; (c) risk coefficient for the first two generations under conditions otherwise similar to (a) above (from Table 7): 3,100 per 10^6 or 0.3%; (d) risk coefficient to the population for the first two generations, per Sv (0.3×0.4): 0.1%. It is useful to reiterate here that these risk coefficients are for conditions of continuous exposure at a finite rate every generation and that they also reflect the total genetic risk from a once-only exposure of the parents.

IV. ESTIMATES OF DETRIMENT

81. The estimates of risk discussed in the preceding sections refer to the expected number of cases of serious genetic disease due to radiation-induced muta-

tions and chromosomal aberrations in the progeny of irradiated parents. The Committee had always realized (although it had not explicitly stated so in Reports prior to 1982) that presenting numerical estimates is just one aspect of risk estimation. Without some objective and quantifiable indicators of severity, it is difficult to perceive the impact of these on the individual and on the society at large and to make comparisons with the risks of induction of other biological effects, such as cancer. Therefore, starting with the UNSCEAR 1982 Report, the Committee began a systematic review of data bearing on this problem, focusing on spontaneously arising Mendelian, chromosomal and other diseases in order to gain some perspective of the detriment associated with these diseases and hoping to be able to use this knowledge to assess the impact of radiation-induced diseases at some later stage.

82. Particularly important in this context are the rough estimates of the disability caused and the length of life lost by the more common genetically determined (i.e., Mendelian and chromosomal) diseases provided by Carter [C4] and discussed in the 1982 Report. Carter's analysis revealed that for monogenic diseases (autosomal dominants, X-linked recessives and autosomal recessives) and for an estimated total birth prevalence of 12,500 per 10^6, about 190,000 years of life are lost, 300,000 years of life are potentially impaired and about 150,000 years of life are actually impaired per 10^6 live births. For chromosomal diseases, Carter's figures are about 89,000 years (lost life), 180,000 years (potentially impaired life) and about 90,000 years (actually impaired life) per 10^6 live births. The average life expectancy at birth assumed in these calculations was 70 years.

83. Czeizel and Sankaranarayanan [C5] extended this analysis to spontaneously arising congenital anomalies in man (these results are discussed in the UNSCEAR 1986 Report [U7]). The data on birth prevalences for the various conditions considered were derived from several epidemiological surveys carried out in Hungary and from the Hungarian Congenital Malformation Registry. Most of the information on mortality profiles was obtained from the records of the Hungarian Central Statistical Office in Budapest. Their analysis showed that with an estimated prevalence of about 60,000 per 10^6 live births in Hungary, the congenital anomalies may cause about 480,000 years of lost life, about 3,700,000 years of potentially impaired life (including congenital dislocation of the hip, whose birth prevalence is 25,770 per 10^6; and which, if excluded, would reduce the potentially impaired life figure to about 1,800,000 years) and about 4,500 years of actually impaired life. In these calculations, it has been assumed that the average life expectancy at live birth is 70 years.

V. UNCERTAINTIES AND PERSPECTIVES

84. The foregoing discussion amply documents the fact that there have been only a few changes in the numerical estimates of risk since the UNSCEAR 1977 Report. However, this statement is not meant to imply that there have been no advances in our knowledge in the areas relevant to genetic risk evaluation in recent years; rather, it reflects the fact that the impact of these advances has still not been fully assessed, as illustrated below.

A. PREVALENCE OF MENDELIAN, CHROMOSOMAL AND OTHER DISEASES

1. Mendelian diseases

85. There is no need to belabour the point that a sound knowledge of the contribution of gene mutations and of chromosomal aberrations to genetic diseases is of paramount importance, not only because it affords a perspective on those diseases to which man is literally heir, but also because it provides a frame of reference within which to appraise the increases expected as a result of radiation (or other mutagenic exposures). Our current estimates of prevalences of Mendelian diseases are based on a total of about 50 entities, the individual birth frequencies of which range from about $1\ 10^{-5}$ to about $1\ 10^{-4}$, with some upward adjustment in the total frequency for those diseases yet to be discovered. For obvious reasons, these estimates pertain to a very small proportion of those listed in the recent update of the McKusick compendium [M4] (i.e., a total of 1,906 conditions that are well documented as being inherited in a Mendelian manner and a further 2,001 for which such evidence is incomplete). The disparity is even greater when one considers that the total amount of nuclear DNA in the human haploid genome is about $3\ 10^6$ kb, which in principle can accommodate between 100,000 and 150,000 genes (on the assumption that an "average" gene is 20-30 kb long). However, this estimate is probably too high, since it neglects introns, pseudogenes, highly repetitive sequences such as satellite DNA and other interspersed and non-transcribed DNA sequences [B7, D2, E5, E6, J1]. Judging from the phenomenal progress in the understanding of the human genome that has been made possible in recent years by applying the methods of recombinant DNA technology (reviewed in the UNSCEAR 1986 Report [U7]), one can confidently look forward to (a) a better understanding of the relationship between genes and mutations and their effects on health and disease and (b) the application of the knowledge so derived to the evaluation of genetic risk.

2. Chromosomal diseases

86. At the chromosomal level, the application of banding techniques to the study of human chromosomes has led to the identification of a variety of chromosomal defects, particularly deletions and duplications involving every chromosome of the human complement (reviewed in the UNSCEAR 1977 Report [U7]). However, since most of the currently available information on these anomalies is in the form of case reports, their prevalences and their contribution to disease states cannot be readily determined. Of particular interest are microdeletions observed in mal-

formed children, especially those predisposed to a number of cancers. They provide new ways to localize and characterize important genes involved in pathology, both at the chromosomal and molecular levels. They also suggest that a pathological character may result from the presence of a pre-existing abnormal allele and a somatic mutation.

87. One exciting development in human cytogenetics in recent years has been the discovery of fragile sites on chromosomes, which can be made visible by appropriate culturing techniques (reviewed in the UNSCEAR 1982 and 1986 Reports [U6, U7]). As was already mentioned, the fragile site on the X-chromosome (at position Xq27) has elicited considerable attention because it is associated with one form of X-linked mental retardation. The prevalence of fragile-X-associated mental retardation has been estimated to be about $4\ 10^{-4}$ both in males and females [S6]: this would make the fragile-X the second most common (after Down's syndrome) chromosomal abnormality associated with mental retardation. It is currently not known whether this abnormality can be induced by radiation, so no genetic risk assessments can be made.

3. Congenital anomalies and other diseases of complex aetiology

88. Previous estimates of the prevalence of diseases of complex aetiology (i.e., congenital anomalies (43,000 per 10^6) and other multifactorial diseases (47,000 per 10^6) are based on the results of the British Columbia survey [T1], in which the follow-up period was from birth to age 21. Recent studies of Czeizel and Sankaranarayanan ([C1] and Czeizel et al. [C7]; discussed in the UNSCEAR 1986 Report [U7]) lend credence to the view that the above prevalence figures (in particular those of the "other multifactorial diseases") are underestimates. Their data show that in Hungary (a) congenital anomalies have a prevalence of about 60,000 per 10^6 live births and (b) the "other multifactorial" diseases may have a prevalence of about 600,000 per 10^6 population when all individuals up to age 70 are included. It should be made clear that the latter figure refers to the number of diseases per 10^6 and not to affected individuals; thus, some individuals have more than one disease.

89. As was discussed in the UNSCEAR 1986 Report (Annex C, paragraphs 51-58; see also Kalter and Warkany [K2]), the congenital anomalies can be subdivided, on the basis of their aetiology, into those caused by: (a) major genes (6% of the total of 60,000 per 10^6); (b) multiple factors (50% of the total); (c) chromosomal anomalies (5% of the total); and (d) environmental, including maternal, factors (6% of the total). About 30% of the anomalies recorded at birth have no known cause at present. One should add, however, that even for those congenital anomalies whose transmission patterns are consistent with a multifactorial aetiology (i.e., those resulting from an interplay of polygenic genetic predisposition and environmental factors), little is known of the mechanisms by which genetic predisposition acts or of the environmental factors involved.

90. The same is true of the "other multifactorial diseases" whose population prevalence has been estimated to be about 600,000 per 10^6. The question of whether and to what extent the prevalence of all these multifactorial diseases will increase as a result of radiation exposures remains a matter of conjecture. Our ability to make reliable estimates for these diseases depends largely on establishing the role of genetic factors in their aetiology. While no quantum leaps are expected in this area, there are persuasive, if not yet compelling, reasons to believe that it may soon be possible to estimate the mutation component in some of the seemingly multifactorially inherited diseases. The use of restriction fragment analysis and the development of more rapid methods for sequencing DNA will, in time allow a search for mutational variation at specific loci known to be involved which contribute to the occurrence of these diseases. The following examples are illustrative.

91. In a large Icelandic family (over 200 members in four living generations) showing Mendelian inheritance of X-linked secondary cleft palate and ankyloglossia ("tongue-tied"), Moore et al. [M10] localized the gene to Xq13-Xq21; the eventual cloning of the cleft palate gene could become a starting point for the analysis of the genetic basis of this developmental abnormality. A similar analysis using restriction fragment length polymorphism in an Old Order Amish pedigree made it possible to localize a dominant gene conferring a strong predisposition to manic depressive illness (a form of affective psychosis) to the tip of the short arm of chromosome 11 [E9]; however, in three Icelandic kindreds [H3] and three North American pedigrees [D3] there was no linkage with any of the chromosome 11 probes used. The inference is that mutations at different loci are responsible for the manic depressive phenotype in the Amish and in the other two population groups studied. Two other areas with particular promise involve lipid metabolism, where it is already possible to specify the contribution of specific loci concerned with the apolipoproteins to the variation in cholesterol levels seen among individuals (see [B9] for a recent review) and the transport of sodium and potassium which looms large in the occurrence of essential hypertension (reviewed in [H4] and [W2]).

B. MUTATIONAL MECHANISMS AND DNA REPAIR

92. An increasingly important area of contemporary genetic research is that associated with the studies on movable genetic elements, the conceptual framework for which was laid by McClintock, over three decades ago, with her work on "controlling elements" in maize [M6, M9]. Now, movable genetic elements have been discovered in a number of prokaryotes and eukaryotes, and some of these elements have been characterized at the molecular level. The rapidly accumulating evidence shows that they play an important role in the genesis of spontaneous mutations and of chromosomal aber-

rations; this knowledge, in turn, is altering our concepts about mutational mechanisms and the stability of the genome (discussed in the UNSCEAR 1986 Report [U7]).

93. Information on mammalian interspersed repetitive DNA sequences and why they are (or have at one time been) considered to be mobile genetic elements has been the subject of a number of recent reviews [R8, S14, S15]. These putative mobile genetic elements are currently classified into two groups: SINEs (for *s*hort *in*terspersed *e*lement*s*), which are typically less than 500 base pairs long and LINEs (*l*ong *in*terspersed *e*lement*s*), which range in length from a few hundred base pairs up to 7,000. Examples of SINEs are the *Alu* in primates and the *B1* and *B2* in rodents; examples of LINEs are LINE-1 and THE-1 in primates. There is as yet no evidence that these sequences move from one genomic location to another or that they are associated with detectable phenotypic effects in mammals including the human species, but such evidence is good in bacteria, yeast and Drosophila.

94. Skowronski and Singer [S15] have argued that the dispersed positions of LINE-1 sequences between genes, in introns and in interrupting tandem arrays of species-specific satellites, as well as the target site duplications associated with many LINE-1 sequences, suggest that the sequences were mobile in the past (in an evolutionary sense); further, these authors have compiled a list of five instances of mammalian (dog, rat and mouse) polymorphic alleles that differ by the presence or absence of LINE-1 units. On the basis of all these data (and some unpublished data on the insertion of a LINE-1 unit into a *myc* allele in DNA from a human breast carcinoma), they suggest that the LINE-1 alleles are still capable of being inserted into genomic loci.

95. Concerning the effects of mutagens, including x rays, on the mobility of these transposable elements, the evidence at present is essentially negative, except in yeast: in this species, McClanahan and McEntee [M11] showed that DNA damage induced by UV-irradiation or 4-nitroquinoline-1-oxide stimulates transcription of specific genes that share homology with the *Ty1* (the yeast transposons) element, the inference being that one or more members of the *Ty1* element is (are) regulated transcriptionally by DNA damage. More recently, Morawetz [M12] showed that *Ty1* insertion mutations at the *ADH-2* locus could be increased by UV- or gamma-irradiation or by treatment with ethyl methanesulphonate in a dose-dependent manner, the latter being the strongest agent in this regard.

96. The relevance of the findings in experimental systems—that is, that mobile genetic elements significantly contribute to "spontaneous mutations" and that mutagens have negligible or no effect on the mobility of these elements—to genetic risk evaluation in man has been addressed by Sankaranarayanan [S10, S16]. He argues that (a) if a major proportion of spontaneous mutations in man that lead to disease states is due to the insertion of mobile genetic elements and if the mobility of these is unaltered by radiation exposures, then some of the principal assumptions of the doubling dose method for genetic risk evaluation would lose their validity; (b) currently, however, there is no evidence that documents a significant role for mobile genetic elements in the origin of spontaneous mutations in man; and (c) consequently, there is no need to abandon the use of the doubling dose method for risk evaluation on these grounds.

97. In the realm of DNA repair, one of the principal questions being addressed by current research is the influence of function or activity of a DNA sequence and its repair following mutagen treatment. All these studies have so far been carried out primarily with mammalian cell lines and UV-irradiation. The findings of interest are (a) there is preferential repair of active (transcribing) genes relative to the genome as a whole [B8]; (b) the regions of active repair, in the system studied, correspond to control regions and the 5′ ends of the transcription unit; and (c) in regions away from the 5′ or 3′ ends of the genes, repair is less efficient [S17 and the papers cited therein]. Indirect evidence for preferential repair of active genes has also been presented by Mullenders et al. [M13, M14]. These and other results support the view that ". . . damage in silent regions of the genome may have greater potential for engendering mutations and DNA rearrangements than damage in or near transcription units" [S17]. The generality of these findings and their implications for germ cell mutagenesis with ionizing radiation remain to be established.

C. NATURE OF SPONTANEOUS AND RADIATION-INDUCED MUTATIONS

98. The methods of recombinant DNA technology have enabled the direct detection of the molecular heterogeneity of spontaneously arising mutational events that lead to disease states in man, as will be evident from the spectacular progress in studies of the globin genes (see, for example, [O3]). Similar studies are being carried out to analyse the nature of radiation-induced mutations in mammalian somatic cells (e.g., [V1, T2]). Most of these data have been discussed in the 1986 Report [U7]. An important technical advance has recently been made that allows the cloning and sequencing of small specific DNA segments from total genomic DNA after in vitro amplification of those segments up to 200,000-fold (the so-called polymerase chain reaction, or PCR [S18]). The use of this technique in induced mutagenesis studies will allow mutation spectra to be determined with great precision [V2].

D. FRAGILE SITES AND SPONTANEOUS CHROMOSOME BREAKAGE

99. There are suggestive indications for the thesis that certain autosomal fragile sites may predispose to chromosome breakage. Hecht and Hecht [H1, H2] have adduced some evidence in this regard by analysing information on the location of breakpoints leading to chromosomal anomalies (deletions, duplications, inversions and non-Robertsonian translocations) found in amniocentesis studies and in studies on spontaneous

abortions, still births and new-borns. They caution, however, that in view of the heterogeneity of the data-base from which the pertinent information was extracted, more evidence is needed to determine whether fragile sites are regions in the chromosome predisposed to breakage in the germ cell lineage. The question of whether those fragile sites may predispose the chromosomes to radiation-induced breakage events is as yet unanswered.

E. ONCOGENES: GENETIC ASPECTS

100. The remarkable insights into oncogenic transformation that have emerged in recent years (reviewed in the UNSCEAR 1986 Report [U7]) amply testify to the fact that cancer studies have become an exciting area for both geneticists and cancer biologists. There is now extensive evidence that mammalian genomes harbour nucleotide sequences related to the retroviral oncogenes as part of their normal genetic make-up, that several of them play an active role in the regulation and control of cellular proliferation and that their activation (either spontaneously or through mutagenic exposures) can initiate cancer. There is thus no a priori reason to assume that germ cells will be immune in this respect, especially after exposure to mutagens. Nomura [N3, N5] has reported high tumour yields in the progeny of irradiated mice and thus has highlighted the possibility that genetic changes induced in the germ cells of parents can cause tumours in progeny. It would therefore appear important to confirm these studies independently in experimental systems. Whether this component of genetic risks would be negligible, small or significant for man is difficult to say at present.

F. OTHER WORK IN PROGRESS

101. Data on chromosomal abnormalities studied through direct analyses of the chromosome complement of human spermatozoa (from normal men as well as from those undergoing radiotherapy) after in vitro fertilization of hamster eggs were discussed in the UNSCEAR 1982 and 1986 Reports [U6, U7]. This technique has now been considerably improved [K3] and extended to studies using human spermatozoa irradiated in vitro [K4, M15]. In these studies, which used spermatozoa from five healthy donors, the frequencies of spermatozoa with structural chromosomal aberrations were found to increase linearly with the x-ray dose (0 Gy, 14.1%; 0.25 Gy, 18.9%; 0.5 Gy, 28.5%; 1 Gy, 42.6% and 2 Gy, 68.0%; total numbers of karyotyped spermatozoa: 0 Gy, 2,097; 0.25 Gy, 491; 0.5 Gy, 543; 1 Gy, 819; 2 Gy, 1,009). Most of the x-ray-induced aberrations were breaks and fragments, but a few translocations (0.03-0.1 per cell) were also found; there was no decrease in the fertilization rate of irradiated spermatozoa, even at the highest dose, 2 Gy. Clearly, this kind of study has relevance to the assessment of genetic risks associated with the induction of chromosomal aberrations.

102. Genetic studies on the Hiroshima and Nagasaki populations are continuing and will remain a major source of direct human data. An international collaborative programme on genetic effects in the offspring of cancer patients exposed to physical and chemical agents for therapeutic purposes is being initiated [L2, O2]. This is a commendable enterprise: it is hoped that it will provide information on hazards attributable to genetic damage in human germ cells, and it will undoubtedly have practical importance for clinicians who must advise cancer survivors or current cancer patients who are pregnant or considering pregnancy. Likewise, the follow-up of persons exposed to radiation in the Chernobyl nuclear power plant accident (and their progeny) that is currently underway in the U.S.S.R. is a worthwhile undertaking.

103. The work of Dobson and colleagues over the past several years has suggested that the exceptionally high sensitivity of mouse immature oocytes to killing by radiation is a consequence of the possibility that the target for killing is not the nucleus but the plasma membrane. More recent results by the same group support this idea [S19, S20]. Furthermore, Dobson et al. [D4] have now reported, using monoenergetic 0.43 MeV neutrons and ^{252}Cf (whose recoil protons have subcellular track lengths of such a nature that the radiation energy can be deposited in the DNA in a calculable fraction of the oocytes that survive), that chromosomal aberrations can be recovered from irradiated immature oocytes and that these are similar to those from mature oocytes. A similar effect has also been reported by Griffin et al. [G2].

G. SUGGESTIONS FOR FUTURE RESEARCH

104. In this Annex, the progress that has been made in areas pertinent to the evaluation of genetic radiation hazards in man has been reviewed and estimates of genetic risks have been presented. The Committee feels that, in order to increase precision in risk assessment, more research effort along the following lines will be useful (the order in which these are listed does not reflect the order of importance): (a) molecular analysis of spontaneous and induced mutations in somatic and germ cells with and without repair; (b) further studies relevant to the genetic radiosensitivity of human oocytes; (c) phenotypic expression and transmission of induced deletions and other relevant genetic changes; (d) a search for mammalian models to study human diseases with complex aetiology; (e) tests on the validity of the assumptions and correction factors used in genetic risk estimation; (f) further in-depth analysis of multifactorial diseases with respect to the mutation component; (g) mechanisms underlying non-disjunction and chromosome loss and effects of radiation on these genetic changes; (h) further work bearing on total genetic damage manifesting in the first generation after radiation exposure; (i) relationships between radiation dose, dose-rate and quality and types of induced chromosomal aberrations; (j) development of methods to reveal chromosomal aberrations and gene mutations in human germ cells in the haploid stage; and (k) the use of transgenic mice to study DNA repair deficiencies and mutational mechanisms.

Table 1

Summary of genetic risks estimated
in the BEIR 1972 Report using the doubling dose method

The effects are those estimated for a population continuously exposed
at a rate of 1 rem per generation (low-LET, low dose rate)
in a population of 1 million live births.

Assumed doubling dose range: 20-200 rem

Disease classification	Current incidence per 10^6 live births	Effect of 1 rem per generation	
		First generation	Equilibrium
Dominant diseases	10000	10-100	50- 500
Chromosomal and recessive diseases	10000	Relatively slight	Very slow increase
Congenital anomalies	15000		
Anomalies expressed later	10000	1-100	10-1000
Constitutional and degenerative diseases	15000		
Total	60000	10-200	60-1500

Table 2

Summary of genetic risks estimated
in the UNSCEAR 1977 Report using the doubling dose method

The effects are those estimated for a population continuously exposed
at a rate of 1 rad per generation (low-LET, low dose rate)
in a population of 1 million live births.

Assumed doubling dose: 100 rad

Disease classification a/	Current incidence per 10^6 live births b/	Effect of 1 rad per generation	
		First generation c/	Equilibrium
Autosomal and X-linked diseases	10000	20	100
Recessive diseases	1100	Relatively slight	Very slow increase
Chromosomal diseases	4000 d/	38 e/	40
Congenital anomalies			
Anomalies expressed later	90000 f/	5 g/	45 g/
Constitutional and degenerative diseases			
Total	105100	63	185
Percentage of current incidence		0.06	0.17

a/ Follows that given in the BEIR Report [B1].
b/ Based on the results of the British Columbia Survey with certain modifications.
c/ The first generation increase is assumed to be about one fifth of the equilibrium incidence for autosomal dominant and X-linked diseases; for those included under the heading "congenital anomalies etc." it is one tenth of the equilibrium incidence.
d/ Based on the pooled values estimated from cytogenetic surveys of new-born children; includes mosaics but excludes balanced translocations.
e/ The first generation increase is assumed to include all the numerical anomalies and three fifths of the unbalanced translocations (the remaining two-fifths being derived from a balanced translocation in one parent).
f/ Includes an unknown proportion of numerical (other than Down's syndrome) and structural chromosomal anomalies.
g/ Based on the assumption of a 5% mutational component.

Table 3

Genetic effects of an average population exposure of 1 rem per 30-year generation estimated in the BEIR 1980 Report using the doubling dose method

Assumed doubling dose range: 50-250 rem

Type of genetic disease a/	Current incidence per 10^6 live births	Effect of 1 rem per generation per million live-born offspring	
		First generation b/	Equilibrium c/
Autosomal dominant, X-linked	10000	5-65 d/	40-200
Irregularly inherited	90000		20-900 e/
Recessive	1100	Very few; effects in heterozygotes accounted for in top row	Very slow increase
Chromosomal aberrations f/	6000	Fewer than 10 g/	Increases only slightly

a/ Includes disorders and traits that cause serious handicap at some time during lifetime.
b/ Estimated directly from measured phenotypic damage or from observed cytogenetic effects.
c/ Estimated by the relative mutation risk method.
d/ No first-generation estimate available for X-linked disorders; the expectation is that it would be relatively small.
N.B.: A typographical error in the BEIR Report is corrected here.
e/ Some estimates have been rounded off to dispel an impression of considerable precision.
f/ Includes only aberrations expressed as congenital malformations, resulting from unbalanced segregation products of translocations and from numerical aberration.
g/ Majority of the Sub-Committee feels that it is considerably closer to zero, but one member feels that it could be as much as 20.

Table 4

Cases of serious genetic ill health in offspring (excluding abortions) from parents irradiated with 1 million man-rem in a population of constant size, estimated by the 1980 ICRP Task Group

Category of genetic effect	Equilibrium a/	First generation plus second generation
Unbalanced translocations; risk of malformed live born	30	23 + 6 = 29
Trisomics and XO	30	30 + 0 = 30
Simple dominants and sex-linked mutations	100	20 + 16 = 36
Dominants of incomplete penetrance and multifactorial disease maintained by mutation	160 b/	16 + 14 = 30
Multifactorial disease not maintained by mutation	0	0
Recessive disease	c/	c/
Total	320	89 + 36 = 125

a/ Over all generations following the generation exposed.
b/ The sum of the three entries above (i.e., 30 + 30 + 100).
c/ No estimates given.

Table 5

Effects of 10^{-2} Gy per generation of low-LET, low-dose-rate irradiation in a population of 1 million live-born estimated in the UNSCEAR 1982 Report using the doubling dose method

Assumed doubling dose: 1 Gy

Disease classification a/	Current incidence per million b/	Effect of 10^{-2} Gy per generation	
		First generation c/	Equilibrium
Autosomal dominant and X-linked diseases	10000 d/	15	100
Autosomal recessive diseases	2500 e/	Slight	Slow increase
Chromosomal diseases			
Structural	400 f/	2.4	4
Numerical	3000 g/	Probably very small	Probably very small
Congenital anomalies, anomalies expressed later and constitutional and degenerative diseases	90000 h/	4.5	45 i/
Total	105900	~ 22	~ 150

a/ Follows that given in the 1972 BEIR Report [B1], except that chromosomal diseases are divided into those with a structural and those with a numerical basis.
b/ Based on the results of the British Columbia survey and other studies.
c/ The first generation increment is assumed to be about 15% of the equilibrium incidence for autosomal dominant and X-linked diseases, about three fifths of the equilibrium incidence for structural anomalies and about 10% of the equilibrium incidence for diseases of complex inheritance.
d/ Includes diseases with both early and late onset.
e/ Also includes diseases maintained by heterozygous advantage.
f/ Based on the pooled values from cytogenetic surveys of new-borns but excluding euploid structural rearrangements, Robertsonian translocations and "others" (mainly mosaics).
g/ Excluding mosaics.
h/ Includes an unknown proportion of numerical (other than Down's syndrome) and structural chromosomal anomalies.
i/ Based on the assumption of a 5% mutational component.

Table 6

Genetic risks of low-dose, low-dose-rate, low-LET irradiation estimated in the NUREG 1985 Report

Assumes a 0.01 Gy dose to the population.
Note that the first-generation increases were estimated directly from measured phenotypic damage; the entries given in column "all generations" were derived using the doubling dose method assuming a doubling dose of 1 Gy.

Type of disorder	Normal incidence a/	Risk of 0.01 Gy b/	
		First generation c/	All generations
Single gene	4800		
Autosomal dominant		15 d/	70
X-linked		5	30
Irregularly inherited	43200	d/	70 e/
Chromosome aberrations f/	2880		
Aneuploidy		4	5
Unbalanced translocations		6	10
Total (rounded)	50900	30	185

a/ For a total population of 10^6 persons (16,000 live births/year) for 30 years (480,000 live births).

b/ Cases expected in each generation of children from a population of 10^6 persons, each receiving a dose of 0.01 Gy; assumes 30-year intergenerational interval and birth rate of 16,000 per year per 10^6 persons, or 480,000 children per generation; the integrated risk over all generations following a parental dose of 0.01 Gy is the same as the risk at equilibrium (the column marked "all generations") when the population is exposed at a rate of 0.01 Gy per generation.

c/ Estimated directly from measured phenotypic damage.

d/ First generation increase in irregularly inherited disorders included within that for autosomal dominant disorders.

e/ Based on a doubling dose of 1 Gy and 10 generations mean persistence time, which is very uncertain.

f/ Includes only aberrations expressed as congenital malformations resulting from unbalanced translocations (2,400/480,000) and from aneuploidy (480/480,000); equilibrium time of 1-2 generations and 1 generation, respectively.

Table 7

Risks of genetic disease per 1 million live-births in a population exposed to a genetically significant dose of 0.01 Gy per generation of low-dose-rate, low-dose, low-LET irradiation estimated in the UNSCEAR 1986 Report using to the doubling dose method

Assumed doubling dose: 1 Gy

Disease classification	Current incidence per 10^6 livebirths a/	Effect of 0.01 Gy per generation		
		First generation b/	Second generation c/	All generations (equilibrium) d/
Autosomal dominant and X-linked diseases	10000	15	13	100
Autosomal recessive diseases	2500			
- Homozygous effects		No increase	No increase	11 e/
- Partnership effects		Negligible	Negligible	4 f/
Chromosomal diseases due to structural anomalies	400	2.4	1	4
Subtotal (rounded)	13000	18	14	115
Early acting dominants g/	Unknown	]		
Congenital anomalies h/	60000	]		
Other multifactorial diseases i/	600000	]	Not estimated	
Heritable tumours j/	Unknown	]		
Chromosomal diseases due to numerical anomalies k/	3400	]		

a/ Based on the results of the British Columbia Study and other studies; for details see [U5].

b/ The first-generation increment is assumed to be 15% of that at equilibrium for autosomal dominant and X-linked diseases and three fifths of that at equilibrium for chromosomal diseases due to structural anomalies.

c/ Not given in the UNSCEAR 1986 Report and estimated here; for autosomal dominants and X-linked diseases, it is calculated as 15% of (equilibrium increase minus the increase in the first generation); a similar procedure applies to chromosomal diseases due to structural anomalies.

d/ These values apply if 0.01 Gy is given in each generation, but they also express the total genetic damage over all generations if the dose of 0.01 Gy is given in one generation.

e/ Frequency of recessives maintained by mutation assumed to be 1100 per 10^6 livebirths.

f/ From partnership between induced mutations and those already present in the population, assuming 2.5% heterozygous disadvantage and a mean number of harmful recessives per gamete of 1 [S9].

g/ The incidence of these in human populations is unknown because they act too early to be recognized as transmissible dominants; they include dominant sub-lethals, the rate for which has been estimated to be 5-10 per 0.01 Gy of paternal irradiation of mice (see Table 23 of the UNSCEAR 1986 Report [U7]).

h/ Studies by Lyon and Nomura and colleagues show that, in the mouse, they are induced by irradiation of male and female germ cells, but the associated risks appear to be low.

i/ The prevalence is much higher than that given in previous UNSCEAR Reports, because diseases manifest up to age 70, instead of mainly to age 21, have now been included, together with some less serious conditions. Furthermore, the figure denotes the number of diseases per 10^6 individuals and not the number of affected individuals. There is considerable uncertainty over whether a doubling dose of 1 Gy and a mutational component of 5% (as used previously) can be justified. The UNSCEAR 1982 Report arrived at estimates of 4.5 extra cases per 1 million in the first generation and 45 per million at equilibrium after a parental dose of 0.01 Gy, on the basis of the previous estimate of population prevalence of 90,000 per million.

j/ Nomura has reported the induction of pulmonary and other tumours in the F_1 generation of mice after irradiation (see [U7] for details), but these have very low expressivity; their likely effects on health are thus unclear.

k/ It is still not clear whether germinal irradiation leads to significantly increased frequencies of non-disjunction, but any resultant genetic risk from the production of trisomic conditions is thought to be low.

Table 8

Summary of genetic risks estimated
in the UNSCEAR 1972 Report using the direct method

The estimates are per rad of low-LET, low-dose-rate radiation exposure.

End point	Expected rate of induction per million		Expression in F_1 per million conceptions after spermatogonial irradiation
	Spermatogonia	Oocytes	
Recessive point mutations	1500 a/	Very low	30-75
	(36) b/	-	(1-2)
Dominant visibles	2	-	2
Skeletal mutations	4	-	c/
Reciprocal translocations d/	15 e/	Very low	2 congenitally malformed children, 19 unrecognized early embryonic losses, 9 recognized abortions f/
X-chromosome losses	Very low	8	8 early embryonic and/or abortions
Other chromosome anomalies	Very low	-	Very low
Total genetic damage	1521 g/		
	(57) h/		
Total genetic damage' i/	300 i/		6-15 i/

Note: Dashes indicate that inadequate or no information is available.

a/ Estimate based on mouse specific locus data.

b/ Estimate based on the per genome rate for recessive lethals induced in mouse spermatogonia.

c/ Included under recessive point mutations.

d/ Figures apply to low-dose x-irradiation. Estimates for chronic gamma-irradiation are 50% lower.

e/ Balanced products.

f/ For low-dose x-irradiation; for chronic gamma-irradiation, figures should be halved.

g/ Obtained by adding 1500+2+4+15 in the column.

h/ Obtained by adding 36+2+4+15 in the column.

i/ Relative to spontaneous incidence of genetic diseases among live born, based on an estimated "doubling dose" of 100 rad.

j/ In terms of incidence of genetic disease among live born (doubling dose method).

Table 9

Summary of genetic risks estimated in the UNSCEAR 1977 Report using the direct method

The estimates are per rad of low-LET, low-dose-rate radiation exposure.

End point	Expected rate of induction per million		Expression in F_1 per million conceptions after spermatogonial irradiation
	Spermatogonia	Oocytes	
1. Autosomal mutations a/	60	-	20 d/
2. Dominant visibles b/	Very low	-	
3. Skeletal mutations c/	4	-	
4. Balanced reciprocal translocations e/	17- 87	Low	Low f/
5. Unbalanced products of end-point 4 above	34-174	-	2-10 g/
6. X-chromosome loss h/	Very low	Low	Low
7. Other chromosome anomalies	-	-	-

Note: Dashes indicate that inadequate or no information is available.

a/ Presumed to include small deficiencies. Based on rate of induction of mutations in mice that are lethal in the homozygous condition, which is doubled to give the overall rate.

b/ Based on those scored in the course of specific-locus experiments in mice.

c/ Detected in mice by dominant effects.

d/ Overall rate of dominant effects, based on skeletal mutations and presumably including dominant visibles and heterozygous effects of autosomal mutations.

e/ Derived from human and marmoset cytogenetic data under the assumption that the frequency of translocations in the F_1 progeny is one fourth of that observed in spermatocytes.

f/ Effects such as those given for end-point 5 in the next footnote will become manifest in generations following the first.

g/ Expressed as congenital malformations. In addition, there would be 11-55 recognized abortions and 22-109 early embryonic losses.

h/ Detected in mice by X-chromosomal markers.

Table 10

Summary of genetic risks estimated
in the UNSCEAR 1982 Report using the direct method

The estimates are per 10^{-2} Gy of low-LET, low-dose-rate radiation exposure.

Risk associated with	Expected frequency (per 10^6) of genetically abnormal children in the first generation after irradiation of	
	Males	Females
Induced mutations having dominant effects a/	~10 to ~20 b/	0 to ~9 c/
Unbalanced products of induced reciprocal translocations	~0.3 to ~10 d/	0 to ~3 e/

a/ Includes the risk from the induction of dominant mutations, as well as of recessive mutations, deletions and balanced translocations with dominant effects.

b/ The lower limit of ~10 is derived from data on cataract mutations and the upper limit of ~20 per 10^6 is derived from data on skeletal mutations and is the same as the one arrived at in the 1977 report. A multiplication factor of 2 has been used in the skeletal estimate but not in the cataract one; this factor is an attempt to allow for the likelihood that many dominant mutations (especially those affecting systems other than the skeleton) remain to be detected. A correction factor of 0.5 to allow for skeletal mutations which are not clinically significant is not required for the cataract estimate.

c/ The lower limit of zero is based on the assumption that the mutational sensitivity of human immature oocytes is similar to that of mouse immature oocytes; the upper limit of 9 per 10^6 is based on the assumption that the sensitivity of the human oocytes is similar to that of the mature and maturing oocytes and that the latter is 0.44 times that of spermatogonia. See text for further details.

d/ The lower limit of ~0.3 per 10^6 is based on rhesus monkey cytogenetic data; the upper limit of ~10 per 10^6 is based on combined marmoset and human cytogenetic data.

e/ The lower limit of zero is based on the assumption that the sensitivity of the human immature oocytes to the induction of heritable reciprocal translocations will be similar to that of the mouse immature oocytes with respect to the induction of chromosome aberration phenomena; the upper limit of ~3 per 10^6 is based on the assumptions that the sensitivity of the human immature oocytes to the induction of translocations will be one half that of the human and marmoset spermatogonia (based on results with mice on heritable translocations), that the frequency of unbalanced products will be six times that of recoverable balanced reciprocal translocations and that 6% of the unbalanced products will result in congenitally malformed children.

Table II

Risks of induction of genetic damage in man per 10^{-2} Gy
at low dose rates of low-LET radiation
estimated in the UNSCEAR 1986 Report using the direct method

Risk associated with	Expected frequency (per 10^6) of genetically abnormal children in the first generation after irradiation of	
	Males	Females
Induced mutations having dominant effects a/ b/	~ 10 to ~ 20 c/	0 to ~9 d/
Induced recessive mutations e/	0	0
Unbalanced products of induced reciprocal translocations	~ 1 to ~ 15 f/	0 to 5 g/

Note: These estimates are the same as those made in the UNSCEAR 1982 Report except for changes indicated in footnotes b/ and e/.

a/ Includes risk from the induction of dominant mutations, as well as of deletions and balanced reciprocal translocations with dominant effects.

b/ Does not include the risk of mortality (between birth and early life) estimated on the basis of data on litter size reduction in mice (about 5-10 cases per million conceptions); see text for details.

c/ The lower limit is derived from the data on cataract mutations and the upper limit from those on skeletal mutations (both in mice); the latter is the same as that arrived at in the UNSCEAR 1977 report [U5]. A multiplication factor of 2 has been used in the skeletal estimate, but not in the cataract one. This factor is an attempt to allow for the likelihood that many dominant mutations (especially those affecting bodily systems other than the skeleton) remain to be detected. A correction factor of 0.5, which allows for skeletal mutations that are not clinically significant, is not required for the cataract estimate. See UNSCEAR 1982 Report [U6] for details.

d/ The lower limit of zero is based on the assumption that the mutational sensitivity of human immature oocytes is similar to that of mouse immature oocytes; the upper limit of ~9 is based on the assumption that the sensitivity of the human oocytes is similar to that of mature and maturing mouse oocytes and that the latter is 0.44 times that of spermatogonia. See UNSCEAR 1982 Report for details.

e/ Although the risk (of recessive disease from the induction of recessive mutations) is zero in the first generation, about 1 extra case per million live births would be expected in the following 10 generations (from partnership effects) and on certain assumptions, about 10 extra cases per 1 million would be expected by the tenth generation (from effects due to identity by descent). See text for further details.

f/ The lower limit is based on combined cytogenetic data from chronic low-LET irradiation experiments involving the rhesus monkey and the crab-eating monkey, and the upper limit, on the combined human and marmoset (Saguinus fuscicollis) cytogenetic data (see UNSCEAR 1986 Report for details). It has been assumed that 9% of unbalanced products of reciprocal translocations will result in birth of congenitally abnormal children.

g/ The lower limit of zero is based on the assumption that the sensitivity of the human immature oocyte to the induction of heritable reciprocal translocations will be similar to that of mouse immature oocytes with respect to the induction of chromosome aberration phenomena; the upper limit is based on the assumptions that (a) the sensitivity of the human immature oocytes to the induction of reciprocal translocations will be one half that of the human and marmoset spermatogonia (based on results with mice on heritable translocations); (b) the frequency of unbalanced products will be six times that of recoverable balanced reciprocal translocations; and (c) about 9% of unbalanced products will result in congenitally malformed children. See UNSCEAR 1982 Report for details.

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U6 United Nations. Ionizing Radiation: Sources and Biological Effects. United Nations Scientific Committee on the Effects of Atomic Radiation, 1982 Report to the General Assembly, with annexes. United Nations sales publication E.82.IX.8. United Nations, New York, 1982.

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back to first page

ANNEX F

Radiation carcinogenesis in man

CONTENTS

Introduction

1. Carcinogenesis is the main late-stage somatic effect of ionizing radiation. The malignancies induced by irradiations are indistinguishable from those occurring from many other causes. Therefore, the observations of these effects in man have been limited to study populations who were more highly exposed in various ways and for various reasons. These results form the primary basis for assessments by UNSCEAR of the risks of ionizing radiation.

2. Previous UNSCEAR Reports on radiation carcinogenesis in man [U2, U3] have adopted a site-specific approach, focusing on cancers of the breast, lung, thyroid and on leukaemia. This was inherent in the basic descriptive epidemiological results available. There has always been a desire to interpret results from the standpoint of an understanding of the mechanisms of carcinogenesis. While there have been some developments, particularly in the field of molecular biology, that are relevant and are reviewed here, the updated and revised epidemiological results continue to serve as the primary basis for the risk assessment. This Annex, however, attempts a more analytical approach to risk evaluation, especially for projection of risks beyond observational experience.

3. It is particularly propitious that risk assessments be made now, in light of the revised dosimetric evaluations of the Japanese survivors of the atomic bombings, the most important study population. This re-evaluation, completed in 1986 and known as the dosimetry system 1986 (DS86), replaces the previous estimates of 1965 (T65). Not all of the revisions could be taken into account in analysing cancer incidence frequencies, but the latest values are used in the Committee's analytical evaluation of risk coefficients for mortality.

4. Of all of the factors that affect the carcinogenesis process, age at exposure is particularly significant. Susceptibility of tissues to radiation effects is related to proliferative activity of cells, and periods of active growth and development can be expected to be periods of greater sensitivity. There are difficulties, however, in accounting for age differences. Many study populations involve individuals of particular predispositions exposed under special circumstances. Data from individuals who were youngest at the time of the atomic bombings are incomplete, as they are just now entering the important stage in their late adult lives when cancer prevalence increases.

5. There are many reasons for uncertainties in the risk assessments. A main concern that cannot be adequately resolved is how to relate the results obtained at high doses and dose rates to the low levels of exposure that may be expected in environmental and routine occupational settings. The difficulties and developments in the field of radiation carcinogenesis are discussed in this Annex. The risk evaluations provided by the Committee reflect the current best knowledge and approaches and are put forward to serve as background information for consideration of international and national bodies of radiation protection.

I. GENERAL CONSIDERATIONS

A. TERMS, APPROACHES AND CONCEPTS

1. Terms and units of radiation exposure

6. A variety of units are or have been used for the quantities needed in assessments of exposures to ionizing radiation. However, it having been decided to adopt the modern metric system, the Système International (SI); throughout this Annex, SI units will be used wherever practicable. A detailed account of the correspondence between the older units and the SI units is found in [N3]. For the reader unfamiliar with these units, it is sufficient to note that 1 rad, the old unit for absorbed dose, equals 0.01 gray (Gy); 1 rem, the old unit for dose equivalent, equals 0.01 sievert (Sv); and, finally, the curie (Ci), the old unit for activity, equals $3.7\ 10^{10}$ becquerel (Bq).

7. The subjective expressions "low", "intermediate", "high" and "very or ultra-high" as applied to absorbed doses of sparsely ionizing radiation are arbitrarily defined here, following the convention established in the UNSCEAR 1986 Report [U1], as 0-0.2, 0.2-2.0, 2.0-10.0, and above 10 Gy, respectively. For other

types of radiation the corresponding dose equivalent ranges are the same, but with units of sievert. This correspondence does not necessarily reflect RBE, but rather assigned quality factor values. Following the same convention, "low" dose rates for all radiations are those below 0.05 mSv/min; "high" dose rates are those above 0.05 Sv/min; and "intermediate" dose rates fall between the two figures quoted.

8. Exposure of the lung to alpha particles from radon daughter products is customarily expressed in terms of working level (WL) or working level months (WLM). The WL is any combination of radon and its daughter products in a litre of air that will result in the emission of 1.3 10^5 MeV of potential alpha energy; the WLM is the exposure resulting from the inhalation of air with a concentration of 1 WL of radon daughters for 170 (working) hours.

2. Concepts of risk

9. Conventionally, the risk associated with exposure to radiation, either sparsely or densely ionizing, has been expressed in absolute or relative terms. Absolute risk is usually taken to mean the absolute increase in the frequency of cancer, mutation, or the like, and the absolute risk coefficient is the increase per unit exposure per unit time of risk after adjustment for confounding variables. Commonly, it is defined as the excess deaths or incident cases per gray (or rad) per ten thousand (or million) person-years and is estimated by regressing the difference between the observed number of events and the expected number, based on a suitable comparison group or population, as a function of dose (see, e.g., [K7] or [W5]). Relative risk is the ratio of the number of cases observed in an exposed population to the number of cases expected in a non-exposed, but otherwise comparable, population.

(a) Dose-response pattern

10. One objective of risk assessment is to estimate the relationship between the dose administered and the response elicited, specific to exposed individuals of differing ages, sex, or other biological characteristics. This relationship is called the dose-response relationship, and is an expression of the form

$$r = f(D)$$

where r is a defined measure of response, e.g., a risk quantity, usually an excess absolute or excess relative risk, and f(D) is a function of absorbed dose. The function f(D) commonly takes one of the following forms: (a) linear, i.e., $a + bD$; (b) linear, non-threshold, better called proportional, i.e., bD; (c) quadratic, i.e., $a + bD + cD^2$; (d) pure quadratic, i.e., cD^2; (e) proper order polynomials. In addition, any of these forms may be modified by multiplying by an exponential term with the exponent containing negative terms in D and D^2. A quadratic term in the dose-response function is usually included when the effects at low doses are less than would be predicted by a strictly linear response to dose at intermediate doses. For dose-response relationships going through a maximum at intermediate doses, it is assumed that cells exposed to high doses may be prevented from dividing by the sterilizing (or cell-killing or inactivation) effects of irradiation. These matters have been thoroughly discussed in Annex B of the UNSCEAR 1986 Report.

11. The age, sex and other characteristics of the exposed individual may or may not be specified in assessing the dose-response pattern. Similarly, the age, the time since exposure and the end-point used to assess the response may vary from one application to another. In practice, all data from the time of exposure to the time of analysis are often pooled, and the cumulative excess risk in the exposed population, commonly expressed as excess deaths or incident cases, is related to the dose received. However, in principle, there may be significant differences in the dose-response relationships between different groups of people, e.g., between exposed children and adults. These might be reflected as host-factor effects on the shape of the dose-response function.

(b) Risk measures and projection

12. A second objective of assessing risk is to follow individuals after exposure has begun, or after it is complete, and to quantify the excess cancers during the lifetime of groups of such individuals. Since the individuals may vary in other respects than simply their ages, different assessments may be made for each sex, age at exposure, exposure to other risk factors and so on. It may also be important to be able to predict future risk in exposed cohorts, even to ages beyond those for which current estimates exist.

13. In this Annex, the assessment of events after exposure and beyond the period of observation will be referred to as the projection of risk. When the risk in the exposed individuals exceeds the spontaneous (non-exposed) risk level by the same amount at all ages, the effect of exposure will be termed additive (often the term absolute has been used in this context, since at all ages the excess risk is constant). When the risk to the exposed is some constant fraction greater than the spontaneous risk, this will be termed a multiplicative effect (often the term relative has been used, in the sense that at all ages after exposure the relative risk, or risk ratio, is constant). Actual effects may fit neither of these simplified models, or they may fit some combination of them.

14. When the risk experiences of several age (or other) strata are combined into a single summary measure, one must be aware of the nature of the exposure experiences over which the single value is computed. If no method is specified in the naming of such a measure, then no assumptions can be made about the way in which aggregate data were used to compute the expected risk. When the measures are single values derived from the cumulative experience of an exposed cohort, this will be referred to as the total relative or absolute risk.

15. The excess risk is usually computed from the risk in a comparable group, either the population at large or a control group. When using an unexposed control group matched by age and sex, the number of cases in

that control group is considered to be the baseline number. Often, however, the control group is the entire population of which the exposed are a part. The risk schedule in that population, say $r(x)$, is the annual incidence (or mortality) per person at age x per year; this can also be made specific to sex and historical time period. In a cohort of exposed persons, over the course of the investigation, a certain number of person-years of experience at age x will arise: this is the sum of all years spent by exposed cohort members at age x. Let this number be $c(x)$. Then the expected number of cancers in the cohort, E, can be approximated by

$$E = \sum_x c(x)r(x)$$

and the excess number of cases is the observed number, O, minus the expected number, O-E. The number of person-year-grays (PYGy) of exposure to which this excess applies is the sum over all exposed individuals of the product of the dose to the i-th individual, d_i, times the number of years that individual was followed in the study (up to the point of manifesting the cancer of interest) y_i. That is, summing over the n individuals in the cohort,

$$N = \sum_i d_i y_i$$

The desired summary measure is then (O-E)/N excess cases per 10^4 PYGy. Note that this measure aggregates the experience of different ages and that it is possible to obtain the same value for N by including exposed cohorts of different age distributions, followed for differing lengths of time, to a different distribution of doses. Life-table methods can make the computation of expected numbers of cases somewhat more precise by accounting for the fraction of the exposed cohort expected to die from causes of mortality other than the cancer of interest.

16. The relative risk is usually computed simply as O/E, again aggregating the experience of individuals at different doses and of different ages. When the irradiated population is actually a subset of the comparison population under scrutiny, this ratio is not strictly the relative risk of exposure compared to non-exposure, and in this case 100 times (O/E), should be referred to as the standardized mortality (or morbidity) ratio (SMR); however, because of its nearly universal use in radiation epidemiology, the term relative risk (RR) will be used, in general, for the ratio of the observed to the comparison group.

17. In addition to depending on the details of the age pattern of cases since exposure, relative to age at exposure and dose, both of these measures also depend on the background, or baseline, risk in the population [$r(x)$], so that it may be difficult to apply excess deaths (cases) or relative risk estimates to populations epidemiologically different from the exposed population.

18. In the latest reports on the survivors of Hiroshima and Nagasaki [P15, S48, S49], excess relative risk, at 1 Gy of exposure, has been computed. This is the excess relative risk value (i.e., RR-1) over all age, sex and other strata in the data. This measure combines aspects of the other measures, but is still a single, cumulative measure of effect. Relative or absolute risks can always be calculated in this manner. For example, they can be computed after any number of years of observation or by pooling individuals with different ages at exposure or pooling both sexes. Such measures are dependent on these pooling or aggregating procedures and may not be comparable between studies. Computation of an absolute risk in this way does not imply that the projection effect (on post-exposure age-specific risks) is additive, nor does computing relative risk mean that the projection effect is itself multiplicative.

19. For these reasons, it is necessary to distinguish between the projection effect (additive or multiplicative) and the measure of risk based on observational data (absolute risk, that is, excess deaths or cases, relative risk, attributable risk [see paragraph 22]). This distinction is not always clear in the literature, where the same terms are often used in both circumstances. To make it quite clear, when projection effects are referred to in this Annex, the terms multiplicative or additive will be used; when measures in data are referred to, the terms absolute or relative risk will be used.

3. Assignment of causation

20. An increasingly frequent question addressed to radiation biologists is whether a specific cancer could have been radiation-induced. This question may arise in litigation, in the adjudication of occupational compensation or in the legislative process or it may arise through simple curiosity. Rarely, if ever, is it possible to state categorically that a specific cancer was or was not due to radiation exposure. At present, there is typically no radiation-specific tumour pathobiology, and radiation-related tumours appear similar to spontaneous tumours at the same site. Inasmuch as some tumours that are extremely rare in the general population are much more common after radiation, it may be that radiation alone is able to produce a tumour. In general, however, radiation exposure only increases the frequency of an already prevalent tumour.

21. Despite these conceptual problems, it is possible to consider causation in a probabilistic manner [B17]. Briefly, if an outcome, A, can occur only when one of a series of exhaustive and mutually exclusive events occurs, and if the a priori probabilities of these latter events are known, then it is possible to compute the conditional probability that A is due to a specific one of the series of events. This latter, conditional probability is sometimes described as the a posteriori probability of the event. In the present context, the probability of causation, P, or, more formally (given the occurrence of a cancer in an exposed individual), the probability that it is due to radiation (one of a series of presumably mutually exclusive causes of cancer) is simply the ratio of the additional risk imposed by the radiation dose D to the total risk; the latter is, of course, the sum of the baseline risk and the radiation risk. It can be written as

$$P = [D \times R]/[B + (D \times R)]$$

where R is the absolute annual site-specific risk per gray and B is the baseline cancer rate, specific for site, age, sex and other pertinent concomitants.

22. A related, commonly employed epidemiological notion is that of attributable risk. The latter is the proportion of a health disorder that can be attributed to a causal factor. Epidemiologists use a variety of mathematical definitions of attributable risk. One is derived by subtracting the incidence of the disorder among persons not exposed to the factor, e.g., ionizing radiation, from the incidence among persons who were exposed. Thus, in the present context, it is merely

$$\text{Attributable risk} = \frac{\text{observed} - \text{expected cases}}{\text{person/years at risk}}$$

4. The analytic approach to radiation carcinogenesis

23. There are a number of factors that influence the risk of cancer in individuals exposed to radiation. These include the nature of the individual receiving the dose, often known as the host (genetic background, general health, specific health problems, sex etc.); the nature of the dose received (high or low, acute or chronic, radiation quality); other factors that may interact with radiation or affect the susceptibility of the host (e.g., smoking, diet, weight, exposures to chemicals, other diseases and medical treatments); and the nature of the carcinogenic process itself. Because of the existence of these factors, there is no single way in which effects should be assessed. Actually, several approaches have been taken.

24. The first is to examine the relationship between biological models of carcinogenesis and the effect of different exposures or host conditions. There have been many different attempts to model radiation carcinogenesis. Generally, the parameters of the process are estimated using epidemiological data on exposed individuals to infer the action of radiation and to estimate the relative importance of different components of the process. Those components usually include mutation, mutation-repair, growth stimulation and cell sterilization. Specifically, various multi-stage processes have been applied to the age-onset distribution of cancers following irradiation in an attempt to determine the consistency of such processes with the action of ionizing radiation and to infer which part of the process is affected by exposure. These investigations do not provide risk estimates for a given dose, but they do suggest aspects of risk, such as vulnerable ages, and whether the effects of exposure can be expected to be long lasting or not.

25. Another approach is to analyse dose-response and risk-projection relationships. Much effort has been devoted to assess the response of various tissues to differing exposure levels or durations, and numerous studies have sought to estimate the cancer risk at various dose levels. Interest has been particularly great in extrapolating risks from high-dose data to low doses and from high dose rates to low ones. It is assumed that most controllable exposures, such as occupational ones, will involve much lower doses (and generally lower dose rates) than those experienced by the studied populations. One goal has been to provide risk estimates so that individual exposures, such as those experienced for medical or occupational reasons, can be controlled in an informed way. Another goal has been to use the limited epidemiological data to infer the risks that attend other conditions of exposure.

26. A continuing objective has been to determine, for given exposures and tissue sites, whether there is a linear or non-linear (specifically, quadratic) dose-response relationship at low doses. This issue has been studied, without resolution, in animals and in man, for decades. There is still no unambiguous answer, but in many cases newer data have contributed to the ability to infer the existence or non-existence of risk thresholds or the likely forms of the dose-response relationships, a subject treated at length in Annex B of the UNSCEAR 1986 Report [U1].

27. A different approach to the dose-response relationship has also been taken [F6]. Species-specific parameters for a theoretical model of cancer have been fitted to experimental data and used to estimate low-dose response rates. While the theory seems to work for cellular effects in vitro, it is not obvious that it applies in vivo, where species-, individual- and tissue-specificities, as well as differences in the nature of the radiation action, are not controllable and often cannot be measured accurately. However, a theoretical approach is necessary when actual observational data do not exist.

28. Finally, there have been direct regression approaches to risk assessments. Recently, multiple regression theory has been extended to the evaluation of risk factors in a complex disease (exposure) phenomenon. This has been very useful in chronic disease epidemiology and, given the nature of the data, seems appropriate to assessing the risk of radiation carcinogenesis. Several applications of these methods, generally those called proportional hazards models (see, e.g., [K12]), have been made in this context and will be reviewed.

29. Since the carcinogenic effects of very low doses cannot be shown directly with existing data (see, for example, [U5]) nor reliably extrapolated from high-dose data (for a discussion of some of the problems associated with extrapolation, see [D25]), the shape of dose-response relationships at low doses of ionizing radiation remains conjectural. An understanding of these relationships will involve a knowledge of carcinogenesis as a biological process and the relation of radiation to this process (e.g., [F5, F6]).

B. SOURCES OF DATA

30. Data on radiation carcinogenesis in man can be derived from a limited number of sources. The main types of human exposures that have occurred and the study populations available are listed in Table 1. These include the survivors of the atomic bombings of Hiroshima and Nagasaki; observers of nuclear tests and those exposed to fallout; patients irradiated therapeutically to treat cancers or other disease conditions; workers in nuclear installations, miners and radiologists; individuals exposed at home to elevated levels of background radiation; and indivi-

duals involved in nuclear accidents. The exposure conditions have included single, multiple and chronic irradiation from external and internal sources. The main categories of exposure are reviewed below.

1. Special exposed cohorts

31. Much of the knowledge about radiation carcinogenesis still comes from the study of the Japanese populations exposed during the atomic bombings at Hiroshima and Nagasaki and from data on Marshall Islanders exposed to substantial doses of fallout during the testing of nuclear weapons. The accident at the Chernobyl nuclear reactor in the USSR has also exposed a sizeable number of people to non-trivial doses, and their experience may become a valuable source of information. These exposures have contributed importantly, and will undoubtedly continue to do so, to the limited data on the lifetime risks, with regard to all cancer sites and all ages and doses, associated with acute radiation, to an ostensibly healthy and unselected population.

2. Patients treated with therapeutic radiation

32. Numerous cohorts of individuals have been identified to whom substantial doses of radiation were administered for various therapeutic purposes. The doses were typically given over a short period of time and usually administered locally. Individuals in these groups were exposed, for example, to radiotherapy for cancer or for ankylosing spondylitis or to radiation to the thymus and thyroid for various reasons, to the head for tinea capitis or various parts of the body for haemangiomas, to treat Hodgkin's disease or to suppress the immune system to prevent the rejection of tissue transplants. Although many of these exposures no longer occur, other forms of radiation therapy continue to increase. The Patterns of Care Study sponsored by the American College of Radiology has shown that in the United States over the 10 years from the first of its surveys, in 1973, to the fifth, in 1982, the number of new radiation therapy patients per thousand population per year has grown over 17%, from 1.46 to 1.71, or from 304,020 to 401,263 individuals [K10]. The improved survival of many cancer patients means that there will be an increasing amount of data on the risk of second cancers among these people, especially those treated in childhood. Such risks will have an impact on the management of selected diseases and thereby on the structure and process of clinical care and outcome [D6, H3, H4, K9]. It is worth noting that the beams in the newer radiological techniques are more sharply edged than in the older techniques, and exposures can be more readily restricted to the tumour itself.

3. Individuals receiving diagnostic examinations

33. Millions of individuals have been exposed to low doses of radiation for the diagnosis of a great variety of conditions or for the monitoring of treatment. With the continued improvement of roentgenographic and other diagnostic techniques and equipment, the doses per examination are being reduced and the exposures becoming more focused. The availability of ultrasound and magnetic resonance imaging techniques is also diminishing the frequency with which ionizing radiation is used. However, the development of computerized tomographic methods, including positron emission tomography, and the various uses of radioisotopes for diagnostic purposes may have increased the doses received and frequency of irradiations. While the individuals exposed to moderate doses may be few, diagnostic radiation, in general, is likely to continue to be an important source of exposure. Since these exposures usually occur where serious disease is present, risk-benefit analysis will be particularly valuable in justifying and optimizing them (see Annex C, "Exposures from medical uses of radiation").

4. Occupational groups

34. Several earlier groups of people have been exposed to substantial doses of ionizing radiation over long periods of time, commonly because the dangers of such exposure were unrecognized. These groups include radium dial painters, radiologists, radiology technicians and industrial radiographers, all of whom were exposed earlier in this century, and miners who worked for many years in environments with high levels of radon. The exposures have generally been in the intermediate-to-high (0.2-10 Gy) dose range.

5. Populations receiving chronic exposures

35. Many individuals are chronically exposed to low doses (below 0.2 Gy) of radiation, but few have been studied as well-defined cohorts. Commonly they have been exposed because of the place in which they live, or because of their occupations, or because of various medical or other exposures (e.g., those that involve radioisotopes with long half-lives), or because of radioactive fallout. In many countries, radon in homes may be the largest single source of chronic exposure to low doses of ionizing radiation that the average person confronts [C13]. Populations in all countries are exposed, but the distributions of individual exposures are largely unknown. Instances have been recorded of persons residing in houses where the exposure to radon amounted to as much as 30 WLM, that is, almost eight times the limit set for uranium miners. These persons are usually unaware of the risk they face, for their homes are not necessarily built on or in the vicinity of mine tailings, but rather on rock containing high levels of natural radioactive substances. Water sources may also contain radon that is released to air in the houses. While inhaled radon is not chemically bound in body tissues nor is its solubility in tissues high, the simultaneously inhaled radon daughter products are deposited in the respiratory tract, and these, notably the shorter-lived ones, decay, exposing the bronchial epithelium. This is discussed in Annex A, "Exposures from natural sources of radiation". As yet, there have been few epidemiological studies of populations exposed in

their homes to radon and its daughter products that enable reliable quantification of the risk of cancer of the bronchial tree.

6. Accident victims

36. There will continue to be isolated cases of high exposures due to accidents of various sorts. Heretofore, these cases have provided little insight into the long-term consequences of exposure to ionizing radiation, but with the establishment of a world-wide Registry of Radiation Accidents at Oak Ridge in the United States, they may become more informative [D7, F4]. At present the Registry lists more than 230 accidents; by far the largest number were the result of either a mishandling of industrial, sealed radioisotope sources or an inadvertent exposure to x rays used for quality control [S25]. Often the accidents involved unsuspecting individuals, not a few of them children, who picked up a metal object and carried it home, where they and other household members became exposed, unknowingly, to the radiation emitted by what was a metal-encapsulated source. No fewer than 1,100 individuals are enumerated, 38 of whom died, presumably as a direct consequence of their exposure; about half of those who died received significant exposures of 0.25 Sv or more to the whole body or of 6 Sv locally to the skin, or of 0.75 Sv or more to a critical organ. The accident at Ciudad Juarez in Mexico is typical of the accidents that involved general populations. There, a cobalt-60 source, improperly disposed of, resulted in the exposure of 300-500 individuals, some of whom received doses of 0.5-1.0 Gy. A similar but more recent accident at Goiania, Brazil, involved some 244 individuals, 54 of whom were subsequently hospitalized and 4 of whom have died; exposure in that instance was to ^{137}Cs. Accidents have also occurred when fuel rods were being inserted or removed from reactors [see, e.g., W7, W8, W9]. In these latter instances, the subsequent health experience of the exposed individuals is generally being carefully scrutinized by the employing laboratories or utility companies.

37. In the review of radiation carcinogenesis that follows, data from all these sources will be considered. Of necessity, this review will overlap in some particulars with material contained in other UNSCEAR documents, specifically in Annexes A and B of the UNSCEAR 1986 Report [U1]. The reader should consult these for further details and for aspects of radiobiology that do not apply directly to human radiation carcinogenesis. Thus, for example, no effort will be made here to review the data on radiation-induced cancers in experimental animals.

C. TYPES OF STUDIES

38. Of the ways in which radiation carcinogenesis may be studied, two have predominated: (a) cohort studies, in which individuals exposed in some special way and individuals not exposed are compared. The follow-up can be retrospective or prospective, or both; and (b) case-control studies, in which cancer cases and matched normal individuals are ascertained and their prior exposure histories compared. These types of investigations on available population groups are outlined in Table 2.

1. Cohort studies

(a) Ongoing investigations

39. Most of the investigations in progress in 1977, when UNSCEAR last reported on human radiation carcinogenesis, have continued, and additional results have since been reported. Moreover, the investigations concluded prior to the last Report have received fresh scrutiny and much of the older data has been subjected to further analysis and interpretation. In several instances, the early findings have been improved and joint estimates of dose-response patterns made more precise by combining data from several investigations (see, e.g., [D11] or [T16]). In other instances, previous results have been called into question because, for example, doubts had been cast upon the dose estimates or ascertainment biases or because better, later studies had yielded conflicting observations.

40. There are further data on women who received chest fluoroscopy (to monitor pneumothorax), irradiation for mastitis, or other exposures to the breast in connection with breast cancer [H6]. These results show (a) a higher susceptibility of the young; (b) dose fractionation does not reduce the risk of breast cancer in all studies; and (c) there is uncertainty as to the level of risk at low dose rates. Similarly, much has been added to the literature on the effects of exposing the thyroid to various kinds of irradiation in childhood [R1, S13]. Quantitative estimates of dose-effects have been improved; however, the pattern of radiation-induced thyroid cancer has been extensively reviewed, leading to the suggestion that, while radiation does have an effect at moderate doses, the clinical significance of this in terms of active thyroid cancer, rather than subclinical thyroid changes, is not patent [C6, P4].

41. Studies of cancer following pelvic irradiation for malignancies of the cervix have not discovered the excess of leukaemia that had been expected [B12, B13, W6], although cancers have appeared at other exposed sites and more information should arise as follow-up continues. New studies of cancer resulting from the use of injected radioisotopes, including radium and Thorotrast, have been reported, and substantial reanalysis of the older data has occurred. The dose-response patterns of bone and liver cancer in relation to sources of alpha-particle radiation have been improved, but some concern has been voiced about the meaning of estimates of effective dose to susceptible cells, in the presence of local cell necrosis. Related issues have been advanced in regard to lung, thyroid and cervical cancers. There are additional data on leukaemia subsequent to diagnostic radiation, showing very low frequencies of occurrence, and there is further information on individuals exposed to localized high doses of therapeutic radiation for the treatment of childhood cancers. Data on radium dial painters

have been re-analysed [R10], and there are more estimates of exposure risks in underground miners [M19, M42, R5, R7, S20, S51].

42. Several studies, interpretations, and reinterpretations have appeared since 1977, particularly in connection with occupational exposures (Hanford [e.g., G12, K20]; Portsmouth Naval Shipyard workers [N6, R17, R21, S33]; and plutonium handlers [C21, V2, V3, W18]). The earlier results have often been shown, on closer examination, to have been spurious, and the effects of different ascertainment or reporting biases have been revealed.

43. The Life Span Study in Hiroshima and Nagasaki, continues and three further reports on mortality data, as well as additional incidence data have become available. The consequences have been to improve estimates of dose effects for some tumours, to include others in the list of radiation-induced sites (e.g., colon, ovary and, possibly, multiple myeloma), to confirm the absence of excess cases at some sites (e.g., pancreas, uterus and chronic lymphocytic leukaemia) and to confirm the existence of only marginal risk for some sites (urinary tract and oesophagus) [K7, P15, S48, S49]. The Hanford study, the study in the Marshall Islands and studies in British patients irradiated for ankylosing spondylitis, pneumothorax, mastitis or thymus-related conditions continue. While details of the dose-response curve at low exposure levels remain unclear, much has been added to the high-level data and to the nature of the relative risks. In some series, the time distribution of leukaemia seen in Japan and elsewhere has been further corroborated. Absolute and relative risks continue to increase in Japan for many sites, and further follow-up could reveal elevated risk at other sites, clarify some relationships and add new ones.

44. The large series of 14,000 British ankylosing spondylitis patients has continued to accumulate person-years of observation, and some of the patients have been followed for more than 30 years [D21, S31]. The doses received by the patients have been re-evaluated, although not on an individual basis, and several new patterns have been observed. Most notably, unlike the findings in Japan and elsewhere, adult solid tumour risk appears to diminish more than 30 years after exposure.

(b) Ascertainment of exposed and comparison individuals

45. A few new problems have been identified in the ascertainment of cases and matched controls, or comparison individuals since the UNSCEAR 1977 Report. Mostly, however, there has been continued concern over the nature and level of exposure to individuals. For example, as a result of variation in the calibration of equipment and therapeutic technique, it is uncertain how much radiation was actually delivered to the thyroid in children irradiated for tinea capitis or to different tissues in spondylitic patients. There is also the problem of resolving whether, in some instances, the cases were similar to the general population in regard to other cancer risk factors. In one study of radiation effects in childhood leukaemogenesis, it has been suggested that children exposed in utero during diagnostic radiation examinations may already have been at risk of leukaemia, because no similar risks had been observed in Japan [K6]. Exposed individuals at the Portsmouth Naval Shipyard seem more likely to have been discovered if they later developed cancer, and a potential ascertainment bias exists in situations where extraordinary efforts were made to detect cancer in individuals or their exposure to irradiation [B6]. This bias may well exist in studies of military servicemen exposed to the testing of nuclear weapons [C16, C17, K21] or of children exposed in Utah to fallout. A "healthy worker" effect, in which employees in a given industry are healthier than the general population, may have led to spurious interpretations in many epidemiological studies and must be accounted for wherever possible. In several studies, the unexposed group disclosed excess cancers at sites that had been associated with elevated risk from radiation exposure; clearly the status of such individuals in regard to other risk factors must be evaluated. Such an evaluation can be especially difficult if the later effects are small (as in low excess risks of late-onset tumours), if the dose estimates are uncertain or if the investigators are so thorough as to perhaps over-define cancer, as may have occurred in regard to thyroid tumours.

46. In population studies such as those in Japan and the Marshall Islands, the exposed individuals are known and the task is to continue reporting the results as the diseases appear. It is clear that even 40 years of surveillance is not sufficient to exhaust all of the effects, so these large studies must be continued throughout the lifetimes of the exposed. Similarly, in smaller special-cohort studies, such as those of uranium mine workers and therapeutically exposed patients, the long-term effects must be continually monitored as the cohorts diminish through attrition.

47. The more challenging ascertainment problems are to determine the actual exposure levels of the individuals and the correct expected cancer risks for them. The exposure levels are reasonably well known in some of the cohorts, but not well known in others. Even when exposure is well known, the amount of radiation delivered to specific tissues or cell types may not be. This has become an important consideration in regard to liver, bone, and lymphatic cancers in patients exposed to internal radium sources and Thorotrast, for example. Furthermore, it is vital to determine the comparability of exposures to other carcinogens to which the individuals in these cohorts may have been subject.

(c) Suitability of comparison group

48. For a number of potentially relevant studies, there is a continuing debate and re-analysis of data to determine whether the control individuals have been appropriate. For example, it is not obvious whether individuals suffering from disorders such as thymus enlargement or tinea capitis, many of whom were economically underprivileged, were normal in all the other health respects that might relate to cancer.

Similarly, second tumours may arise because of other effects of the treatment for cancer, independently of irradiation: the treatment may debilitate or it may alter hormonal or other physiological states. It is difficult to know the extent to which this may occur, since in the absence of therapy most cancer is fatal, and not much data exist on the subject. If an inappropriate comparison is made, the relative risk applies only to that comparison, not to a more general population. It is essential that the control data, or the population-based expected risks used, do not confound different exposures with other risk factors.

(d) Accurate determination of expected risks

49. One of the most serious problems that has arisen in connection with estimating risks in exposed cohorts is the problem of defining the appropriate expectations for the cohort. The first question is whether the exposed are comparable to the contemporary population from which the expected rates are derived. Specifically, it is critical to determine whether they are similar to the general population in regard to the cancer-related aspects of general health, to socioeconomic status as it bears on exposure to other agents, and to other factors that may have affected ascertainment or that may be affected by the subject's awareness of his continued surveillance. In the Life Span Study in Japan this matter of comparability is less likely to be a problem, although it could be serious in the smaller studies in which specific cancers (such as thyroid cancer, following childhood exposure, or leukaemia, following fallout exposure) may have been screened much more carefully, or defined more loosely, than in the general population. In Japan, internal controls have often been applied, whereby those exposed to high doses are compared to those receiving essentially no exposure.

50. In a large series of cervical cancer patients, there is evidence that other risk factors, presumably involving environmental exposures, make the results less representative for more general populations. In a similar way, the results of the ankylosing spondylitis series indicate various causes of death at different rates from the general British population.

(e) Meaning of relative risk in a changing exposure environment

51. Many significant changes in exposure to non-radiation risks are taking place in regard to diet, the use of tobacco, exogenous hormones and other drugs, toxic agents in the work-place, pollutants, and the like. When these interact non-additively with radiation, they may materially alter the lifetime radiogenic risk of cancer. Such changing regimes of exposure to other risk factors may seriously affect the meaning of relative risk and the dose-response patterns for radiation exposure unless these other factors are taken into account. This is clearly important in risk assessment, because for many organ sites the relative risk for a given radiation dose is a function of the general risk for the tumour.

52. The dependence of the cancer risk due to radiation on the general level of risk for that cancer may not be a serious problem at high doses if those effects appear as very unusual tumours, or as tumours in locally irradiated tissues. However, this dependence is more likely to be important in regard to low-dose effects or to late-onset, long-latency tumours that occur naturally with substantial frequency.

(f) Identification of new exposed cohorts

53. Since the UNSCEAR 1977 Report [U2], several additional groups of exposed individuals have come under scrutiny, and others are potentially available. The groups being studied include nuclear workers [B22, C21, D24, R21] and military servicemen from Great Britain [D26] and the United States [C16, C17, R16] exposed to fallout from nuclear weapons testing. The results are equivocal and may remain so even if the cohorts continue to be followed. To date, most claims have been for leukaemias, and the occurrence of new radiogenic cases, if any, should have ceased, based on what is known about radiogenic leukaemias in other exposed cohorts. Another group is composed of children exposed in southern Utah to fallout from nuclear weapons testing [L4]; this study is controversial, and its estimates of risk are not accepted by most critical investigators (see, e.g., [L5]).

54. A more consequential group of individuals now being studied is one that comprises children exposed to therapeutic radiation for childhood cancers. The continuing investigation of these individuals suggests that second primary tumours occur more frequently than in non-irradiated children. Many of these are leukaemias or sarcomas in the irradiated areas, but carcinomas also occur, including those of the thyroid. These subjects may afford risk estimates for second tumours, especially for sarcomas for which little data exist (except for bone); the doses in these cases were large, and they are reasonably accurately known.

55. Another important new group is a series of over 180,000 women, in a number of countries, who have been followed after treatment for cervical cancer. These women received high pelvic doses and moderate to low doses to more distant organs. Since the UNSCEAR 1977 Report was issued, many reports have appeared on this set of patients. The number of person-years of observation has become substantial and excess cancers are appearing [B12, B23, D9].

56. Many disparate groups of individuals have in common the fact that in the course of their lifetimes they have been or will be exposed to atypical amounts of external, low-LET radiation. Among these groups are radiologists and radiographers, nuclear shipyard and atomic energy workers, as well as segments of the general population exposed to high-LET radon daughter products in their homes or to higher-than-usual backgrounds as a result of where their homes are sited. Most receive small to modest amounts of radiation above the average, but some (early radiologists, for example) may have accumulated lifetime exposures of 2-20 Gy. More and more data are becoming available on the cancer risks in these groups [see, e.g., M18, M30]. Data are also accumulating on nuclear laboratory employees who have been exposed

in the course of their occupational lifetimes (e.g., [W7, W8, W9, W20]). For example, causes of death have been examined for employees of the Chalk River Nuclear Laboratories in Canada who received lifetime occupational doses of 0.2 Sv or more. Through 1982, 413 long-term, traceable employees had accumulated exposures of this magnitude (their average lifetime occupational dose was 0.42 Sv). There have been no excess cancer deaths among the 64 members of this cohort who succumbed; indeed, only 12 cancer deaths were observed where 17.6 had been expected [W9].

57. Information should be available shortly from the study of employees of the United States Atomic Energy Commission (and its administrative successors) who have received occupational exposures of 0.05 Sv or more.

2. Case-control studies

58. Case-control studies have been, for several reasons, less valuable than cohort investigations in the present context. The shortcomings of case-control studies are several. First, in most situations the frequency of prior radiation exposure among cancer cases will be low, requiring very large case numbers in order to estimate relative risks accurately. Second, the absolute dose-response relationship cannot be estimated statistically from retrospective designs alone, since the affected and non-affected fractions are specified through the sampling strategy rather than being the observed outcome proportions among exposed and unexposed individuals [B19]. Third, it is often difficult, retrospectively, to select a truly comparable control group whose risk-factor characteristics closely match those of the cases; this is not a problem in some prospective designs. Finally, there are several sources of potential bias in terms of case ascertainment when exposure history must be ascertained long after the fact (such as a more intensive search for a history of exposure among the exposed group than among the ostensibly non-exposed group).

II. ASSESSMENT OF DOSE-RESPONSE AND RISK PROJECTION

A. PROBLEMS ASSOCIATED WITH ASSESSMENT

1. Form of the regression of response on dose

59. One of the central problems in risk estimation continues to be the shape of the dose-response relationship, an issue treated exhaustively in Annex B of the UNSCEAR 1986 Report [U1]. A number of models have been used or advocated; these include a linear model, a linear-quadratic model, and a quadratic model, to each of which a separate term (or terms) may or may not be added for neutron exposures and for cell sterilization (a decline in response at very high doses). Many of these alternatives are special cases of the more general form:

$$R = (a + bD + cD^2) \exp(-fD - gD^2)$$

where R is the response, or increased risk of cancer (such as absolute or excess relative risk), D is the absorbed dose, and a, b, c, f, and g are coefficients to be estimated from the epidemiological data. These coefficients are usually determined by either the method of least squares or the method of maximum likelihood. In many circumstances it is more accurate to express the value of R as a function of variables in addition to dose; for example, age, sex and history of exposure to other carcinogens.

60. The attractiveness of this model resides in its simultaneous provision for the estimation of linear and quadratic effects ascribable to radiation and those competing effects of radiation, such as cell sterilization or killing, that could obscure the carcinogenic effect itself. Commonly, when the absorbed doses are not large, the exponential term is ignored. A still simpler, frequently used model is of the form:

$$R = (a + bD^h) \exp(-fD - gD^2)$$

Its merit rests largely in its incorporation of either a pure linear effect (when h = 1) or a quadratic effect (when h = 2), with or without competing effects (f and g) and in the fact that it approximates a linear-quadratic form when h has a value between 1 and 2. Thus, it can reflect a convexity in the dose-response curve. However, h can also be less than 1, which poses problems, for then the slope becomes infinite at zero dose.

61. Each of these approximations to the true, biological dose-response relationship has its limitations or potential pitfalls. Common to all the approximations is that inferences based on the shape of the dose-response curve are more susceptible to error than inferences based on the overall slope. In addition, there are errors that stem from (a) an inappropriate choice of the reference value; (b) systematic or random mismeasurement of exposure; and (c) inadequate allowance for latency or too short a period of follow-up. Errors in the measurement of exposure are particularly troublesome, for even the inevitable random mismeasurements can introduce a spurious curvilinearity and cause the slope to be underestimated [G13, G15] and the intercept to be overestimated, unless the latter is constrained to its true value, which is, however, rarely, if ever, known.

62. Much of the data that have helped to identify radiogenic tumours have limited applicability to the analysis of the dose-response relationship, for the doses are either too poorly known or too invariant to permit discrimination among different models. The data on the atomic bomb survivors constitute one of the very few bodies of relevant information.

2. Contingency tables and proportional hazard models

63. Past analyses of the Hiroshima and Nagasaki data have leaned heavily on contingency table methods (a full explication of these can be found in Appendix 3 of [B16]). Essentially, the subject population is divided into several categories (by age, sex, exposure level and so on), and the relative risks within given exposure categories are determined among individuals similar in

other characteristics. This identifies category-specific patterns in which risk is elevated and estimates the excess in each such category. More recent analyses have employed proportional hazard models.

64. Proportional hazard models combine features of traditional multivariate analysis and life-table analysis. The latter method allows one to calculate survival rates and cumulative survival rates making use of all of the data, even if the periods of observation of the subjects differ; the former method allows one to estimate, when several factors are associated with a disease, the extent of the association for a specific factor when all of the other factors are considered. All methods entail assumptions about the presence or absence of interaction and about the nature of the relationship of the causal variables to the occurrence of cancer, which assumptions may not obtain.

3. Mortality versus morbidity data

65. Dose-response relationships at Hiroshima and Nagasaki had at one time been based almost exclusively on the results of the continuing mortality surveillance. However, in 1958 tumour registries were established in these two cities under the auspices of the respective City Medical Associations and with the technical support of the Atomic Bomb Casualty Commission (ABCC), predecessor to the present Radiation Effects Research Foundation (RERF). The specific purpose was to develop and maintain a source of information on tumours diagnosed in the two cities. Like most such registries in Japan and elsewhere, they incorporate various kinds of information (clinical, pathological, radiological, etc.); however, because they employ field investigators who visit all large hospitals periodically to collect data, their ascertainment of the occurrence of a tumour and its confirmation is more complete than that of most other registries, which depend upon voluntary reports from participating hospitals. Thus, for example, in the Nagasaki registry, 72% of the tumour cases are confirmed (that is, there is, in addition to the clinical report, autopsy, surgical pathological or surgical operational data on the tumour) and only 7% of cases are ascertained solely through death certificates. This contrasts markedly with the figures obtained from other registries in Japan, where, on average, only 50% of the cases are confirmed and 37% are ascertained through death certificates alone. These superior methods of ascertainment notwithstanding, the utility of the registry data hinges ultimately on the absence of bias. No exposure status bias in data collection has been revealed in the data of either city [W5]. Method of diagnosis of the tumour, reporting hospital and frequency of doubtful cases do not differ as a function of dose.

66. For fatal cancers, the relative risks based on excess incidence cases, rather than excess deaths, and on T65 doses are generally either the same as or slightly higher than the relative risks based on mortality for the same years (1959-1978); however, the absolute risk estimates (excess incidence cases per 10^4 PYGy) are higher. The mortality data suggest an overall average excess risk of death from a solid malignant tumour of about 2 per 10^4 PYGy; registry data from Nagasaki, on the other hand, limited though they are, suggest a morbidity risk six to seven times higher. Thus, for all cancers except leukaemia, the number of excess incidence cases per 10^4 PYGy is 9.6 whereas the number of excess deaths is 1.4. In Hiroshima, similar data suggest a twofold greater absolute risk; the values are 13.6 excess incidence cases and 6.2 excess deaths per 10^4 PYGy. In both cities, a substantial proportion of this difference is of course accounted for by tumours of the breast, prostate and thyroid, which are seldom identified immediately as the causes of death. However, an important contribution is also made by cancers of the digestive organs, notably the stomach.

B. DOSE-RESPONSE PATTERNS

67. The accurate estimation of dose-response patterns for each tumour site and the evaluation of low-dose effects are impeded by several facts: (a) the long average latent period (the continuing increase in absolute risks of cancer among the atomic bomb survivors in Japan suggest this period exceeds 40 years); (b) the relatively small expected additional risk, even at intermediate or high doses; and (c) changes in exposures to other carcinogens, which could interact with radiation exposure and make it difficult to interpret dose-response patterns in terms of the future risks of current exposure levels. It is certain also that the increased accuracy of dose measurements and the increasingly sharp focus of the beams used in therapeutic radiation will cause dose-response estimates to change.

1. Assessment of the effects of low dose

68. As was stated and thoroughly discussed in Annex B of the UNSCEAR 1986 Report [U1], an assessment of the effects of low dose is clouded by the need for large samples, the difficulty of accurately estimating exposure and the growing importance of extraneous sources of variation, including diagnostic and therapeutic exposures that are less compromising when the doses are large. Two of these difficulties warrant special consideration. Precise direct estimation requires impracticably large samples. Estimates of low-dose risks based largely on high-dose data must depend heavily on the assumptions about the shape of the dose-response curve and are, of necessity, no better than the model is applicable. Current data suggest that resolution of these difficulties will not be easy, and it seems likely that there will be many site-specific differences.

69. Many individuals who entered Hiroshima or Nagasaki soon after the bombs (to carry out relief or other activities) are included in the "not-in-city" group of the Life Span Study cohort in these two cities, a group which has not been used in recent analyses of the Life Span Study data. Early entrants, defined as individuals who entered Hiroshima or Nagasaki within one month of the bombs, are represented by 4,512 individuals in the cohort sample

[K13]. Most presumably, they received some exposure to residual radiation from fallout and neutron activation in soil (if they were in the vicinity of the hypocentre). It is difficult to estimate precisely the dose received by these individuals, for it attenuates rapidly with distance from the hypocentre, and the exposure depends upon their proximity to the hypocentre and time spent in a particular location. However, since it is improbable that individual exposures could have been large, as they were for directly (promptly) exposed persons, a remarkable increase in radiation-induced cancers is highly unlikely.

70. Mortality among early entrants has been followed, and some site-specific incidence studies have sought to determine whether they are at increased risk of the specific malignancy. Kato and his colleagues [K13] found no increased incidence of leukaemia and other cancers among the early entrants. However, Rotblat [R8] has described an increased incidence of leukaemia among these subjects, based on a report of Hirose [H9], and maintains that this is an example of a low-dose effect. The latter report is flawed in many ways. First, there is a problem in the estimation of the denominator (the base population) used to calculate the leukaemia incidence. In the studies of Hirose and Rotblat [H9, R8] the number of early entrants residing in Hiroshima and Nagasaki is estimated on the basis of data from two or three cross-sectional surveys conducted from 1950 to 1974 and not on the basis of data from a cohort. Second, migration is not taken into consideration. Estimation of the base population is a particularly serious problem since the authors did not employ a compelling method to examine the dose-response through grading early entrants by time and place of entry. Third, although the leukaemia cases among the early entrants have a distribution by type similar to that seen among atomic bomb survivors, the peak annual incidence does not occur in the early 1950s, as it does among atomic bomb survivors, but in the early 1960s, when leukaemias among the survivors themselves were few in number. Ohkita has called attention to still other difficulties [O4].

71. More recently, as part of a general study of the incidence of thyroid cancer among atomic bomb survivors, Ishimaru and his colleagues looked for, but did not find, excess risk of malignancy among early entrants in Hiroshima and Nagasaki. Indeed, only one case of thyroid cancer was observed in Hiroshima (and none in Nagasaki) among those early entrants who were close to the hypocentre within two days of the bombing. Patently, the number of cases are too few to evaluate the effect of exposure to residual radiation rigorously, but no difference in incidence was seen among early entrants, late entrants and survivors who had received a dose of less than 0.01 Gy in either Hiroshima or Nagasaki.

72. The discussion here focuses on specific studies, namely, those that involve high dose rates (albeit low doses), from which most of our knowledge is derived. However, to the extent possible, it will also consider low-dose, low-dose-rate exposures such as those received occupationally or those received by individuals who live in houses with high radon levels. Limited though the data may be, they are summarized on a site-specific basis to illustrate differences and to indicate approximately values of relative risks.

2. Assessment of the effects of high dose

73. The mortality experiences of the survivors of Hiroshima and Nagasaki have been, and will undoubtedly continue to be, the most relevant single source of information on the frequency of occurrence of radiation-related cancers. These experiences have not only identified those malignancies that increase in frequency following exposure but also provided insights into the probable dose-response relationships that obtain. These differ by site in biologically consequential ways.

74. Estimates of tissue-absorbed dose for Hiroshima and Nagasaki were published in 1978 [K14] (see also for the foetus [H7]), though they had already been used in the UNSCEAR 1977 Report; these estimates will be discussed later in connection with the reassessment of the individual exposures of the survivors of the atomic bombings. Revised estimates for the Marshall Islanders will soon be available.

75. Re-evaluation of the exposures of the survivors of the atomic bombings of Hiroshima and Nagasaki has disclosed that their estimated neutron doses were substantially lower than had previously been thought [R9, R20]. The findings, particularly those for Hiroshima, are therefore much less informative about the effects of neutrons than heretofore presumed. Differences, albeit not statistically significant ones, exist between the cities, and there remains a need to find alternative explanations for these. It will also be necessary to re-examine carefully the even more limited epidemiological data on the relative biological effectiveness (RBE) of neutrons, if this source of radiation is to figure appropriately in the estimation of risk.

76. Risk assessment from results of the cervical cancer series are complicated by the very different doses delivered to the various organs. In particular, pelvic organs were exposed to doses high enough to make cell sterilization probably quite important (i.e., risk is less than would be expected under linear dose-response assumptions), while other tissues were so little exposed that accurate dose-response information cannot be obtained. Under such circumstances, it is difficult to use the results of this series to determine whole-body risk estimates.

C. RISK PROJECTION

77. From the public health and regulatory points of view, it is important to know as accurately as possible the impact which a given radiation exposure would have on a population, so that criteria for controlling such exposures, or for anticipating the results of accidents, can be developed. An important aspect of such knowledge is the need to estimate the lifetime cancer experience of an exposed cohort of individuals. Lifetime data are rare, even for single-site risks, and

complete lifetime multi-site data are not yet available from the major cohorts that have been under surveillance for the past three or four decades. As a consequence, it is still necessary to project lifetime risks from data based on only portions of the lives of exposed individuals. Such risk projections depend heavily on: (a) the actual risks observed in the available cohorts and (b) the model used to extend the risk beyond the currently available data. Thus, in comparing the results to be discussed below of different projections made at different times, one must recognize not only the changes in the observational data as a result of further follow-up or estimated doses, but also in the projection model that was used. It must also be borne in mind that the projections are invariably least certain for those individuals exposed early in life.

78. Risk projection, generally or site-specifically, requires knowledge of at least the following: (a) the latency time (that is, the time from exposure to the first expression of excess risk) and the plateau period (that is, the time from the first expression of excess risk until the excess risk disappears); (b) the relationship between excess risk and baseline risk, as a function of time since exposure; (c) the age distribution of the exposed population and the baseline pattern of age-specific mortality rates from all causes and from the cancers under consideration; (d) the effect of age at exposure; (e) the effect of sex; (f) a dose-response function; and (g) the effect of environmental exposures. Other factors may also need to be considered, such as the different effects of low- and high-LET exposures, and of low and high dose rates.

79. Knowledge of these factors can only be derived from the experience of a small number of cohorts (the survivors of Hiroshima and Nagasaki, the ankylosing spondylitis patients, and the large international series of cervical cancer patients). The details of these studies and their site-specific risk coefficients will be presented later in this Annex. Most of the other studies serve primarily to confirm these three studies. (These comments pertain mainly to low-LET, high dose rate exposure.) There are as yet no definitive studies from which to estimate lifetime effects of exposure to high-LET radiation (e.g., occupational exposure in mines, radon in homes) or very low-dose and low-dose-rate exposures of either high- or low-LET radiation.

80. There have been several recent attempts to project the long-term post-radiation effects of whole-population exposure. Notable are (a) the BEIR 1980 Report [C4]; (b) a study by the United States Nuclear Regulatory Commission regarding risks in a population from exposures due to a nuclear accident [G11]; and (c) an attempt by the National Institutes of Health of the United States to estimate the probability that a specific cancer was radiation induced at times subsequent to exposure [U4]. These studies have had specific objectives, and all have been applied to the population of the United States.

81. The purpose of the BEIR computations was to estimate as accurately as possible the lifetime risk in a population of the United States exposed to a given dose of radiation according to two different projection models [C4]. The purpose of the report of the Nuclear Regulatory Commission was to provide estimates of the lifetime additional risk from a whole-population exposure, such as in a reactor accident, based on a range of assumptions that were consistent with the available radiation data. The purpose of the National Institutes of Health study was to assess the probability of causation of a given cancer by radiation exposure as a function of time since exposure; the computations could be useful for assigning compensation to persons in whom radiogenic cancer may have occurred.

82. This section will review the factors that must be known in order to make risk projections. It will also summarize the main studies that have attempted such projections, and will outline the basic concepts employed by the subsequent review of the literature. The same concepts will be used in chapter VII of this Annex, where new lifetime risk projections will be made. Most of the parameters for the risk projections have been estimated from the Japanese data [K7, W5] and the ankylosing spondylitis data [S28, S31]. Both sets of data have undergone major risk estimate revision [e.g., D21, P15, S48, S49] and dose estimate revision [L16, N9, R20]. While these revisions do not alter the number of excess cases from these exposures, they do change the level of risk per unit dose. While the following discussion on risk projections is not based on the most recent dose-estimate data, the methods themselves are appropriate. Chapter VII provides risk projections based on the most recent data.

1. Latency time and the plateau period

83. Epidemiological data cannot distinguish between the first occurrence of a radiogenic tumour and its clinical appearance, so that in this Annex the time until the tumour is clinically detectable is referred to as the "latency time" for a cancer. As will be shown below, different human cancers have clear and characteristic latency times following radiation exposure [L9].

84. For adult exposures, leukaemias and bone cancers have a minimum latency time of 2-5 years, whereas solid tumours have a minimum latency time of approximately 10 years [U1]. For solid tumours, excess tumours commonly occur at ages comparable to those at which spontaneous tumours of the same site occur. The evidence is not clear or consistent as to whether other risk factors, such as smoking in the case of lung cancer, interact with exposure to hasten the onset of radiogenic tumours.

85. The pattern following childhood exposure is somewhat variable. Tumours that typically arise in childhood, such as osteosarcomas, occur in the exposed at ages similar to those at which they occur naturally. Bone cancers and leukaemias have a 2-5 year latency. For carcinomas of typically adult onset, the latency time is 10 years or more, and current evidence suggests that they also arise at their normal ages, late in adult life.

86. The appearance of radiogenic leukaemias and bone cancer commonly follows approximately a log-normal distribution [C4]; as earlier noted, the excess risk appears after about 2 years and reaches a peak by 10 years. Data on other tumours are less clear, and it is usual to assume that after the latency time full excess risk is, approximately, attained [C4]. One report [U4] has fitted a cubic function in order to produce a smooth transition from zero risk to maximum risk over the period from 5 to 10 years after exposure.

87. The plateau periods, or periods of expression of excess risk, observed for specific tumours are generally consistent over a variety of studies, although there are exceptions. For leukaemias and bone cancers, excess risk typically declines with time, but still exceeds that in the controls as much as 40 years later in the case of the atomic bomb survivors and the ankylosing spondylitics, though it has ended after 25-30 years in other studies.

88. The plateau period for adult carcinomas among individuals exposed as adults appears to be open-ended; that is, in almost every instance, once risk has become elevated it remains elevated for the rest of the life of the exposed individual. Most major exposed cohorts are still under investigation, and this finding could change; in one major study, that of the ankylosing spondylitics [D21], the excess risk of adult carcinomas seems to disappear 25 years after exposure. Since this finding with respect to the spondylitics has not generally been seen with respect to adult atomic bomb survivors or the subjects of other studies, it may be unique to that study. However, it should be noted that in Hiroshima and Nagasaki among the two youngest cohorts, i.e., 0-9 and 10-19 years of age at the time of the bombing (ATB), the risk has been declining significantly in the 0-9 year group, also in the 10-19 age group, but not significantly so.

2. Excess and baseline risks as a function of time since exposure

89. As was stated in paragraph 13, there are two basic models for the pattern of expression of risk after exposure (once the latency time has passed). These are often known as projection models. The first is the constant additive projection model, according to which there is a constant number of excess cancers in any given year per unit number of persons exposed per unit dose. That is, the number of excess cancers is fixed, regardless of the baseline risks:

$$R(\text{exposed}, t) = A + R(\text{unexposed}, t)$$

where A is the absolute excess risk for all $t >$ latency time. The value A is usually estimated in one of two related ways. In the first, the total number of cancers expected in the cohort had they not been exposed (i.e., the baseline risk) is computed, and subtracted from the number observed in the cohort, and divided by the total number of person-year-Gy (PYGy) of observation. In the second, a regression model may be fitted to the time of onset of every cancer; such models express the excess risk and the baseline risk as a function of age, sex, time since exposure and perhaps other risk factors. If the additive projection model is correct, then at any post-latency time the difference between observed and expected cancers, divided by the total PYGy observed, will be constant. Sometimes a variable excess risk model is used, in which the value of A is estimated from the data by regression methods, specific to sex, age at exposure, time since exposure, and/or other variables.

90. The second basic projection model is known as the multiplicative projection model. According to it, the ratio of incidence or mortality rate in the exposed to that in the unexposed is constant once the latency time has elapsed. That is,

$$R(\text{exposed}, t) = RR \times R(\text{unexposed}, t)$$

where RR is constant for all $t >$ latency time. The value RR has been estimated in two ways. First, the number of observed cancers at some time t after the latency time is divided by the number of expected cases. Sometimes, the excess relative risk per Gy is computed. If the multiplicative projection model is descriptively correct, there should be an approximately constant relative risk at any post-latency time in an exposed cohort. In some instances, a variable multiplicative risk model is used, in which the value of RR is estimated from the data by regression methods, specific to sex, age at exposure, time since exposure and/or other variables.

3. Age and sex structure of the population and baseline mortality rates

91. To predict future cancers in an exposed cohort, it is important to know the age (and sex) distribution of the cohort. This is so because with either the multiplicative or the additive risk models, since baseline cancer risks change with age and sex, the number of cancers expected depends on how many person-years of experience at different age (and sex) categories occur in the data. In an exposed population of mixed ages the number of expected cases per exposed person of age t is E(t) and a fraction f(t) of the exposed cohort is in that age category, the total number of expected cancers will be

$$\sum_{t=0}^{\infty} f(t) \times E(t)$$

A similar weighted expectation can be computed for each sex. The observed number of cancers can then be compared to this aggregate expectation.

92. The number of expected cancers depends on the number of person-years at risk experienced at each age (after the latency period) and the baseline risk. The number of person-years to be lived between ages y and y + n, per person now in age group x to x + n, is a standard life-table function which depends solely on the baseline age-specific mortality rates, m(t), for ages $x < t < x + n$. The number of person-years declines each year as mortality occurs (i.e., as survivorship declines). The cause-specific mortality rate for a specified tumour site is a component of the m(t) schedule, and the expected deaths at any given age can

be computed approximately by multiplying the cause-specific rate by the number of person-years. This approach is followed in risk computations for this Annex in chapter VII, and is essentially the method used by the BEIR III, the National Institutes of Health, and the United States Nuclear Regulatory Commission computations referred to earlier [C4, G11, U4].

93. On the assumption that current mortality rates do not change, life-tables may be constructed for an actual or hypothetical exposed cohort to compute the person-years and expected cancers. The excess cancers are determined by multiplying the person-years by a series of coefficients appropriate to a given projection model. For example, with the additive projection model, the number of excess cancers per person-year is the coefficient. With the multiplicative projection model, the baseline rate [m(t, cause)] is multiplied by the relative risk coefficient, RR. This is multiplied by the person-years to determine the number of cancers in the exposed group. Excess cancers are this number minus the number expected in the population in the absence of exposure. Given the assumption of unchanging risk coefficients and baseline mortality rates, it is possible to compute the additional risk to any exposed group of persons in the population for which the baseline risks are applicable.

4. Age at exposure

94. The age at exposure can affect the coefficients of subsequent absolute or relative risk (i.e., the values of A or RR can be specific to age at exposure). Few statistically sound generalizations can be made about this, except (as will be shown later) that for some tumours, notably those of the female breast, exposure in childhood can lead to much greater risks, and exposure after age 50 to lesser risks, than exposure at intermediate ages. Childhood exposures leading to childhood cancers are generally treated separately; other than for leukaemia and bone cancer, there is relatively little data on the details of the projection effects for such exposures because the Japanese exposed to the atomic bombs in childhood (the main source of data) are still too young for the late-age effects to have been expressed.

5. The dose-response function

95. The dose-response function is discussed fully in the UNSCEAR 1986 Report [U1]. For projecting risk, the dose received by each exposed individual must be taken into account. Where a linear dose-response pattern is assumed, the dose is used directly. Where a more complex pattern is assumed, the dose is translated into some selected function of dose via the equation relating risk to dose for that pattern. The risk is then linear relative to this function of dose. The equations used in human studies have essentially all been variants of those described in paragraphs 59 and 60.

96. Usually, in projection, a model of excess deaths (cases) or relative risk per unit dose is determined. If a non-linear dose-response is desired, the number of excess deaths (cases) or the relative risk per unit dose is multiplied by the appropriate function of the dose (e.g., $a + bD + cD^2$).

6. Other exposure factors

97. Where adequate information is available, the projection of risk may take into account such factors as exposure to smoking or other environmental hazards, other radiation exposures, or the different biological effectiveness of high-LET radiation. As long as one can supply an appropriate dose-response function, a projection model, and an estimate of additive or multiplicative projection coefficients, the same principles should apply.

7. Previous approximations of lifetime risk projection

98. It is useful to summarize the most important recent attempts to estimate lifetime risks, or related measures, from population exposures. As was noted earlier, while these studies attempted to synthesize risk coefficients from the world literature, they relied most heavily on the Japanese and ankylosing spondylitis data; however, the latter studies have since been updated, in terms of both new dose estimates and longer follow-up times, so the specific risk estimates they once provided must now be reconsidered.

99. *The BEIR 1980 Report* [C4]. The BEIR Committee attempted to synthesize the data on radiogenic cancer risk as of approximately 1979. It used a life-table projection approach, employing the 1969-1971 life-tables from the United States, and baseline cancer mortality rates for five-year age groups. A table was devised to convert mortality data to incidence data, based on cancer survival rates, so that estimates could be made for both the commonly fatal and the rarely fatal radiogenic cancers. This is given here as Table 3. A table of risk coefficients, excess cancer incidence per 10^4 PYGy was derived, and estimates of the lifetime risks associated with single exposures and continuous exposures were computed, based on linear and linear-quadratic dose-response functions, for both risk projection models. Estimates were made separately for leukaemia and bone cancer and for all other cancers combined. Representative summary tables from the BEIR III Report are repeated here as Tables 4, 5, 6 and 7 for comparison with the projections presented in chapter VII.

100. *The United States Nuclear Regulatory Commission study.* As part of a study sponsored by the United States Nuclear Regulatory Commission, Gilbert [G11] developed estimates of lifetime risks that would pertain to the population of the United States were it exposed to a nuclear accident. These estimates are for low-LET, single-exposures and are specific to the population of the United States (e.g., baseline cancer rates from the United States were used).

101. Gilbert used (a) the age and sex distribution in the United States; (b) the age, sex, and cause-specific

mortality rates in the United States; (c) a model of the dose-response pattern, latency period, and projection effects; and (d) estimates from past studies of the absolute or relative risk per unit exposure, largely from the BEIR 1980 Report updated by subsequent papers from Japan and the spondylitics. She provided methods for computing the total number of years of life expected to be lost as a result of the exposure incident. The input characteristics used in her study are given in Table 8, which is an adaptation of material from the Nuclear Regulatory Commission study.

102. Gilbert's projection of lifetime effects is based on a linear-quadratic dose-response model, with non-linear effects at intermediate dose rates (<0.05 Gy/day) of low-LET radiation, as might obtain in a nuclear power plant accident. Upper and lower bounds and central estimates for the effects of exposure were computed. These do not have statistical meaning as, for example, mean and confidence limits do; in fact, there is currently no way to provide probability statements on the likelihood that the true effects will take any particular value. Gilbert merely provided what appeared to be reasonable limits for the plausible range of effects.

103. Gilbert used a linear-quadratic dose-response equation to account for the incomplete human data for low-LET radiation [C4], consonant with animal experimental data [N1]. The extent of effect-reduction at low doses and low dose rates is not yet known, but the National Council on Radiation Protection and Measurements of the United States (NCRP) has suggested that the correction coefficient is in the range 2-10 [N1], which is the range used by Gilbert [G11].

104. For comparison with chapter VII, a summary table of Gilbert's results (Table 9), provides her bounds, for various sites, for years of life lost as well as excess cases. Gilbert provided both relative and absolute projection computations. Her absolute risk coefficients were based on empirical data and her relative risk coefficients were those multiplying factors that would produce the same number of excess cases as actually observed.

105. *Probability of causation: the radioepidemiological tables of the National Institutes of Health* [U4]. For some purposes, it is of interest to estimate what fraction of cancers occurring in a given exposed population at a specific time post-exposure may have been caused by the exposure. Even if a specific cancer cannot be said to be radiogenic, it can be estimated by what fraction the baseline risk is elevated. In general epidemiology this would be termed the attributable risk, but in radiation epidemiology it is often referred to as the probability of causation (PC). The probability of causation of a specified cancer by radiation was defined earlier as the excess cases due to radiation divided by the total cases (see paragraph 21).

106. The study of the National Institutes of Health used essentially the same data as Gilbert, with modifications to various components but a comparable life-table approach. The study computed a value, R, defined from PC as follows:

$$PC = [\text{Prob(cancer w exp.)} - \text{Prob(cancer w/o exp.)}]/\text{Prob(cancer w exp.)} = R/(1 + R)$$

where R is the excess relative risk, defined as the increase due to dose D as a proportion of the probability of cancer in the absence of the exposure. These probabilities are specific to a given dose, sex, and age at and since exposure.

107. The National Institutes of Health report defined R in terms of its components as follows:

$$R = F \times T \times K \times W$$

where F is a function of dose, T gives the dependence of R on time since exposure, K is the dependence of R on age at exposure and W is the effect of an additive interaction between radiation and other (known) risk factors. The study described each of these parameters for each tumour site.

108. Qualitatively, the results of this study can be summarized as follows. For leukaemias and bone cancers, the probability of causation rises rapidly with time after the minimum latency time, reaches a peak (whose height depends on dose and which is maintained for 10-20 years) and then falls to zero at the end of the risk period. For other cancers, the probability of causation is roughly constant at all ages after the minimum latency time has passed, but is a function of age at exposure. It is typically highest for young ages at exposure, declines to a minimum (which varies by site) for ages 40-50 at exposure and then may rise slightly or stay roughly constant.

8. Risk coefficients for high-LET radiation

109. Much of the collective dose from high-LET radiation received by human beings comes from exposure to inhaled radon daughters, thorium decay products and the like. The radiation dose in these instances is mainly from alpha emissions. In addition, the exposure is chronic over many years, as, for example, in the cases of underground miners in hard-rock or miners of radioactive ore, and of the many individuals who live where the bedrock or soil provides a source for radon gas entry into homes.

110. Because the doses received under these circumstances are chronic in nature, the models discussed above are not really applicable (they project the risk subsequent to single or short-term exposures.) In chronic exposures, the cancer effects are the results of a dose that continues to build over many years. The most widespread risk is cancer of the lung due to the inhalation of radon daughters.

111. Thomas and McNeill [T11, T16] estimated excess deaths and relative risk of lung cancer from available data on exposures of underground miners to radon and daughters. Their results, which are given in Table 10, are discussed further in chapters III and VII.

112. The risk estimates varied considerably from one study to another in the survey by Thomas and McNeill. This variation may be due to several factors, including the effects of smoking, differing dose rates (i.e., ambient concentrations of radionuclides in the air of the mines), inaccuracies in dose estimation, or other confounding factors. Some of these factors are discussed below. No simple, single risk pattern emerges; there is about a fourfold difference in estimated lifetime risks, per WLM, depending on which exposed cohort is used as the basis for the estimate.

113. The risk coefficients derived from these high-LET exposure data have not been used to project lifetime risks in exposed cohorts. A method for such computations will be suggested later in this Annex.

9. Selection of preferred projection model

114. The BEIR 1980 Report [C4] predicted lifetime risks under a variety of assumptions by projecting risks estimated from observed data into the future, but unobserved, lifetimes of exposed individuals. The number of excess cases estimated using an additive model of risk per 10^4 PYGy was about a factor of three less than the number of excess cases estimated using a multiplicative model. Although they had employed somewhat different assumptions and updated data, Gilbert's results were essentially the same [G11].

115. When the additive and multiplicative projection models provide differing results, it is obviously important for practical applications to determine which, if either, model is to be preferred. In examining this problem and its consequences for risk projection, Muirhead and Darby [M36, M37] developed a generalized statistical model for risk projection and tested its fit, as well as the fits of the additive and multiplicative projection models, which are special cases of the generalized model, to the available data. The authors expressed risk in the exposed, R(d), in relation to the age-specific risk, R(0), in the unexposed, as a function of dose, d. They used the general function

$$R(d) = \{[R(0)]^{\gamma} + [1 + \alpha d \exp(\Sigma \beta_i x_i)]^{\gamma} - 1\}^{1/\gamma}$$

where the x values in the exponential term are covariates and the β values are their regression coefficients, taking into account age at exposure, time since exposure and sex, and Σ implies summation over all such covariates. The parameter γ can be thought of as indicating the model type: if $\gamma = 1$, the additive projection model results; if $\gamma = 0$, the multiplicative model results; other values of γ express intermediate types of model.

116. Muirhead and Darby tested this approach with mortality data on all cancers except leukaemia in Hiroshima up to 1978 between the 0-0.09 Gy and above 1 Gy dose groups. Table 11 shows the results of some of their fitting efforts, and Table 12 the implications of the different models for lifetime risk projection. While this work was not based on the most recent dose estimates, the qualitative nature of their findings seems unlikely to be changed appreciably with new doses.

117. In the absence of covariates, the best-fitting value of the parameter lies between 0 and 1, but the fit of this model to the data is not good. Adding age at exposure improves the fit, which is even further improved by adding time since exposure. Those models which do fit well are shown in Table 11. What is clear is that none of the simplest models fits the data best, and a variety of models can generate a statistically comparable fit. Yet, as shown in Table 12, (a) these models lead to very different lifetime risk projections; (b) the multiplicative and additive projection models do not necessarily provide upper and lower limits to the risks among this family of models; and (c) the number of years of life lost, which Gilbert's projections had shown to be relatively similar under multiplicative and additive models (compared to the excess number of deaths), are quite variable among the possible models. The latter difference is probably due to Gilbert's using a constant risk coefficient for all ages at exposure.

118. This work shows the importance of knowing the nature of the effects of radiation after exposure, on the projection of lifetime risks, and that it is difficult with present data to determine a clearly best-fitting model. Different data sets would be fitted best by somewhat differing models. Only if the total lifetime effect of radiation is known can a choice of a projection model be made confidently. If, as has recently been found in the spondylitis data, excess risk of solid tumours, in fact, diminishes or disappears after 30 years, then the bulk of tumour expression may have been seen in some of the current cohorts, and projection efforts could be made with relatively less uncertainty. However, the Japanese data do not yet show such a decrease, except at the youngest ages at time of exposure.

119. The major importance of the work by Muirhead and Darby is to suggest that current data cannot yet provide a model by which to project lifetime risk accurately, or even confidently to bracket the range of likely risks. Indeed, even if (with currently available cohorts of data) one projection model fits the data best, this must be taken to be a numerical rather than biological fact; not enough is known about radiation carcinogenesis to construct a single biologically correct projection model, if indeed one exists.

III. BIOLOGICAL ISSUES IN THE ASSESSMENT OF RADIATION CARCINOGENESIS

120. The data available for the assessment of radiation carcinogenesis in man come from several sources, the most important of which have already been cited. A number of different approaches have been used to evaluate the patterns of cancer that occur in irradiated individuals. While some studies have combined several approaches, and the approaches are not incongruent, most have employed only one, or a few.

A. BIOLOGICAL MODELS OF RADIATION CARCINOGENESIS

1. Multi-stage models and the role of radiation in carcinogenesis

121. At the cellular level, cancer is a clonal, molecular-genetic disease. One way to understand the response of an individual to radiation is to model the process of carcinogenesis itself in terms of the events thought to occur at the cellular level during the transformation of cells from normal to malignant. Generally, such models consider cancer to be a multi-stage process; it is presumed that for a cell to be affected, a series of k events must occur in its lineage; the time, or age, to a tumour is a function of the rate at which these events take place. The k events must occur in a single cell lineage, the last event rendering some single cell cancerous and causing it to become the progenitor of the entire subsequent tumour and its metastases. This has been established biologically for such a wide variety of tumours that it can be accepted as a fact.

122. Numerous multi-stage models have been proposed (see [W1] for a review). One of the motivating factors behind the development of these models has been the observation that many tumours in man and animals exhibit a linear increase in the logarithm of the incidence (or hazard) function, h(t), plotted against the logarithm of age, t; that is,

$$h(t) = At^{(k-1)}$$

where A is a constant of proportionality, usually a function of the transformation rates of the k events, among other things. Empirically, the slope of such a plot on a log-log scale is 4-6 for a wide array of human [C1] and animal [P1] cancers. It should be noted that many non-mutational chronic diseases show similar patterns.

123. Many other variations of multi-stage models have been proposed, but there are problems associated with any purely formal approach to radiation-induced carcinogenesis. Stochastic models of carcinogenesis model the process at the cellular level, yet data on human radiation carcinogenesis used to test those models come from observations on populations of individuals. It may be unrealistic to infer from such data much about the nature of the process itself or of the role of radiation. Additional errors may arise from the heterogeneity of the exposed population, which is not considered by statistical models.

124. Based on the way the multi-stage statistical models were developed, some investigators have interpreted the slope of a log-log plot of cancer incidence data (4-6) as directly reflecting the number of stages involved. For a variety of reasons, it is unlikely that this interpretation, based on population data used to infer cellular processes, is useful for this purpose [W3]. However, it is possible, without specifying the nature or even the number of stages, to express the effect of exposures of varying intensity to carcinogenic agents that affect only one of the required stages. This has been done by Whittemore [W2], by Day [D2] and by Day and Brown [D1]; Whittemore derived a table of the expected effects on both the absolute and relative risks of constant exposures, single exposures and short-term exposures [W2]. If the first of the necessary transforming events is affected by the exposure, the number of individuals already partially transformed should increase, and even after the specific exposure terminates, they will remain at excess risk, having to await only a smaller number of events in subsequent years. If the last stage is affected, then those cells that have already experienced some events will be quickly transformed, but once the exposure ceases there will be no further excess risk in the exposed cohort relative to the unexposed cohort. If an intermediate event is affected by the exposure, the fraction of the cohort in a more highly prepared state (and, hence, the rate of occurrence of disease) should increase, as with an early stage event. However, after some time has passed, the remainder of the cohort will also gradually accumulate these stages, and the excess risk in the exposed cohort will diminish.

125. These predictions have been applied to age-onset data in exposed cohorts of individuals to infer what event may be affected by radiation in the generation of cancer in various organs; the results are summarized in Table 13. The bases for these inferences are the relationship between age at exposure, the relative risk, and time since exposure. A major point is that if a late stage is affected, then individuals who have already experienced all prior stages will quickly manifest a cancer; because a higher fraction of older individuals are presumably in such a condition, late age of exposure should, according to this model, manifest more, and quicker, excess cancers than in younger individuals. On the other hand, if an early event is affected by radiation, there will be a long time until those affected will manifest their excess tumours. Further, the effect of radiation would be less at later ages, since more of the older population would already have experienced these early stage events. The logic of the interpretation is given in [C22], and several applications can be found in [W2].

126. As Table 13 shows, these ideas may have merit, but they have not yet led to easily understood conclusions. In leukaemia and other tumours, there are no simple or clear patterns of relationship of latent period with age at exposure, or latency itself. Therefore, epidemiological evidence alone cannot be used to make reliable inferences about the nature of the carcinogenic process either in terms of the number of stages involved, or which of those stages is affected by radiation.

127. To account for smaller numbers of events, or for the differential growth of tumours relative to normal tissue, several models have been proposed [W1]. One is a three-stage model that includes dose-dependent cell killing (or sterilizing) effects, originally proposed by Neyman and Scott [N2]. It is consistent with data on radiogenic osteosarcomas in dogs and humans caused by ^{226}Ra (half-life: 1,600 years) in showing that a response is proportional to the square of the dose at low doses; that incidence is not dependent on time and dose at high doses; and that radiogenic tumours may appear at a much later time

after exposure than simply the tumour growth period [M3, W1, W4]. The first two events are assumed to be affected by the radiation directly, perhaps as mutations with effect proportional to dose, while the third is a bone growth phenomenon related to bone remodelling and the eventual stimulus for transformed cells to grow.

128. This result is somewhat different from the result observed in individuals, both children and adults, who had been given ^{224}Ra, which has a shorter half-life and a very different skeletal dosimetry. In those individuals, excess cancers occur a few years after exposure but no longer occur about 25 years after exposure. The Marshall-Groer model calls for a cell-division effect, which should lead to different results in children, whose bones are more actively growing, and adults; however, this has not been observed [M22].

129. In an attempt to generalize the carcinogenic process, taking into account the promoter and mutational effects, and still generating age-incidence curves which are proportional to the 4-6th power of age, Moolgavkar and his colleagues [M1, M2] have developed a two-stage model, with differential growth of normal or partially transformed cells (or both). This is an improved version of earlier work of Armitage and Doll [A2]. The Moolgavkar paradigm makes predictions similar to those of modified Armitage-Doll models [A1, W2] in regard to the effects of age at exposure and the incidence pattern as a function of time since exposure. It has been fitted to data on breast cancer [M5], where hormonal (and possibly dietary) effects are influential, and to data on smoking and lung cancer. The results are consistent with radiation being a mutagen for both of the mutational stages required in the model, if the known facts of breast tissue growth are taken into account. First, nulliparous women have fewer cells susceptible to transformation which will later undergo extensive mitosis [B1]. Second, the risk of post-pubertal radiation carcinogenesis decreases with increasing age at exposure, again agreeing with the circum-pubertal tissue growth. Finally, pre-pubertal irradiation should have less of an effect, since few breast cells are dividing at that time. Until recently, no pre-pubertal effect had been seen in atomic bomb survivors [T1]; however, this no longer is the case [T6, T7]. From the most recent data, it now appears that in fact the risk may be highest in ages under 10 years and greater than in ages 10-19 years. The longer the interval between irradiation and menopause, the longer will be the period during which partially transformed cells can proliferate and hence be vulnerable to a final transforming event; this age effect has been observed [B2].

130. A number of experimental observations support a model based primarily on the biological nature of tumour formation rather than on formal concepts. Within the framework of this model, two biologically different stages of carcinogenesis are singled out: initiation and promotion. To develop a specific model of radiation carcinogenesis based on a two-stage theory it is necessary to discover the mechanisms underlying these processes (for a comprehensive review see [F9, F10, P13, P14]).

131. The chain of events culminating in a clinically manifest tumour starts with the initiation process in a normal target cell. It is clear that in at least some tumours the event causing initiation is a mutation in the DNA. This can be a point mutation or a chromosomal rearrangement; many examples of both are known. Filyushkin and Petoyan [F9], Petoyan and Filyushkin [P13], and Sandberg [S42] have suggested a hypothesis relating carcinogenesis to symmetrical chromosome translocations (reciprocal translocations without loss of chromosome material). Initiation seems to occur frequently [G16, G17], but most initiated cells never result in a tumour. Several mechanisms ensure that most potentially carcinogenic cells do not cause a cancer.

132. One of the principal mechanisms preventing the development of a tumour, even though mutation has occurred, is the repair of the damaged DNA within a few hours or days of initiation. The time between a mutation and the next mitosis is critical for the final result: stimulation of proliferation after exposure results in a higher number of transformed cells [B32, K24]. This may be due to the diminished time available for repair before fixation of the lesion during mitosis [B34, M33]. Proliferation seems also to be essential with regard to the persistence of the potentially carcinogenic character of the initiated cell. Studies of three different cell lines in vitro revealed that between four and six mitoses must occur after irradiation to lead to a fixed transformation; the first mitosis has to take place within the first 24 hours [B33, K25, L14]. If it does not, the potentially carcinogenic character of the initiated cell is lost.

133. In radiation-induced mouse myeloid leukaemias a partial deletion of the long arm of chromosome 2 is necessary but not sufficient for the disease [H29, H30]. The cells with the deletion proliferate without manifesting malignant phenotypes in the mouse unless a second transforming step occurs [H30]. This stage may be explained as the loss of a suppressor gene or by a second somatic mutation at the gene located on the intact homologous chromosome, as occurs in retinoblastoma and other human cancers.

134. Another important, though seemingly trivial, fact should not be overlooked. An initiated cell may be the cause of a tumour only if the radiation-induced lesions are compatible with cell survival. In the high-dose range, there will be competition between cell transformation and cell death.

135. A fixed transformation still does not mean that a tumour will inevitably develop. The affected cells can apparently remain quiescent for a long time, during which they may be recognized and eliminated, perhaps by the immune system. This process surely takes place, even though it is poorly understood. If all of the mechanisms mentioned above fail, there is still the need for the transformed quiescent cells to start dividing. This activity is thought to be induced by a promoter. Hormones are of particular interest in this connection, as has been shown by different experimental approaches [N7, S43, Y5, Y6]. Generally, substances that stimulate cell proliferation enhance

carcinogenic processes [S44]. It is not clear to what extent radiation can act as a promoter [F1, U7]. Finally, the developing tumour must be vascularized when it has reached about 0.2 millimetres in diameter, in order to maintain an oxygen supply to the cells. Only after all of these processes have taken place and additional growth has occurred will a tumour be clinically manifest [S44].

2. Consideration of results of oncogene studies in statistical models

136. Recently, a rather elaborate picture of the nature of some of the genetic events involved in carcinogenesis has emerged. A limited series of genes, commonly called oncogenes, has been implicated in the transformation of cells to the neoplastic state. Some of these oncogenes seem to be incorporated into the cellular genome by viruses that transfect cells, but the genes themselves, or structurally very similar ones, are known to exist in normal cells. It has been shown in some cases that simple mutations in members of these gene families have transforming activity. Mutation in an oncogene can lead to a modified structure in the coded protein, or, by changing the mechanisms that regulate the coding and expression of such genes, it may cause the ectopic production of a normal gene product, or its production in improper amounts. The biochemical activity of several of these agents has been characterized; they appear to affect a variety of pathways in the control of cell division and proliferation. Other work has suggested that cell transformation may occur after a small number of events, possibly two or three, although in some instances (for example, retinoblastoma) recessivity at a single locus (i.e., two events) may suffice. In the case of recessivity at a single locus, genes of protective effect, now often called "anti-oncogenic", are turned off. This subject is reviewed in Annexes A and B of the UNSCEAR 1986 Report [U1].

137. In the case of these "anti-oncogenes" there is evidence that after a first mutation at one of these loci, a somatic recombination event occurs which replaces the normal gene on the unmutated chromosome with the mutated gene, leading to cell transformation. One recent report from an in vitro study of yeast cells has shown that radiation may induce somatic recombination, thus being able to affect both the initial and the second of these stages.

138. Evidence has accumulated that various carcinogens, including radiation, tend to break human chromosomes at specific locations. While these "fragile sites" are not yet well understood, some correspond closely to known cancer-related chromosomal break sites, for example, known rearrangement points or oncogene locations [Y1]. However, it is not clear whether radiation causes cancer in ways different from other carcinogens.

139. Several studies have shown that mouse cells may be transformed by the application of chemical carcinogens such as N-methylnitrosourea and benzo(a)pyrene [G7, M21, Z1]. This activates the N-ras and H-ras oncogenes, and in one case [Z1] the transformed gene is due to a guanine-to-adenine (G-A) nucleotide substitution (a point mutation). In a different study, the c-K-ras oncogene was activated by gamma radiation, which also caused a G-A substitution [G6]. These findings suggest that, at the oncogene level, the carcinogenic effects of radiation are at least similar to, if not identical with, the effects of other carcinogens. The studies cited involved different tumour sites, so that more direct comparisons are difficult.

3. Does radiation induce unique cancer characteristics at the cellular level?

140. Several investigations have shown that the normal somatic cells of cancer patients differ from their tumour cells in respect to oncogene activation or other chromosomal changes. This finding documents the clonal nature of the tumour and, more importantly, the specific events involved in the tumour's origin. Similar studies should be undertaken in radiation-exposed individuals where it would be valuable to determine whether the tumour cells alone manifest the chromosomal or oncogene-related changes attributable to irradiation or whether the normal cells, too, manifest these changes. In particular, it may be important to examine affected and normal cells in individuals exposed in utero, in order to relate the effects of radiation to prenatal age at exposure, especially for those individuals exposed early in prenatal development.

141. For many reasons it is desirable to know whether the cancer cells produced by ionizing radiation differ cytologically or biochemically from the cancer cells produced by other carcinogens at the same organ site. A clinically detectable difference could have uses in screening and in testing. It could also be valuable in determining which cases of cancer are due to radiation exposure and which are not; this would be useful for epidemiology as well as for occupational safety, liability and the like. DNA sequences at certain loci affected by different mutagens in some experimental systems show characteristic patterns (e.g., base changes, deletions), suggesting that different mutagens have preferential effects at the DNA level. It has been possible, for some cancers, to determine whether or not radiation induces similar changes at the genetic level to those caused by other carcinogens. Experimental studies of chemical mutagens on cell lines and the finding of the "Philadelphia chromosome" in the Japanese atomic bomb survivors with chronic granulocytic leukaemia (as is observed in spontaneous cases) suggest no radiation-specific mutational pattern [F3].

142. The available information generally suggests that radiation induces the same cellular anomalies as other carcinogens. Data from Hiroshima and Nagasaki on breast cancer suggest no differences in histologic type nor tumour size by age at exposure or radiation dose [T9, T14], nor were atypical changes or residual proliferative lesions seen in women exposed to radiation but free of cancer. These observations led

Tokuoka and his colleagues to conclude that "radiogenic breast cancer does not differ histologically from spontaneously occurring (breast) cancer in Japanese women" [T9]. Similarly, Matsuura and his colleagues [M15], in an analysis of the histological types of stomach cancer seen among the survivors, found no compelling evidence of a radiation-specific histological type, although there was evidence that the degree of differentiation of adenocarcinomas is poorer in high-dose than low-dose groups. On the other hand, a comparison between exposed and unexposed stomach cancer patients from Japan showed a higher frequency of better-differentiated tumours in the exposed, who were also several years older than the unexposed [S35]. Twenty years ago, gastric carcinoma in the exposed occurred at the same age as in the unexposed.

143. While in all risk groups the lower third of the stomach was the site of most cancers, there seemed to be an increase in the degree of intestinal metaplasia of the gastric mucosa with increasing dose [M15, Y2]. The Japanese population in general has been prone to develop gastric carcinoma in the lower third of the stomach, in areas affected by intestinal metaplasia. This is also characteristic of stomach cancer in high-risk areas of Andean Latin America and may be related to dietary constituents. However, it is conceivable that the stomach as a whole was predisposed to carcinogenesis by irradiation and that in the lower third of the stomach the normal risk processes were accelerated. If this is true, it represents an interaction with environmental factors and will change, as the frequency of stomach cancer in Japan is changing.

144. An examination of the cytopathology of lung cancers in uranium miners in New Mexico, United States, suggests that the same array of cell types is observed in roughly the same proportion as would be expected [S20]; others have found some differences in proportion, but most of the usual cell types are seen [C4]. Some malignancies have not yet been shown to arise after exposure to radiation; chronic lymphocytic leukaemia and polycythemia vera, Hodgkin's disease and cervical cancer are examples.

145. In one autopsy series [K17], about 25% of the liver cancers caused by Thorotrast exposure were angiosarcomas, a tumour type also caused by chemical agents but which is otherwise quite rare. In this series of 29 autopsies of Thorotrast-induced angiosarcomas, the authors found that the cell types and histopathology were similar to those of angiosarcomas from other causes. Thus, while a radiogenic tumour may be of a relatively rare tissue type, it is not itself different from a non-radiogenic tumour of the same type. The presence of the Thorotrast as an internal, long-term resident in the liver may induce histological changes by means other than simply the radiation effect.

146. In a series of 180 autopsies of Japanese Thorotrast patients who had malignant hepatic tumours, Mori et al. [M31] reported a preponderance of cholangiocarcinomas and especially of haemangioendotheliomas.

147. Biopsies of thyroid cancers from 31 patients who had been given external x-irradiation have been compared with biopsies of thyroid cancers from 389 non-irradiated patients. The irradiated patients were significantly more likely to have the papillary type of tumour, with a higher incidence of metastatic lymph nodes [T13]. While these results suggest that the tissue types are not unique to radiogenic thyroid cancers, they may depend on the external source of the irradiation and may not be comparable to the histopathology after exposure to internal nuclides.

148. A recent study [M34] has found that the distribution of cell types in radiogenic acute leukaemias in adult atomic bomb survivors and cervical cancer patients did not differ from that in spontaneous leukaemias. In spondylitis patients secondary acute leukaemias were of all cell types other than chronic lymphocytic leukaemia.

149. A general conclusion from the available data is that there is no diagnostic difference between the cells of radiogenic tumours and the cells of the spontaneous tumours of the same site. This conclusion is consistent with the fact that cancer is a mutational disease that can be caused by any mutagen.

4. Causal mechanisms: gene activation or inactivation?

150. As mentioned above, some tumours (especially retinoblastomas) are apparently caused by gene deletion, which presumably inactivates necessary genes or gene repressor regions of chromosome 13. The result, if both homologous chromosomes are affected, is cancer. In other tumours, cancer appears to be caused by the incorrect activation of a normal gene, or the activation of a mutant version of a normal gene; in these cases, the tumour occurs in heterozygous cells and the effect is "dominant" at the cell level. Oncogene amplification, one of the means by which oncogenes are activated, occurs in a variety of human tumours including neuroblastomas (where amplification of the N-myc occurs), retinoblastomas [L13], glioblastomas [L12], leukaemias and carcinomas. Radiation may work in different tissues by inducing chromosomal translocations, by deleting repressor sequences, by deleting or inactivating necessary genes or by causing point mutations in normal genes. Further studies will be required to identify the molecular nature of the lesions caused by radiation.

151. As noted earlier in the section describing dose-response models, there is a variety of evidence suggesting that high doses of radiation can damage cells to such an extent that DNA-repair mechanisms are ineffective; apparently, such cells are often so damaged that they are either non-viable or cannot replicate. At least, they do not seem capable of further transformation to malignant states. This form of cell inactivation has been found in several studies. Mole has argued that high doses of radiation to foetuses in utero seemingly show this effect [M7], though this has not been proven directly. Among adults, a relative deficit has been seen in osteosarcomas in radium dial workers subject to very high doses [R12]; in breast

tissue irradiated in the course of mastitis therapy [L6], in thyroid cancer after irradiation including ^{131}I ingestion for hyperthyroidism [D10, H12]; and in the pelvic organs of women who were heavily irradiated to treat benign gynaecologic disease [W6] and cervical cancer [B12]. Leukaemia in ankylosing spondylitis patients demonstrated a similar deficit [D11]. Finally, the deficiency in breast cancer among women treated for cervical cancer [B12] may be due to a different cell-sterilizing effect. Ovaries subject to substantial irradiation may become deactivated, thus indirectly protecting the breast from carcinogenic effects. Higher-order terms in dose-response curves may not be trivial ones, and dose-response estimates should take them into account; this is increasingly relevant as therapeutic radiation concentrates higher dosages on smaller and better-defined tissue areas. On the other hand, the relatively lower dose outside the primary target area may have the inadvertent effect of generating some secondary cancers in cells that would have been sterilized by less advanced equipment and techniques.

B. TISSUE SUSCEPTIBILITY IN CHILDREN

152. Tissues differ substantially in their susceptibility to radiation carcinogenesis, and the age and sex of the exposed individual may also affect their responses. These differences are seen in sites with very low or very high relative risks for given exposures, in patterns of latency or the cell types of post-irradiation tumours, in age and sex vulnerability, and in some aspects of the age of onset of such tumours. It is enlightening to examine the relationships between age at exposure and tumour onset and between proliferating and non-proliferating tissues, as well as the life-cycle events of specific tissues as they relate to susceptibility in that tissue.

153. Data on these special tissue effects come from several sources, including (a) in utero exposure and (b) age at exposure for tissues with marked periods of proliferation or development. The special vulnerability or insensitivity of tissues is informative, for it may identify the process by which radiation induces cancer in specific tissues and the special risks attendant on certain types of exposure. Such knowledge could refine our understanding of which human subpopulations are more susceptible to radiation effects, relative to radiation protection guidelines or to medical therapeutic practice.

1. Exposures in utero

154. The risks to the irradiated embryo or foetus were discussed extensively in Annex G of the UNSCEAR 1977 Report [U2] and again in Annex C of the UNSCEAR 1986 Report [U1]. Those findings are briefly reviewed here, summarizing the best currently available dose-response estimates and considering how current data on prenatal exposure relate to the biology of radiogenic cancer.

155. If cancer is caused by a series of mutational steps, along with the effects of growth proliferation and promoters, the embryo or foetus should be highly susceptible to radiation-induced cancer. The available data are equivocal at best; indeed, animal experiments have failed to find a particular sensitivity [B5, U1]. There are basically only two ways to collect data on this topic. One is to examine the children of women irradiated, while pregnant, for diagnostic or therapeutic purposes, and the other is to examine the children of women irradiated at the time of the atomic bombings. To date, the findings from the two sources seem contradictory. The findings are summarized in Table 14, which only provides published estimates of the approximate average relative risks, and does not directly reflect the controversial aspects of these studies, which will now be discussed briefly.

156. Two large-scale investigations have undertaken to assess whether the fraction of children exposed to x-irradiation in utero was higher for children who died of cancer (generally, prior to age 10-15) than for control children who did not die of cancer. One of the studies was in the United States and the other in Great Britain; both have now been accumulating evidence for about three decades. The first to be reported was the Oxford survey [S5]. It suggested a radiation effect, a cumulative relative risk (which was only crudely correlated to dose) of around 8.25 in the first trimester and 1.45 in later trimesters, a linear dose-response pattern, a relative risk that decreased with historic time, and an age-onset distribution with a slightly higher mean age among cases judged to be radiation-induced than among all cases in the population [S6]; the reasons for the latter finding, if it is other than a statistical artefact, are not evident.

157. This study [S5] has been criticized because the T65 data on the survivors of the atomic bombings at Hiroshima and Nagasaki exposed in utero, a direct and prospective set of observations, do not suggest such an effect [I1, J1] and because the British results apparently conflict with animal data (see [B5, U1]). In particular, it has been suggested that various biases exist in the data, factors that would lead a woman who was predisposed to bear children prone to juvenile onset cancers to be more likely to be irradiated during pregnancy (for example, to diagnose problems already manifest in the pregnancy) [D3]. That poor health might predispose one to be exposed to irradiation was suggested in a study in the United States showing an excess risk in white, but not in black children [D3]. The excess risk persisted after considering sources of bias, which were comparable between the two ethnic groups; this suggests that the effect is real in whites. However, the authors propose an ethnic-specific difference in radiation susceptibility, for which there is no other basis, making it more likely that aspects of black-white differences in socio-economic or environmental conditions are responsible.

158. Stewart and Kneale [K1, K2] have addressed these issues in several ways, primarily by using Mantel-Haenszel [M6] multiple contingency table methods to assess associations between various risk factors, irradiation and childhood cancer. The factors examined include socio-economic status, birth order of the affected child, age of the mother at pregnancy and

birth year. They were indeed found to influence the occurrence of childhood cancer, but a radiation effect persisted even after they had been taken into account statistically [K1]. Stewart and Kneale also contend that there is a detectable dose effect, that the first trimester is a period of high sensitivity, and that the extra x-rayed cases in their survey are indeed radiation-induced.

159. Mole [M7] has argued from data on twins that the selection factor was probably not a serious potential bias. Twins are known to be about five times more likely than singletons to be irradiated in utero, but Mole found their post-irradiation risk of cancer to be basically the same as that of singletons; hence, at least this particular factor predisposing to foetal irradiation did not seem to lead to an altered risk. Twin foetuses are not more susceptible to diseases than singletons so that their predisposition to be irradiated may be different from the data on non-twins reported by Stewart and Kneale. On the other hand, it is curious that twins do not, overall, experience more childhood cancer than singletons, as would be expected based on the greater likelihood of their having been irradiated [B15].

160. This was confirmed by an investigation of 32,000 twins in Connecticut, United States, born from 1930 to 1969; however, this same study found that the frequency of x-ray exposure was 2.4 times as high in twins who suffered childhood cancer as in a fourfold greater set of matched control twins [H11].

161. The Connecticut study was of twins who had received a dose estimated to range between 0.0016 and 0.04 Gy, with a median of 0.01 Gy. After follow-up to 15 years of age, the crude relative risk associated with pre-natal exposure was 1.8 (95% CI: 1.4-1.9), and even after adjustment for confounding factors which could be studied in the sample, the relative risk range was 1.4-1.9. The relative risk for leukaemia was 1.6 (95% CI: 0.4-6.8) and for all other cancers of childhood 3.2 (95% CI: 0.9-10.7). While the magnitude of the confidence intervals shows that not all of these results are significant, the data agree generally with those from the British and other United States studies, suggesting that the effect is real, even if a radiogenic cause cannot directly be proven.

162. T65 data from Japan [I1] reveal no dose-response pattern in those resident in Hiroshima and Nagasaki, although the zero-dose group had a higher rate of leukaemia than the not-in-city group. Further, the highest risk group, which had received 0.5 Gy or more as foetuses, showed no leukaemias. Hence, there is no evidence for an excess risk. Because subsequent analyses in Japan have continued to confirm this (and for leukaemias these analyses now include follow-up through 1979, i.e., adult ages as well as childhood [I1]), and for other reasons related to various aspects of the data available to Stewart and Kneale, the findings of the latter have come under frequent criticism. Most trenchant has been Totter and MacPherson [T2], who showed that the Oxford survey's x-rayed control individuals had not been similar to controls who had not been x-rayed in regard to confounding social and biological variables (e.g., social class, birth order and age of mother), and that, therefore, the estimates of relative risk derived from these retrospective data, while valid in regard to the association they show between various factors, including foetal irradiation, do not demonstrate a causal relationship between prior irradiation and cancer. These criticisms have received response [K4, T3]. There are also problems with the effects-measure and the dose-response data. First, relative risks of radiation carcinogenesis, for similar estimated dose, have declined over historical time, for unknown reasons. Second, dose estimates are not directly available, and crude, uncertain measures (i.e., number of exposures) have been used as a surrogate.

163. In sum, these studies have shown an association of childhood cancer with prior irradiation but have left doubts about the nature of the sample, especially in light of the Japanese observations, so that one cannot be certain that radiation is involved. On the basis of all the evidence, a selective factor of susceptibility to general ill health in the exposed seems unlikely to explain the result, although limited medical care in Japan just after the war might account for some of the difference in the findings [I1]. Another study, in the United States, by Monson and MacMahon [M8] lends some support to the general conclusions of Stewart and Kneale by replicating some of their results. These authors compared exposure in a population sample of all births with that in a sample of retrospectively ascertained children who died of cancer, i.e., it did not rely on retrospective matching of controls.

164. In particular, the kinds of confounding noted by Totter and MacPherson were considered, but not found, in the Monson-MacMahon study, and there is no proof of better access to medical care for those destined to develop post-natal cancer, as Totter and MacPherson hypothesized. Thus, while there are problems with these studies, they suggest a 1.4 or 1.5 cumulative relative risk of prenatal exposure, with no reliable data on dose-response patterns. The relative risk of solid tumours is slightly, but not significantly, less than that of leukaemia. A study in Finland [S7] disclosed a comparable leukaemogenic effect, but it was not statistically significant. Other studies with similar findings include [D3], [M9] and [S8]; two small surveys [C3, O1] reported no increase but were not large enough to exclude a 40% excess.

165. Several other factors are of interest. The greatest effect in the Stewart and Kneale survey seemed to have been in the first trimester, and the discrepancy between their data and the atomic bomb data has been attributed to the greater cell-sterilizing effect of high, early prenatal doses [M7] or to radiation-induced immune deficiencies (e.g., [I1], [K3], [K5] and [U1]). However, there was an excess of overall mortality in Japan only in those who had been irradiated in the third trimester of their gestation [K6], but these children had no excess cancer [I1], and these are the foetuses most likely to survive irradiation. A bias does not seem likely, therefore, in the Japanese data. Monson and MacMahon [M8] showed that the only radiation excess was in those leukaemic individuals who had been irradiated in the third trimester of their

gestation. Hence dose-effects or gestational-age-effects are difficult to show or to assess, and no unmistakable pattern has been seen. It is also of relevance that dogs do not show the specific trimester effect. There remains the possibility that concomitant sources of variation, and not irradiation, produced the cancers. These problems are discussed in detail in the UNSCEAR 1986 Report [U1], which concluded that there was no firm evidence of a trimester effect.

166. Another relevant question is whether these data afford a basis for inferring a genetic susceptibility to radiation carcinogenesis. Such a susceptibility might be expected, or is at least plausible, given the known genetic susceptibility to perinatal cancers such as retinoblastoma and Wilms' tumour. Kneale and Stewart [K3, K5] found a correlation between childhood diseases and cancer in those who had been x-rayed, perhaps indicating susceptible genotypes. The fact that this was not observed in those who had not been x-rayed does not offer strong support for a genetic hypothesis; instead, it suggests that the irradiation may have had an immunosuppressive effect (but, see [L18]). Genetic susceptibility would be difficult to demonstrate, however, if such genes are rare and the probability of cancer, even in those individuals, is small.

167. The BEIR 1980 Report [C4] concluded that there was probably a cumulative relative risk of 5.0 for a first trimester exposure and 1.47 for later trimester exposures, with the increased risk appearing as tumours prior to 12 years of age for leukaemias and 10 years of age for solid tumours. The risk was estimated at 25 excess fatal leukaemias per 10,000 exposed children per PYGy, and 28 excess fatal cancers of other types. These estimates must be viewed circumspectly for a number of reasons, including (a) the lack of clear effect of gestational age on the occurrence of leukaemia and the small effect for other tumours in the study by Monson and MacMahon [M8]; (b) the fact that these authors found only a 1.06 relative risk in recent data for solid tumours; (c) the declining radiation risk over time observed in the Oxford survey; and (d) the finding of later cancer risk in survivors, now adults, exposed in utero in Japan [Y8]. Finally, although there had appeared to be a linear dose-response relationship at least down to doses between 20 and 25 mGy [S5], this, too, is now suspect, for it was not found by Monson and MacMahon [M8] and the Kneale and Stewart estimate was based on heterogeneous data in which exposures were not accurately known.

168. While it is not strictly classifiable as an in utero exposure, the exposure of a mother prior to conception may also provide information about radiation cancer risks. A considerable amount of work has been done in animals along this line (see [B5] and [U1]), but only a little in humans. The human data [G1, S8, S9, U1] have shown a significant excess of malignant tumours among the offspring of individuals irradiated prior to conception (even prior to marriage [S9]), though the effects are not large. The relative risks are about double the expected cancer rate. Again, these data are not consonant with data on the offspring of exposed parents in Japan. Here, within a cohort of 52,725 individuals followed prospectively, 50,689 of whom have individual T65 dose estimates, 36 deaths were attributed to leukaemia through 1979, of a total of 3,552 deaths [11, S22]. The frequency of death due to leukaemia is not functionally related to the sum of the parental exposures; indeed, Ishimaru found the standardized relative risk of the offspring of parents collectively receiving 0.01 Gy or more of gonadal exposure to be 0.8. Ten leukaemia deaths were observed where 11.9 had been expected [11]. There was, moreover, no indication of an increase in any of the solid tumours of childhood [S22]. These studies have, of course, relatively low discriminatory power; it was estimated, for example, that 23 cases of leukaemia would have had to have occurred among the 16,713 offspring born to exposed parents for a radiation effect to have been demonstrated.

169. It is appropriate to summarize the conclusions offered by the UNSCEAR 1986 Report [U1]. The lack of an effect on early childhood cancers even after higher doses in Japan is a ground for considerable caution in interpreting the positive effects from the medical irradiation studies, especially in light of the many possible confounding or biasing factors. In a similar way, the fact that early post-natal irradiation in Japan showed carcinogenic effects only many years later (agreeing with other data on post-natal radiation effects), whereas the medical series showed their effects soon after birth, raises serious radiobiological problems if both observations are strictly the result of radiation. The doubts are enhanced by the absence of serious effects in animal experimental data. Also, the constancy of relative risk values for many cancer sites in the medical series is at variance with other data that suggest site-specific effects. Finally, haematopoietic stem cell differentiation does not occur during the first trimester of foetal development, so that the excess leukaemogenic effect in first trimester irradiation is difficult to understand.

170. It is patent that the existing data cannot resolve the question of pre-natal irradiation with clarity or much confidence. However, it would be prudent to assume that pre-natal irradiation does have an effect, especially with regard to leukaemogenesis. If pre-natal irradiation is reduced or largely replaced by ultrasonography, the problem may become less critical, but it would be incorrect to assume that this reduction or replacement will be an immediate, world-wide phenomenon or that accidental or industrial pre-natal exposures will not occur. Thus, it is important to continue to collect data on this point.

2. Exposures in childhood

171. The age at exposure to radiation may have a profound effect on the susceptibility of the individual to the induction of cancer, especially in those ages that are known to be characterized by high rates of stem-cell proliferation. One of the most obvious of these cases is exposure in childhood. While in a sense childhood is a continuation of the in utero growth and

development (which would make childhood experience merely an extension of the in utero experience), in fact there may be different sensitivities in childhood and in utero.

172. There are several sources of data on childhood exposure, but four predominate: (a) children exposed to therapeutic radiation to treat other primary cancers; (b) children exposed for the treatment of non-cancerous diseases; (c) children exposed to radiation from the atomic bombs, to fallout from nuclear weapons tests or to accidental discharges from nuclear power reactors; and (d) female children whose breasts were exposed to radiation in childhood and peri-menarchial ages. These groups may not be comparable directly, because the first group is largely composed of individuals with a genetic predisposition to cancer. The groups will be considered separately, although risk summaries for the cancer-treated group and all other groups will be presented.

(a) Children exposed to radiation for treatment of primary cancers

173. Though the number of such cases is small, because childhood cancers are infrequent, second primary malignancies have occurred in children who received radiation for the treatment of an initial cancer. These populations are of interest because they contain a high fraction of children who are genetically susceptible to cancer. These individuals may be distinct from the general population of children. They are all, however, children in whom actively growing tissues had been subjected to high doses of radiation.

174. There have been two major studies of the risk of second primary cancers in children irradiated for cancer: one, by Li and colleagues, who used data from the United States National Cancer Institute [L1, L2]; and the other, a survey of data from the United States, Italy, Federal Republic of Germany, Canada, England, France, and the Netherlands, known as the Late Effects Study Group [M10, M28, T4, T17]. Both studies suggest a 15+ relative risk of a second cancer of any site (i.e., pooling data on all primary sites and counting all secondary sites), with a 3-12% or more cumulative probability of cancer by age 25. About 68% of all second tumours in the Late Effects Study Group survey developed in the field of the original irradiation. The median latency time was 10 years, but only 5 years for second tumours not associated with radiation. Retinoblastoma and nevoid basal cell carcinoma patients, however, had a reversed latency pattern. Good dose-response information is generally not available from these data.

175. Tables 15 and 16 provide basic data from the Late Effects Study Group [M10, M28, T4]. These Tables show two concentrations: (a) that of second tumours in patients originally treated for tumours of genetic origin, i.e., children probably genetically susceptible to cancer; and (b) that of primary tumours in sites known to be radiogenic (haematopoietic, bone). The presence of a primary tumour may imply that the child is especially radiosusceptible. Second tumours were frequently associated with radiation in children known to be genetically susceptible to cancer: of 96 second tumours in such children, 42 (44%) were radiation-associated [M28]. The expected rates were based on the United States Connecticut Tumor Registry, although the cases were international. Hence, while the qualitative conclusions seem sound and agree with what is known genetically and biologically, the details of the results may not be precise.

176. In a study of the leukaemogenic risk posed by therapy in these children, it was found that the use of alkylating agents was associated with elevated risk while the use of radiation therapy was not [T18], consistent with the findings of other studies summarized in this Annex.

177. In a detailed study of bone sarcomas in the Late Effects Study Group, Tucker et al. [T17] found that among the 9,170 children in the analysis, the relative risk of bone sarcoma was 133 (95% CI: 98-176). There was a 20-year cumulative risk of 2.8%, or 9.4 cases per 10^4 PY. A comparison was made between 64 of these children for whom detailed treatment data were available and 209 children who had not developed bone cancer by the time of the study. The radiotherapy relative risk was 2.7 (1.0-7.7), after mean doses to the bone site in question estimated to be 27 Gy; there was a sharp dose-response gradient reaching a relative risk of 38.3 with doses above 60 Gy but decreasing above 80 Gy, and an overall excess relative risk of 0.0006/Gy. No excess risk was detected with doses under 10 Gy. Eighty-three per cent of the tumours were within the field of radiation. Osteosarcoma had the highest relative risk value, followed by chondrosarcoma.

178. An investigation of 64 cases of second cancers in children in the Federal Republic of Germany found mainly osteosarcoma, thyroid cancer, and acute non-lymphocytic leukaemia (ANL); 36 of 50 irradiated children manifested their second tumour in the irradiated field; a separate chemotherapy effect was not seen [G14].

179. Overall, these studies indicate that the general range of absolute risk per unit dose of a variety of second tumours is within about a single order of magnitude and much less than, for example, the variation in absolute risks for a comparable set of adult-onset tumours. Solid as well as haematopoietic tumours resulted from this irradiation, and they affected many organs. Although interpretations based on heterogeneous data collected retrospectively from a widely dispersed group of hospitals must be guarded, it is probably safe to assume that there is a general radiation-induced risk of second tumours in children.

180. To determine whether orthovoltage (140-500 kV peak range energy) or megavoltage (10 MV energy) therapy is safer, a study was made of 330 children in Minnesota, United States, who had been given megavoltage therapy [P16]. Only second tumours arising five or more years after irradiation were considered, and all children known to be genetically susceptible were excluded. The 30-year cumulative risk of cancer was 9.6%; this is somewhat less than the orthovoltage

risks [L1]. Nine of 14 second tumours were within the radiation field; five children had received chemotherapy as well, and two tumours developed outside the radiation field in these children. Thus, the apparent radiosusceptibility in children treated for primary cancer cannot be ascribed solely to chemotherapeutic effects or to genetic susceptibility.

181. In the Late Effects Study Group osteosarcoma study [T17], mega- and orthovoltage therapy had similar risks. There were also comparable risks following chemotherapy in these patients, but the radiation was shown to have risks independent of chemotherapy.

182. Table 17 provides details of the cancer risk following irradiation for three cancers of which a substantial fraction are known to be familial (retinoblastoma, Wilms' tumour, Ewing's sarcoma). Most of the subsequent tumours are sarcomas, especially in irradiated bones; however, carcinomas can arise in heavily irradiated epithelial tissues, as the Wilms' tumour data disclose. The general range of risk is 1-3% at 10 years post-exposure, rising to 15-35% or more after 30 years, based on life-table methods of calculation. One can assume that virtually all of the in-field tumours were radiogenic; but the spontaneous rate in these patients is also quite high, sometimes nearly as high for all sites combined as for the irradiated field. Hence, radiation interacts with host susceptibility, perhaps causing it to be expressed in the specific target field.

(i) Treatment for retinoblastoma

183. Retinoblastoma is an embryonal tumour of the retina, usually occurring at, or shortly after, birth. It is easy to diagnose and is restricted in age, largely because it arises in a tissue where mitosis ceases at about the time of birth. Though it is a rare tumour, its epidemiological importance has motivated several studies of patients in major referral centres. One of the interesting facts about this tumour is that approximately 25% of cases are of a heritable kind; this is detectable either because the initial presentation is with bilateral and/or multifocal disease or because other family members are affected [V1]. Epidemiological and molecular data have shown convincingly that this heritable form of the disease is attributable to the inheritance of a single damaged gene region on chromosome 13; retinal cells which then suffer a somatic mutation at the same region on the homologous chromosome are malignantly transformed. The tumour cells are clonal descendants of the first transformed cell.

184. Osteosarcoma is a tumour to which genetic retinoblastoma patients are known to be at high risk as a second primary malignancy [F2, V1]. Studies consistently show that about one third of the second primary tumours in these patients are osteosarcomas; the remainder consist of soft-tissue sarcomas, brain tumours, leukaemias, and melanomas. Osteosarcomas may occur in the irradiated field, but will spontaneously occur in distal, non-ocular sites not subject to radiation therapy and in non-irradiated patients. While other malignancies are seen in retinoblastoma patients, about 80% of all second tumours occurring later in childhood or early adulthood are osteogenic sarcomas. At present it seems that these cancers result from the production of a second mutation in an exposed osteoblast. The osteoblast becomes homozygous for the chromosome 13 gene deletion, just as the retinal cell does, leading to the neoplasm [D4, D19, H18]. This could even occur in individuals who do not carry the familial retinoblastoma gene [D19] and is being seen in some other radiogenic second tumours (osteosarcomas of the orbit [S41]). Because osteosarcoma occurs even in non-irradiated bones, bone growth and development must involve the same genes involved in the development of the retina. The concentration of osteosarcomas in these patients during childhood and early adulthood shows that the tumours occur during the normal cell proliferation periods for this tissue. It is now known that these tumours are generally caused by somatic recombination, leading to cellular homozyosity for the deleted or inactivated gene.

185. Family members of retinoblastoma patients who do not themselves manifest that disease seem to be somewhat more susceptible to other cancers than the general population, although the reasons for this are at present unclear [C5, S12]. There are no data on radiation effects in such individuals as yet.

186. A Japanese study of 2,609 cases of childhood cancer has found roughly similar results [T12]. Seventeen of 50 second cancers were in retinoblastoma patients, and most were related to radiation therapy. Of all second tumours, haematopoietic, bone, and thyroid made up the bulk. Thus the pattern is present in all ethnic groups that have been studied to date.

187. In several general studies, the risk of radiogenic second primaries has been estimated to be 1-15% in 20 years of follow-up [A8, F2, L1, T4, V1]; since the population risk of an osteogenic sarcoma of the orbital area (or even of the skull) is very low, the radiogenic risks are well over 100 times the baseline risk in most estimates. Current dose rates are 35-60 Gy, using megavoltage equipment; this has been reported to reduce the risk to 1-2% (see [F2] and [V1]), which is lower than the risk associated with orthovoltage equipment. It is not clear whether there is a cell-sterilization effect.

188. In retinoblastoma, not only genetic susceptibility but also concurrent chemotherapy must be considered. The literature is consistent in finding that almost all independent second tumours have been observed in familial cases [A3, D15, F2, V1]. The effect of combined therapy is discussed in section V.C, where it is shown that an excess risk is experienced, at least in genetic cases [D15].

189. Two studies warrant special discussion [A3, S26]. Abramson et al. [A3] followed 693 patients with bilateral, presumably heritable, retinoblastomas and 18 patients with unilateral retinoblastomas. These data are summarized in Table 18. Although previous estimates from this series on the risk of second tumours have been between 10% and 15%, they did not fully account for the variable length of follow-up. If the time-specific incidence rates are computed, using

the number of new cases divided by the number of individuals not yet affected up to the time of follow-up, time-specific cumulative risks can be calculated and a life-table cumulative incidence obtained. With this method, Abramson et al. found a mean latency of 10.4 years. For individuals undergoing neither radiation (or with second tumours outside the irradiated field) nor chemotherapy (and yet surviving, having been treated by enucleation alone), the incidence of tumours was 10% after 10 years, 30% after 20 years, and 68% after 32 years.

190. For all patients, the risks of second tumours were considerably higher. After 10 years, the risk was projected to be 20%; after 20 years, 50%; and after 30 years, 90%. This is much higher than reported in other instances, at least partially because of the method of computation which accounts for differential follow-up and at-risk periods. These results need to be confirmed. Tumours that developed outside of the radiation field (or in non-irradiated individuals) had significantly later ages of onset than those in the field.

191. By comparison, the Late Effects Study Group sample of retinoblastoma patients exhibited a 14% cumulative risk after 20 years [T17]; this group included a mixture of heritable and spontaneous cases, which may account for some of the difference with the Abramson study. It found that the relative risks of second bone cancers following primary therapy for retinoblastoma were similar to those following comparable therapy for other childhood cancers, presumably because the baseline bone cancer risk is higher in patients with retinoblastoma than in patients with other types of childhood cancer. That is, retinoblastoma patients have higher baseline osteosarcoma rates, so that a larger numerical excess number of cases produces only a modest relative risk.

192. Dose effects have been estimated in a general way for the Abramson study data. Thirty-seven patients received orthovoltage therapy only, with doses ranging from 3.5 to 260 Gy. Thirty-five received betatron radiation (3.5-120 Gy). Fifteen received combinations of these with some other miscellaneous treatments. Life-table analysis showed that the risks of second cancers following one course of betatron radiation (3.5-4.5 Gy) were not different from those following multiple courses (70-90 Gy). Similarly, patients treated with orthovoltage doses of less than 110 Gy had the same life-table risks as patients treated with more than 110 Gy.

193. In the second study [S26], Sagerman showed a 2.5% risk of second tumours for patients receiving 60 Gy. 5.5% of those receiving up to 110 Gy, and 32% for those receiving more than 110 Gy. The variability in follow-up times and the fact that those patients receiving higher doses had longer follow-up complicate the interpretation of these results. Lower doses have been used more recently, and hence have shorter follow-up times. The life-table methods of Abramson et al. did not demonstrate a difference related to dose. Hence, it is not clear how much dose reduction, fractionation or energy level of the therapy affects the risk in retinoblastoma patients.

194. A study of 882 retinoblastoma patients in Britain reported results that were qualitatively similar but that, quantitatively, showed less risk [D15]. In this series, 30 second malignant neoplasms were seen. The results are presented in Table 19. Twenty-six of the second tumours occurred in the 384 patients with genetic retinoblastoma; using life-table methods to account for differential follow-up times, the risk of a second tumour was estimated to be 8.4% after 18 years (6.0% for osteosarcoma alone). Of these 26 tumours, 12 had developed outside the radiation field (a risk of 3.0% after 18 years), all of them osteosarcomas (2.2% among those with neither radiation nor chemotherapy, i.e., the baseline risk level). The British population risk of osteosarcoma by age 18 years is about 10^{-4}, so that the relative risk in genetic retinoblastoma was 200 (95% CI: 50-500).

195. Within the radiation field, where second tumours are probably radiogenic, the risk in the genetic cases was 6.6% after 18 years. Average doses were 35-40 Gy, with little variation. While the radiation effects in these data are not dose-specific, they do indicate the nature of the risks. The follow-up period was shorter than that reported by Abramson et al. [A3]; otherwise, the difference in risk is probably due to different efficiencies in the ascertainment of secondary neoplasms. Whether the true actuarial risk is as high as Abramson et al. suggest, or somewhat lower, retinoblastoma patients are obviously at very high risk of secondary neoplasms.

196. A brief report by Koten et al. [K27] has given an estimate of 19% for second tumours after 35 years in patients in Holland. This is intermediate between the other studies.

197. All of these children were irradiated at or near birth, that is, at about the same age, so there are no data on the effect of age at exposure and no effort has yet been made to project the lifetime risk, although the two major studies do offer some indications. In Abramson et al. [A3], the cumulative incidence curve in those without radiation effect (but who are genetically susceptible) is slightly concave upward from birth to age 32, the maximum number of years of follow-up. The curves for tumours inside and outside the radiation field show similar shapes through age 25, after which the data are sparse. If this finding is correct, then radiogenic tumours are occurring at about the same ages as spontaneous tumours in these children, as is the case with radiogenic adult-onset carcinomas generally (as will be seen below). The cumulative increase rises non-linearly with age of follow-up, suggesting at least crudely that a multiplicative risk projection is more applicable than an additive projection, again in agreement with most adult cancer patterns. The non-linear cumulation of risk is also apparent in the study by Draper [D15], although less detail is given. As shown in Table 19, the absolute life-table probability of second cancers rises by a factor of two in the years 12-18, relative to the increase in the 12-year interval 0-11. This is true for radiogenic as well as non-radiogenic second tumours.

198. It should be stressed that retinoblastoma patients constitute the only significant group of human beings

in which the carcinogenic process can be observed, after radiation, in exposed cells in which (a) radiation is in a sharply delimited area and can be compared with the same area without irradiation and the rest of the body; (b) the cells at risk are effectively haploid in regard to cancer risk; and (c) all exposed individuals are of essentially the same age. The subsequent projection effect should differ in ways that are informative relative to multi-stage models, because only one second stage is needed for cancer to occur in these individuals. It would be useful to know how the effect of cell sterilization, which must occur in the periocular region after intense irradiation, manifests itself in regard to (a) the dose-response relationship and (b) the probably smaller number of carcinogenic events needed to take place as a consequence of irradiation.

(ii) Treatment for Wilms' tumour

199. There has been considerable work, mainly by Li and colleagues (e.g., [L3]), on the risk of a second neoplasm after radiation treatment for Wilms' tumour of the kidney in childhood. After nephrectomy, patients receive radiotherapy to any areas that might be the site of further tumours or potential metastases, usually the renal fossa, elsewhere in the abdomen, or in the thorax. In 487 patients and 4,255 person-years of observation, Li et al. observed 11 second tumours, with latent periods ranging from 7 to 34 years. Most patients were given orthovoltage therapy with average doses of 25 to 30 Gy to the abdomen; other sites received varying doses. There was a second tumour in 2.8% of the patients. Nine of the second malignancies were solid tumours and occurred in areas that had been given 6-40 Gy. One was in a non-irradiated area. There was one case of acute myelogenous leukaemia. The cumulative risk of a second tumour was 18% (SE: 6%) in 34 years after diagnosis of Wilms' tumour.

200. The relative risk of radiation itself was not estimable (i.e., was not finite), since no case occurred in the 75 non-irradiated patients. Another study has yielded similar results, with 2.3% of cases having a second tumour [S10]. A third study reported a variety of sites for second tumours, including the thyroid, gastrointestinal sites, and bone, all with relative risks over 80 [T4]; leukaemias were increased 13 times. Many of these sites, e.g., the thyroid, are unlikely to have been in the intended irradiated field, and the authors do not report radiogenic cases separately. Wilms' tumour, too, may be heritable in a large fraction of cases. Its controlling gene region, along with the gene for insulin and the H-ras-1 oncogene (rat sarcoma), is on chromosome 11 and, as is true for retinoblastoma, somatic homozygosity at the relevant loci may be sufficient to produce the disease.

(iii) Treatment for Ewing's sarcoma

201. Ewing's sarcoma is a form of bone cancer, typically treated by local irradiation and chemotherapy. Lately, long-term survival rates have improved, and several studies have demonstrated a substantial risk of secondary osteosarcomas in the irradiated field [G2, S11, T4]; there may also be a risk of leukaemia [T4]. These patients received 44-55 Gy, followed by a 10-15 Gy boost to a reduced field, over a six-week course of administration [S11]. Patients receiving orthovoltage radiation were ordinarily treated without chemotherapy, whereas those receiving megavoltage radiation had combined therapy. The increase in risk is consistent for all studies. A dose-response of 7.2 cases per 10^4 PYGy and a cumulative risk of 35% over 10 years have been estimated [S11]. There also seems to be an increased risk associated with combined therapy, i.e., newer modalities may have increased the risk of secondary malignancies. The evidence suggests that bone tissue is susceptible to carcinogenesis if it is irradiated before or during the adolescent growth spurt.

(iv) Treatment for Hodgkin's disease

202. Some information exists on the risk of second tumours after irradiation in childhood for the treatment of Hodgkin's disease. Although data from the treatment of this malignant disease in children are limited, the radiosusceptibility of certain tissues, specifically, bone, haematopoietic, and thyroid, has been reported [T4]. However, in children treated with radiation alone, solid second primary tumours have occurred, leukaemias occurred only when chemotherapy or combined radiation and chemotherapy had been given [M28]. This is consistent with findings in adult Hodgkin's patients. There was no indication of a familial susceptibility to Hodgkin's disease in these 40 children. Dose information is not available.

203. To summarize, the evidence from children irradiated to treat a primary cancer shows very high susceptibility to second cancers and promises to be informative concerning the nature of the genetic changes caused by radiation and their relationship to carcinogenesis. However, since these children are probably not generally representative of the whole population, these results are not useful for estimating risk coefficients or for risk projection for the general population.

(b) Children exposed to radiation for treatment of benign conditions

204. There have been a multitude of uses of irradiation for the treatment of benign conditions in childhood. The exposures in question primarily involve (a) the head and neck, to treat tinea capitis (scalp ringworm) [M13, R1, R22, S16]; (b) the thymus, to treat what was thought to be a pathologically enlarged thymus [H1, S14]; (c) the neck, to treat tonsilar and nasopharyngeal conditions [S15]; and (d) various sites, but especially the head and upper body, to treat haemangiomas (benign superficial tumours usually present shortly after birth) [L10].

205. The tumours induced by these exposures are primarily leukaemias and cancer (as well as benign lesions) of the thyroid. Risks of these tumours attendant on exposure of the head and neck in children are summarized in Table 20. At doses of less than 1 to about 8 Gy, the absolute risk of radiogenic cancer varied from <0.1% at 10 years to about 8% at 30 years after irradiation for malignant neoplasms of the thyroid and was much lower for leukaemias. One

study [S27] has reported an increased incidence of basal-cell skin cancer in tinea capitis children; risk rates are difficult to calculate because of the complex ascertainment of cases, mixed ethnicity, and other risk factors, but among white children, there is an increase. Some aspects of this study are discussed in the sections on host factors and on the interaction of irradiation with environmental factors. Other cancers have been reported as well: brain and parotid tumours have arisen in small but excess numbers in the tinea capitis series and bone sarcomas have developed in children treated with radium for bone tumours.

(i) Thyroid and thymus exposures

206. The thyroid gland is susceptible to radiation-induced cancer, especially when exposure occurs in the first two decades of life. The effect of internal irradiation in children exposed to ^{131}I in the Marshall Islands, where a special age effect was not seen, will be discussed later. The risk coefficient estimate from many studies is consistently in the range of 1.6-9.3 cases per 10^4 PYGy [C4] in mean doses which ranged from 0.09 to more than 10.0 Gy (see [S13], [S38]) in childhood exposures. These values are based on thymus irradiation [H1, S14, S38], on tonsilar x-irradiation [S15], head and neck exposure [M13], and on the follow-up of the tinea capitis patients [R1, S16], as well as on the Japanese data. A summary estimate based on data from North American childhood exposure to under 15 Gy was 2.5 cases per 10^4 PYGy for exposure under age 18 when both sexes were pooled [N5]. Animal data and the Japanese atomic bomb experience suggest about a twofold higher susceptibility for childhood exposure (under age 18) [N5]. There is a marked excess of cases in females, in general 2-3 to 1 [N5, S13], uniformly across many studies; this ratio is similar to the sex ratio in spontaneous cases and does not indicate a particular radiosusceptibility in females [S13].

207. Several other studies of cancer after miscellaneous exposures of the thyroid have reported similar results [B6, C4, D5, M12]. Jewish children may be especially susceptible [C4, S13, S14, W11]; when matched to comparable non-Jewish children, the relative risk has been at least as high as 3.5 [S14, W11]. Whether this is an artefact of the exposure regimen or is due to other factors is not certain.

208. Because spontaneous thyroid cancer is quite rare, it is likely that most of the observed tumours are radiogenic. The bulk have been of the papillary type [C4], and there appears to be a latency period of at least 5 years [C4, S13], perhaps with a peak excess risk about 20 years later [C4], though excess risk has been seen to persist up to 40 years after exposure [S15, S38]. Consistent with studies of specific radiation exposures, a retrospective examination of female thyroid cancer patients (i.e., ascertained because of thyroid cancer not because of irradiation) showed an increased risk with radiation and with younger age at exposure, a relative risk of 16.5 overall, but 42.2 for exposure at less than 20 years of age [M14].

209. The best dose-response data come from Japan [W5] and from a study of the thymus-irradiated children in the population around the city of Rochester, New York, United States [S38]. In the latter instance, a cohort of about 2,650 children irradiated from 1926 to 1957 and 4,800 sibling controls were followed for about 30 years. The doses ranged from 0.05 to 11 Gy, with 62% having received less than 0.5 Gy; the mean was 1.2 Gy. Those who had received higher doses were treated earlier in the time period and hence have a longer follow-up. The dose-response data are summarized in Table 21. Recently, the pathology of 75% of the reported benign thyroid nodules was studied, and many were reclassified as non-neoplastic, thus sharpening the dose-response estimates. Almost all of these children had been irradiated in their first year of life. In many ways, this makes the cohort similar to that of the retinoblastoma patients (who exhibit no excess of thyroid cancer following radiation of the orbit). The relative risk, after 29 years of follow-up, was 49.1, based on sibling controls, and the SMR was 44.6 (based on New York State cancer rate data); for benign thyroid adenomas the relative risk was 15.0. These values are significant at much less than the 0.001 level. They also show the sensitivity of the relative risks to the choice of controls.

210. Figure I indicates the dose-response pattern. An additive model yielded 3.46 ± 0.82 excess cases per 10^4 PYGy; the sex difference was marked with risks of 5.25 per 10^4 PYGy for females and 2.05 cases per 10^4 PYGy for males, which is similar to estimates from other studies. In Japan, exposed persons under age 19 showed a value of just over 2.1, with a sex ratio of about 2.9 (derived for all ages) [W5]. The dose-response pattern fitted a linear model ($P < 0.0001$), not improved by adding a quadratic term. The authors also fitted a relative risk model to their data, based on the Cox regression method. This fitted well ($P < 0.00001$) with a linear dose term (not improved

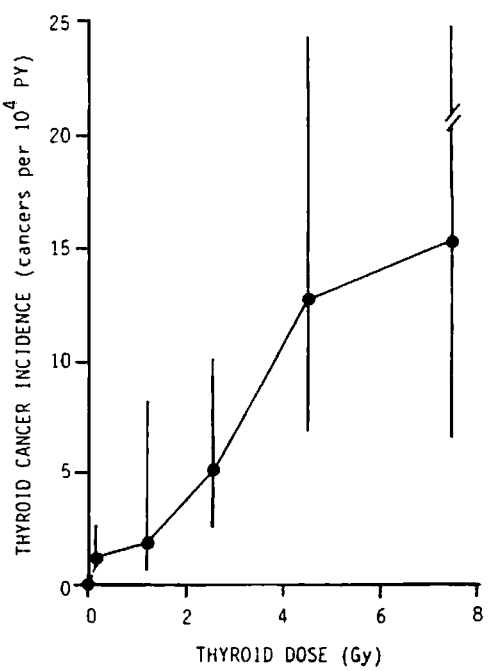

Figure I. Thyroid cancer incidence in relation to thyroid dose. [S38]

by adding a dose-squared term), and the relative risk estimate after exposure was 1.58 times the prevailing risk. Data for benign adenomas were similar, both qualitatively and, to a great extent, quantitatively; the relative risk was 1.44. Figure I yields a risk coefficient similar to the figure from Japan, for lower doses, but in the Japanese data the risk falls after about 3.5 Gy.

211. This study [S38] also allowed a dose-fractionation assessment. Such data are rare in general, and the results are summarized in Table 22. In sum, the low-dose per fraction group had higher risks per Gy, which is not consonant with the decreased effect of dose fractionation that has commonly been theorized. The results were the same for adenomas (not shown in the Table). Thus, neither the number of fractions nor their dose was protective.

212. Finally, Shore et al. [S38] examined the projection effect in this cohort. The excess in thyroid cancer began 5-9 years after irradiation and seems to have continued even after 40 years of follow-up (i.e., in patients irradiated in the 1920s). However, there appears to have been a slight decrease in the excess after about 25 years (although adenomas continue to increase). The mean latency time was 29.3 years, with somewhat shorter latencies (27.4 years) for those having received the highest doses. The Japanese data also show a continuing effect after at least 35 years [W5]. These data follow the age-specific incidence distribution of spontaneous thyroid cancer in adults, which reaches a peak in early adulthood and changes relatively little thereafter.

213. This study agrees generally with the Japanese T65 data [P4] in suggesting a linear dose-response pattern for thyroid cancer in irradiated children (as well as adults). Data from tinea capitis patients are similar in this regard, although the slope appears to be steeper. Japanese data [P4, W5] suggest a higher risk in childhood than adulthood. In North American children having received doses in excess of 15 Gy, however, a reduction of effect appears to occur, presumably as a consequence of cell sterilization [N5].

214. Although these thyroid neoplasms are of the relatively benign papillary type (and radiation effects include purely benign nodules as well) they may eventually pose a serious risk. Thyroid neoplasms of whatever aetiology can become life-threatening, and the mortality after 25 years of follow-up is about 7% [N5]. Thus, the risk in children may be substantial, and further time may be necessary to assess its actual level.

215. As was discussed briefly earlier, there has been speculation on whether radiation-induced thyroid cancers differ from naturally occurring thyroid cancers. It has been found that radiogenic thyroid cancers, somewhat more often than their naturally occurring analogues, tend to be of the papillary type, and to have metastatic lymph nodes [T13].

216. Some concern has been expressed about whether the apparent excess in the exposed individuals is a function of the detailed follow-up to which many of them were subjected. However, a radiogenic excess of thyroid cancer may also have been reflected even at the population level. In a study in the state of Connecticut, United States, based on its tumour registry, an excess of thyroid cancer was correlated with the childhood time period during which these exposures occurred [P5]. In several instances, the risk has continued to be elevated as much as 35 years after exposure, so that the lifetime excess in thyroid cancers may be more substantial than the very small and uncertain excess in the first decade or so after exposure.

(ii) Leukaemia as a general outcome in exposed children

217. At present, the evidence for excess leukaemia in exposed children comes largely from two sources: children exposed in Hiroshima and Nagasaki and children exposed to treat various benign conditions of the head and neck, specifically of the thymus and scalp (tinea capitis). The Japanese data are reviewed in section III.B.2.c.

218. Studies of children with scalp irradiation to treat tinea capitis [R1, R22] have produced results in accord with the Japanese studies. The relative risk of tumours of the head and neck was 3 in a matched case-control study of 10,834 cases in Israel, and for leukaemia, specifically, 2.3. The marrow dose was about 0.3 Gy, leading to an increase of 0.9 excess leukaemias per 10^4 PYGy. There was also a clear increase in brain tumours. Mean follow-up time was 26 years. Children of less than 10 years of age who were irradiated had higher risk than those exposed at older ages. Latency for leukaemias was 9.3 years. No other causes of death were elevated. Other studies have found comparable results [A4, M13, S16].

219. In a study on thymus-irradiated children there was a relative risk of leukaemia of about 3.1 [H1], though a dose-response relationship could not be estimated.

220. There are no precise estimates of the dose-response risks of leukaemia in children. The general range of risk for single or short-duration exposures combining data from all ages, over the 25-year risk period following irradiation was estimated in the BEIR 1980 Report to be between 0.01 and 2.2 excess cases per 10^4 PYGy, or 0.25-55 cases over the 25-year risk period [C4].

221. Approximate risk coefficients from [R1] can be derived as follows. Ron and Modan report 10 leukaemia cases in 10,842 irradiated children and seven in 16,241 non-irradiated controls. They report that the sex-adjusted risk of leukaemia was 4.0 cases per 10^4 in males and 6.0 per 10^4 in females. These persons were followed for an average of about 22.8 years, so that the number of excess cases was 0.18 per 10^4 PY in males and 0.26 per 10^4 in females. At an average dose of 0.3 Gy, this corresponds to 0.60 male and 0.87 female cases per 10^4 PYGy. These are crude estimates which assume that age is not a material factor, so that all person-years of observation are equivalent. They agree

roughly with the BEIR estimates. Because the results are not statistically significant, only the mean estimate is given, with no confidence interval.

(iii) Exposure of the central nervous system

222. The Israeli tinea capitis study [R1, R22] has found an excess of brain cancers, with a relative risk of 2.5 (95% CI: 0.9-7.4), with males appearing to be more susceptible. The dose to the brain was 1.2-1.4 Gy to the upper layer and 0.95-1.2 to the layer 2.5 cm below. The risk coefficient estimates, in excess cases per 10^4 PY, were 2.3 for irradiation less than four years of age, 7.5 for five to nine years, and 3.2 for ages above 10. Another study of irradiated children has produced a risk coefficient estimate of 2.9 ± 2.4 brain cancers per 10^4 PYGy from five to 22 years after exposure [L11]. No significant increase in intracranial tumours has been seen in Japan among any of the age groups [S23].

(iv) Exposure of the skin to treat haemangioma and other skin disorders

223. Many children were irradiated to treat haemangiomas, in general in visible areas of the skin, from roughly 1910 to 1960. Treatment was by ^{226}Ra implants. Over 20,000 of these patients have been followed in Sweden [F12]. Among 10,000 of these patients [L10], 75 cancers occurred during the registry period 1958-1979. Only 55.6 had been expected, based on the same registry and a national birth record system, yielding a relative risk of 1.35. Although this was a prospective investigation in that a cohort was identified as being at risk and compared with the nation as a whole, cases were ascertained retrospectively from a registry, and some cases may have been overlooked. A preponderance of tumours of the central nervous system, breast, and thyroid was found [L10, H22], confirming both the radiosusceptibility of these sites and the reliability of the data. Furthermore, the frequency of birth defects among the mothers in this study was close to that expected, suggesting that this was a representative cohort. The occurrence of thyroid malignancy supports other studies and indicates that over-screening bias is not the only reason that excess thyroid tumours have occurred in other children given radiation to the head and neck area.

224. There has been widespread use of radiation (generally of "soft" x rays) to treat acne in adolescents. Some of the doses were administered with cone shielding, to protect the thyroid and other areas; others were administered without such shielding. Data from the United States thus far provide no evidence that this use of radiation has led to thyroid or other cancers [G8]. The doses were fractionated, and the patients were generally 16-18 years of age or older, when the thyroid appears to be less radiosusceptible than in early childhood.

(c) Children exposed to atomic bombings and fallout

225. Several groups of children have been studied who were exposed to radiation caused by the detonation of nuclear weapons. The best documented are the children exposed in Hiroshima and Nagasaki and the children exposed in the Marshall Islands (the latter were primarily exposed internally, to ^{131}I). Three other groups have also received some attention; namely, children exposed to radioactive fallout from the distant atmospheric testing of weapons, children exposed to accidental discharges from nuclear power installations, and children exposed to relatively elevated levels of cosmic radiation by virtue of living on the Colorado plateau in the United States or at similar altitudes; the last-mentioned children have been studied only cursorily, e.g., all of them lived in industrialized countries and none lived at elevations as high as the Andean altiplano or the Tibetan plateau. The cancer risks resulting from these exposures are consistent with the risks from other exposures (reviewed above); there is a susceptibility to leukaemia, to thyroid cancer, and, in a special and only partially understood sense, to breast cancer (see below). In fact, the leukaemia risk has been satisfactorily studied only in Japan; other studies of it remain controversial.

226. *Thyroid.* The risk of thyroid cancer has already been reviewed in connection with other exposures, and some of the Japanese findings have been given. In Japan, about 3.6 additional incident cases per 10^4 PYGy were seen among females exposed before 20 years of age, as contrasted with one additional incident case among males in these same years. There is mounting support for a lessened susceptibility in those over 20-30 years of age at the time of the bombing [B5, P3, P4]. In the Marshall Island cohort, a group of 250 individuals that has now been followed for 30 years, the only substantial risk is that of thyroid cancer due to ingestion of ^{131}I, ^{133}I etc., with doses estimated to be 7-14 Gy in young children and perhaps 20 Gy in infants (< 1 year) [C5, C6]. No tumours were detected in the first 10 years of follow-up. Only one case of leukaemia has been seen. While there have been cases of benign thyroid nodules, it is uncertain how much excess of malignant thyroid cancer has occurred. Only papillary cases have arisen, and the relative risk of the non-metastatic carcinomas has been 0.82 for individuals exposed before 10 years of age and 3.94 for those exposed after this age (computed from [C5]). The risk shown in Table 20 is 1.8 per 10^4 PYGy for the children; curiously, this study is unique in finding a smaller risk in younger children, but this could be artefactual. The thorough search for thyroid anomalies has led to the surgical removal of most benign cases, perhaps sparing some individuals the risk of developing thyroid carcinoma. Furthermore, the long duration of the excess risk for this tumour means that increased risk may continue to reveal itself.

227. In an investigation of thyroid malignancy in children exposed to atomic testing near the Nevada test site, United States, there was an excess of cases relative to expectations based on data for the whole United States, but no excess relative to expectations for neighbouring states [Z3]. Sample sizes were small, and it was concluded that the numbers of exposed children might not be large enough, given current risk estimates for thyroid cancer, to demonstrate excess risk from nuclear-testing fallout.

228. The most recent report on the population of the Marshall Islands [H32] has found an inverse relation-

ship between the prevalence of thyroid nodules and the distance from the BRAVO explosion to the atoll of residence at time of exposure. The results suggest a linear dose-response relationship and an increase in the risk estimate by 33% to 11 cases per 10^4 PYGy.

229. Finally, a recent case-control study in Japan has found that irradiation of the head, neck, or chest in infancy to doses of 0.0002-0.4 Gy was the only identifiable risk factor for the occurrence of thyroid cancer in teenagers [Y7].

230. *Leukaemia.* The leukaemia data from children irradiated by the atomic bombings have recently been reviewed [e.g., C4, F3, P15]. Where it had once been thought that the distinction between Hiroshima and Nagasaki would be informative with respect to the different RBEs of high- and low-LET radiation, it now appears that there is little useful data in that respect, owing to the revision of the dose estimates. In Japan, the most informative data are the contrasts in risk after exposure between the 0-0.09 Gy and the > 1 Gy groups, under age 10. Figures II and III show the general risk pattern quite clearly for children and for adults (from [B24], based on T65 doses). The latent period is brief, beginning 3-5 years after exposure and reaching a peak 7-8 years later [F3], after which time the excess risk levels off; subsequent studies have shown that significant excess cases disappeared by about 1959; that is, after 15 years or so [O3]. The latency time is shortest in exposed children, i.e., it is inversely related to age at exposure [I7]. Most cases were of acute leukaemias, and these showed the most age-dependent subsequent risk; chronic granulocytic leukaemias showed very little age-dependence [I7]. The subsequent risk can be fitted adequately to a log-normal distribution [I7].

231. At a dose of 1 Gy, the relative risks for children (< 19 years) and adults have been estimated to be 6.2 and 3.3, respectively [P15]. Overall relative risks (i.e., for all doses > 1 Gy and all ages > 15 years at the time of the bombing) are 31-33 [S17]. The risk coefficient for Japanese children has been estimated at 2.8 additional cases per 10^4 PYGy (T65 kerma), with a lower risk in those exposed between ages 10 and 19 [F3]. This finding is similar to that for adults, except that the cell types in children have been of the acute lymphocytic and the acute and chronic granulocytic, which are typical of spontaneous childhood leukaemias. The highest sensitivity seems to have been for chronic granulocytic leukaemia.

232. For leukaemias in children exposed at less than 10 years of age, sex ratios were 0.76 (M/F) for doses < 0.1 Gy; 1.90 for doses 0.1-0.99 Gy; and 3.71 for doses > 1 Gy. For children 10-19 at the time of the bombing, the respective values were 1.05, 10.32 and

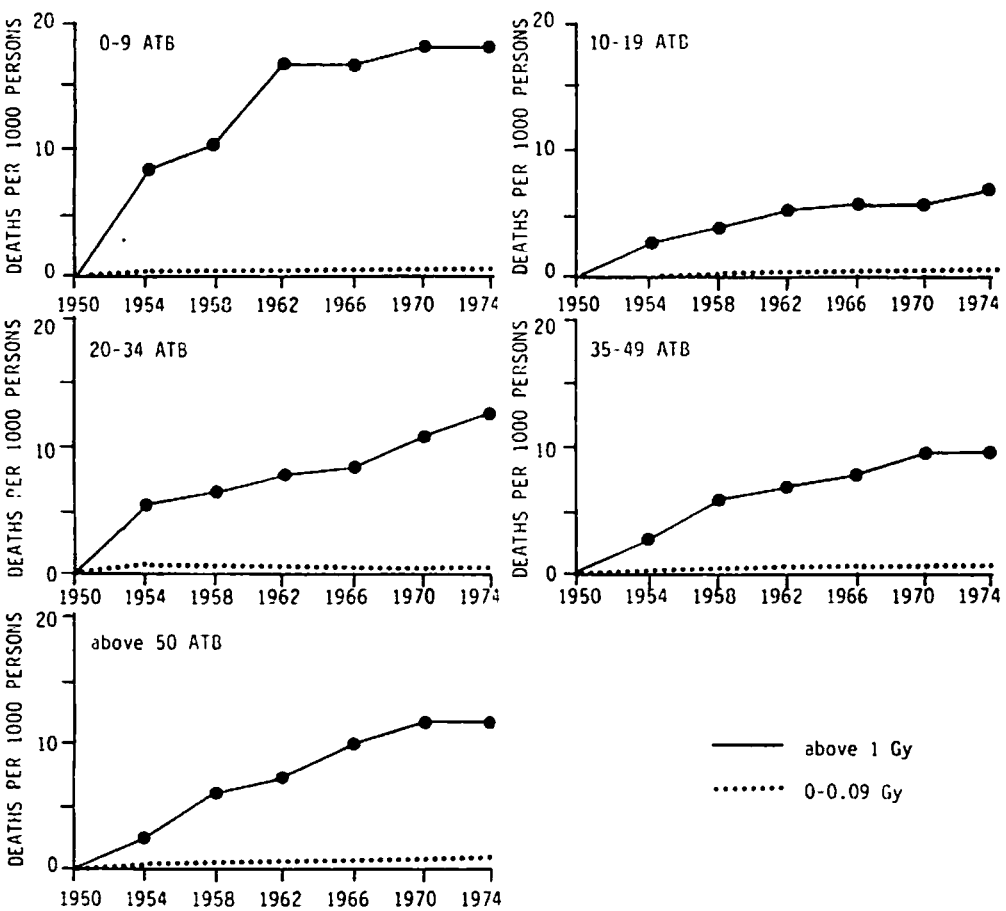

Figure II. Cumulative deaths from leukaemia, 1950-1974, per 1,000 persons alive in 1950, for various age groups at the time of the bombings (ATB).
[B24]

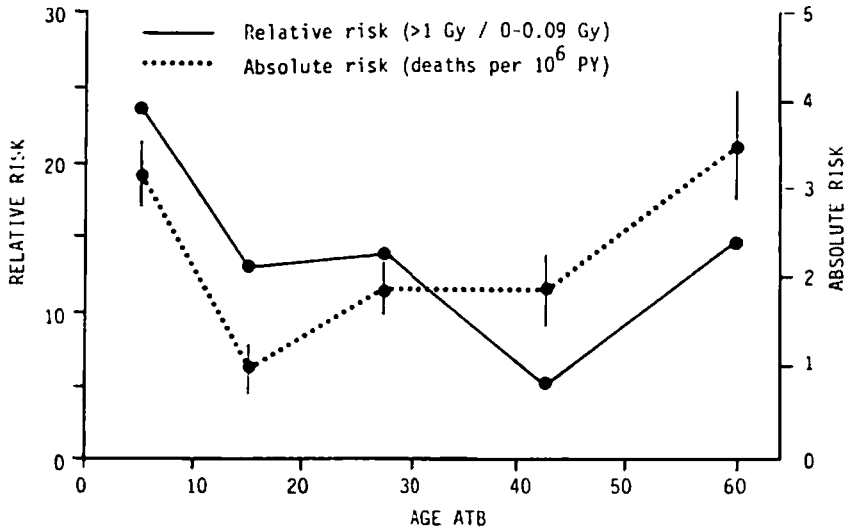

Figure III. Absolute and relative risks of leukaemia by age at the time of the bombings, 1950-1974, average for both Hiroshima and Nagasaki, T65DR doses. [B24]

0.84 [C4]. However, the background rates are higher in males, and the relative risk is not statistically different between the two sexes [P15], an argument favouring a multiplicative projection model.

233. There is no excess of childhood cancers other than leukaemia and breast cancer in these children. Evidence is now available on their later adult experience, but to date there are no new adult-onset tumour risks clearly identified with childhood exposure, with the exception, perhaps, of stomach cancer (4.7 cases per 10^4 PYGy) [S17, W5].

234. Most of the controversy in the United States on distant nuclear fallout exposures has centred on its possible leukaemogenic effect. Comparisons have been made for all childhood cancers and separately for leukaemias, based on mortality data for counties with high and low exposure before, during, and after fallout periods. It has been argued that the data supported an excess in the high-exposure areas of southern Utah [L4], which excess was correlated in time with the period of exposure to fallout and which was not evident in low-exposure areas. This interpretation has been criticized, in particular because the data are not compatible with a simple radiogenic effect; rates in different areas over these time periods do not vary simply as a function of exposure, suggesting that other factors are involved (e.g., [B6], [L5] and [R4]). Reviewing a broader set of data, Land et al. [L5] argued that in southern Utah the leukaemia rate has been lower than in other parts of the United States in years prior to the exposure. Further, there was a deficit in juvenile cancers at the remaining sites in the exposed area.

235. On the basis of Utah-wide data on fallout, Beck and Krey [B7] have contended that the suggested low-dose area in northern Utah actually received higher mean doses, so that the gradient in risk described by Lyon et al. [L4], even if correct, does not correspond to exposure. Johnson [J2] used a large interview study of Utah Mormon families to argue for a very large excess in cancer (at all ages) at known radiosusceptible sites. Because of problems with its methodology, the Johnson study has not generally been accepted as valid. In a study, which attempted to determine if Johnson's results could be correct, Machado et al. [M20] restricted their analysis to a small area of southern Utah. In the proper time period following exposure to fallout, 1955-1980, there was a small excess number of leukaemias with onset at ages 0-14 (RR = 2.84 for exposed children, much less than found by Johnson) [M20]. There was also an excess of cases at all ages (RR = 1.42). Machado et al. estimate that the doses were between 0.001 and 0.021 Gy, so that small effects, at most, would be expected.

236. There may, accordingly, be a small increase in the rate of leukaemias in these exposed children, although more accurate estimates of dose in other parts of Utah might reduce the size of this effect. However, since southern Utah rates were about 35% higher than expected from the rates in the United States as a whole, at least some of the increase is probably real. The individual doses are not known, so a dose-response analysis is not feasible.

237. Analysis of leukaemia mortality rates in the United States through the period of atmospheric nuclear testing has suggested patterns which are consistent with fallout effects [A18]. States were classified according to the level of exposure based on the assessment of strontium-90 in cow's milk samples in 1959-1960, in human bone in 1966-1967, and in total diet in 1964-1965. The leukaemia rates during 1950-1976 were correlated with exposure in states classified in high, intermediate and low exposure groups. The pattern of leukaemias showed a 5.5-year latency and a 15-year plateau, and were only of the acute and myeloid types, consistent with what is known from other studies of radiation-induced leukaemias in children. There was also a consistent nation-wide pattern of these types of childhood leukaemias, and no other exposures to radiation or other agents could be identified by the author to account for

this. The estimated risk coefficient was 6.46 ± 0.16 excess leukaemia deaths per 10^4 PYGy. The author reviews other data on childhood leukaemia, arguing that his risk estimates are consistent with those, further supporting a fallout-related explanation.

238. Recently, two preliminary surveys of the frequency of cancer among young people living near nuclear power installations in the United Kingdom, specifically at Dounreay and Sellafield, reported an increase in leukaemia [G19, H26]. Since the estimated radiation doses in the vicinity of these plants seem too low to account for the increase, other studies were initiated. One of these addressed the situation at Dounreay [D23]. These authors conclude that their findings weigh heavily against the hypothesis that the increase had been due to radioactive discharges from the plants, unless the doses to the stem cells from which childhood leukaemia originates have been grossly underestimated. Cook-Mozaffari and her associates have undertaken a far more comprehensive study of mortality in the years 1959-1980 in the vicinity of all of the nuclear installations in England and Wales [C19]. Their findings are succinctly summarized in [F14]. Briefly, there was no evidence of a general increase in cancer mortality near nuclear installations in the 22-year period. Leukaemia in the age group 0-24 years may be an exception, however: standardized mortality rates for lymphoid leukaemia increased with increasing proximity to an installation. In Local Authority Areas where at least two thirds of the population resided within six miles of an installation, relative risks were invariably greater than 1, ranging from 1.12 (British Nuclear Fuels Limited, Capenhurst, not statistically significant) to 3.95 (Amersham, statistically significant at the 5% level on a one-sided test), as compared with Local Authority Areas not in the vicinity of nuclear installations and selected to have a similar urban-rural status and population size.

239. Over all installations, the relative risk for leukaemia within the six mile distance was 2.0. Since the individual exposures are unknown, no dose-response estimate was possible. The investigators noted that it was not clear whether the effect could be due to a general confounding of other environmental or socio-economic factors. One of these may be the venting of pesticides into the air when the cooling towers were cleaned. Although it is known that the subsequent risk of leukaemia among individuals exposed in the first two decades of life is substantially higher than among those exposed later in life, based on the Japanese experience and a linear dose-response model, a doubling of the risk would imply a dose of hundreds of mGy which seems unlikely given present estimates for exposures within a few miles of a nuclear installation. Thus, while the risk observed may be real, it cannot be accounted for on the basis of the doses received.

(d) Exposure of the female breast in childhood and at perimenarchial ages

240. In general, radiogenic breast cancer follows the natural age distribution for this tumour, suggesting that, as they do with spontaneous breast cancer, other host factors, specifically the endocrine and cell proliferation status of the exposed breast tissue, modify the susceptibility and latency of radiogenic breast cancer [B6, H6, T6]. Data on age at exposure are available from women receiving chest fluoroscopies for pneumothorax monitoring in New York [L6, S1], Massachusetts [D8] and Canada [H6]; from women treated for mastitis in Massachusetts [B3, L6]; from women treated for benign breast disease in Sweden [B8], and from atomic bomb survivors [T1, T6].

241. These data consistently disclose a declining relative risk of cancer with increasing age at exposure, and a markedly high relative risk for exposure in adolescence and young adulthood. For individuals under age 25 at exposure, there is a latency period of at least 15 years, and excess risk persists for 40 years or more [H6]. A linear model gave the best fit in the Japanese [T6], New York, and Massachusetts series [H6], but a quadratic model fit better in a combined analysis of the Canadian fluoroscopy data [H6]. Except in the case of the Japanese study, the young women ordinarily received courses of treatment involving several (1-10) exposures and a total of about 1.5-2.5 Gy to the breast. With the possible exception of a large Canadian study (to be discussed later, in the section on adult breast exposures), the evidence does not document a fractionation effect [B6]. The absolute risk is in the range 3-8 additional cases per 10^4 PYGy [B6, T6] based on a linear model, with higher values having been found in women who were younger at the time of exposure. Women irradiated at the time of their first pregnancies were especially vulnerable [B6]. Because the Japanese women who were less than 15 years old at the time of the bombing are only now reaching the ages when breast cancer becomes most common, it is not yet possible to derive satisfactory lifetime dose-response estimates.

242. Several major findings have recently emerged from Japan [P15, T6]. In corroboration of the studies reviewed above, an elevated risk among women aged 10-19 at the time of exposure has been clearly shown. The relative risk for women who received 1 Gy when they were under the age of 19 has been estimated to be 2.51 ($\pm$ 0.75), compared with a relative risk of 1.45 ($\pm$ 0.24) for exposure over that age in the extended Life Span Study (LSS-E85) using the T65DR dose estimates [P15]. At present, the data are not completely consistent with respect to the level of risk for post-menopausal exposures, and further studies of the exposed cohorts are needed. The most important result may be the finding that women under age 10 at exposure exhibit an excess risk ([T6, T7, T14]; see Table 23). The excess is related to dose, but did not appear until this cohort reached the ages at which breast cancer normally arises, consonant with previous age-onset results. These are higher relative risks than observed in any other age group having received comparable doses. Absolute risks are high at ages 10-19 [T6], though they seem roughly comparable to ages < 10 [T14]; that is, childhood may be the peak years for susceptibility. The Japanese findings are summarized and discussed in more detail in section III.C.4 on adult exposures. Breast-tissue susceptibility

below age 10 has also been seen among infants irradiated for thymus enlargement [H19] and in children irradiated for other primary cancer [L12].

3. Tissues apparently not susceptible to radiation-induced carcinogenesis

243. There is accumulating evidence that certain tissues, generally those involving non-proliferating cells, are relatively non-susceptible to radiation-induced cancer. In children, cancer caused by radiation is restricted commonly to the white blood cell and bone-forming tissue and to the thyroid. Except for breast and perhaps stomach there are not much specific data in regard to solid adult cancers. However, the indications from the most recent Japanese (DS86) data suggest a high general susceptibility, expressed at the normal adult ages for carcinomas [S49]. Children surviving irradiation to treat childhood cancers may in coming decades reflect risk at a variety of irradiated sites.

244. The information presently available thus suggests that if there is an effect of radiation in childhood for other sites, the result will be similar to that found in the breast, in that excess cancers will arise at roughly the same ages at which they do naturally. If this occurs, then a variety of tissues may be shown to be susceptible to radiation in childhood, but perhaps without an unmistakable excess susceptibility due to the young age of the exposed tissues. This would provide an important contribution to our understanding of radiation carcinogenesis and of the tissue biology of epithelia.

4. Summary of exposure effects in children

245. In summary, the experience of children who have received substantial doses of ionizing radiation demonstrates the susceptibility of the thyroid, bone, bone marrow and the breast. The bulk of the children who have been successfully treated by radiation for cancer initially presented with tumours characterized by a large heritable component. From observations on some of those children who received neither radiation nor chemotherapy, it can be seen that they are obviously more prone to develop cancer than normal children. In general, certain sites are susceptible, and the evidence is now clear that this has to do with gene regions expressed in both the tissue involved in the original primary tumour and in the tissue of the second tumour, particularly in the case of secondary osteosarcomas occurring in treated retinoblastoma patients. Individuals with the hereditary form of this tumour are known to develop osteosarcomas away from the irradiated field or in the absence of irradiation. There is also evidence that other relatives of some retinoblastoma [C5, S12] and rhabdomyosarcoma [S50] patients are at excess risk of cancer of various types at a number of organ sites.

246. Current evidence, though not conclusive, suggests that children are susceptible to the induction of most cancer types by radiation, and that there are characteristic patterns to the expression of that risk. Tumour types arise at ages that are typical of the spontaneous childhood tumours of the same sites, but exposure in childhood also appears to generate tumours that are typical of adults and that appear in the usual adult ages rather than in childhood. There are indications in the case of second tumours following retinoblastoma treatment that a multiplicative projection model may apply, as it does in the case of most adult tumours; data from other sources are at present still too sparse to reach any general conclusions.

247. With the exception of the Japanese data, the studies on childhood induction of cancer following ionizing radiation provide convincing evidence that there are effects, but they do not provide risk coefficients useful in lifetime risk projection for adult tumours.

C. TISSUE SUSCEPTIBILITY IN ADULTS

248. It is to be expected that age is a critical factor in determining the radiation risk, since childhood is a period of tissue generation and, for many tissues, differentiation. The question is, how different is susceptibility in the tissues of adults?

249. The bulk of our knowledge of dose-response, age and projection patterns after adult exposure comes from three studies: women treated for cervical cancer, patients (mainly men) treated for ankylosing spondylitis, and the Japanese exposed to the atomic bombings. These studies will be presented in detail in chapter VI. In this section, their results and those of the remaining literature will be summarized in the context of the biological issues involved.

250. As was seen with juvenile exposures, there may be biological differences between tissue types of adults relevant to their radiosusceptibility. The assessment of radiation effects must be made in the context of the exposed populations that are available for investigation. Most adult exposures occur as a result of treatment for disease; such individuals may not be biologically representative of the population at large, either because they are genetically different or because their disease itself changes their susceptibility to radiation. This is likely to be especially true of individuals irradiated for the treatment of cancer itself.

251. Two principal sources of possible non-representativeness are important in the interpretation of cancer induction in irradiated cancer patients relative to other adult cohorts. First, it is known that a fraction of any population is genetically susceptible to cancer, e.g., cancers of the colorectum and the breast. Second, most cancers are thought to result from specific environmental exposures [D13]; cancer patients may not be representative of their population in terms of their history of exposure to such risk factors. To the extent that this is true, their tissues may already have been partially affected by these risk factors. Comparing their post-irradiation experience with that of their age- and sex-matched peers may not be an appropriate way

to estimate the effects of radiation. Evidence on this point will be given later.

252. There are important informative cohorts of adults who were initially exposed for other diseases, including tuberculosis, mastitis, thyroid anomalies, benign gynaecological problems, conditions requiring diagnostic x-ray examinations, and ankylosing spondylitis. It is difficult to know, however, if these patients are random representatives of their populations; there are indications that some, at least, are not. Their risk should be compared with that of the most representative possible unexposed groups.

253. Several other cohorts of individuals, in effect randomly selected from their populations, have been exposed to large doses of radiation, and they are naturally the most reliable sources of data on the susceptibility of normal adults. They include occupationally exposed groups (radiologists, radium dial painters, miners, nuclear workers) and individuals exposed to nuclear testing or to the atomic bombings in Japan. To gain information on the biological aspects of radiogenic cancer, it must be determined if these groups have different experiences.

254. As essential as it is to consider separately the risk for individuals who may be susceptible to radiation carcinogenesis and the risk for those who are probably more representative of their population, it is equally essential to consider the risk in terms of the nature of the tissues exposed to radiation. There are different tumour groups in this regard. The first is haematopoietic tumours following irradiation of the bone marrow and osteogenic tumours. The risks here are unique. Due to the widespread distribution of active bone marrow, such tumours have been observed following many different exposures. The second group is nerve and connective tissue tumours, where tumours that occur in normally dividing cells should be differentiated from those in non-dividing cells. The third and largest group of adult tumours is carcinomas of epithelial tissues, which tissues have life-long patterns of cell division and specialization.

1. Adults exposed to radiation for treatment of primary cancers

255. Relatively little is known on a population basis about the risk of radiogenic second tumours in the many adults who have been exposed to treat a primary malignancy. To date, most of the evidence has been derived from investigations of the consequences of radiotherapy for cervical and ovarian cancers, for Hodgkin's disease and non-Hodgkin's lymphoma and for the tumours of childhood reviewed earlier. There has also been one large general study of secondary leukaemias in cancer patients.

256. Second tumours in adult cancer survivors may take the form of local solid tumours in the irradiated field, if cell sterilization has not been excessive. Radiogenic sarcoma has been observed to arise from the area of skin, or the underlying fascia, in an irradiated area or even from a radiation ulcer (e.g., [B20]). Second tumours may also be systemic ones such as leukaemias, presumably caused by a transformation of the cells in exposed bone marrow. Finally, second tumours sometimes occur at remote sites; whether such tumours could be radiogenic, e.g., due to scatter, is essentially impossible to determine.

257. Since many cancer survivors treated by radiotherapy were also treated by chemotherapy, it is necessary to ascertain what portion of the second-cancer risk is due to the chemotherapy or to an interaction between the two modes of therapy. This can be difficult. Alkylating agents have a high carcinogenic potency; they are delivered in large doses, and most of the body is exposed. Radiation is locally delivered, in general. Interactions will be considered in chapter V.

258. In the case of leukaemia following radiotherapy for lymphomas, it is presently difficult to differentiate between truly radiogenic second cancers (i.e., leukaemias) and leukaemias that have been caused by the conversion of an initial lymphoma cell to leukaemic form; this has been suggested to be a normal stage in the natural history of lymphomas, although some studies reveal that it is not [e.g., B10]. Molecular genetic methods may be able to resolve this issue if the original lymphoma cells have specific genetic markers for which one could screen the subsequent leukaemia cells.

259. Only a minority of irradiated patients develop radiogenic second tumours. The relative risks are often rather small, especially for cancer patients who are elderly and have fewer years of life during which to be at risk.

(a) Treatment for Hodgkin's disease

260. One of the most intensively studied groups of adult cancer survivors, in terms of risk of second cancers, is that of persons treated for Hodgkin's disease. While the treatment modalities varied, they often involved radiation to affected lymph nodes. In some patients, no other therapy (or only surgery) was carried out, but in the majority of long-term survivors of Hodgkin's disease, chemotherapeutic agents were also used. The results of several studies are reviewed here [B9, B10, B11, B37, C7, C8, G4, P12]; [B11] is a summary of results reported to 1984.

261. The most notable second cancer in these patients is leukaemia, specifically, acute non-lymphocytic leukaemia (ANL), although solid tumours and other leukaemias have also been observed. In the many studies reported, the results are similar. X-ray therapy alone does not seem to be a risk factor for the leukaemias, and it may or may not be a risk factor for solid tumours; evidence on the latter point is conflicting, and since the latency period is longer than for leukaemia, there may not yet be sufficient data on long-term survivors.

262. Boivin and O'Brien have analysed data pooled from seven reports of second-cancer risk after treatment for Hodgkin's disease [B37]. After radiotherapy, the

relative risk values for solid tumours were as follows: all sites. 2.2; bones and joints, 20.0; soft tissues, 18.3; non-Hodgkin's lymphomas, 8.1; melanomas of the skin, 6.7; buccal cavity and pharynx, 4.1; nervous system, 3.6; respiratory system, 2.5; and digestive system, 1.8. Unlike for leukaemias, no elevated risk of solid tumours was found in the chemotherapy-only group, possibly because the studies had shorter follow-up times.

263. Chemotherapy has been strongly associated with subsequent tumours and with the special risk of acute non-lymphocytic leukaemia; combined therapy appears to lead to an increased risk based on the interaction between the radiation and chemotherapy. However, support for an excess effect of x-ray therapy is weak [B11]; the relative risk of combined therapy versus chemotherapy without radiotherapy was slightly, but not significantly, lower (125 vs. 136) in one series of patients from the United States [B10, B11], and there was no interaction effect in another [C7]. Briefly, there is little evidence for a radiation hazard relative to secondary acute non-lymphocytic leukaemia in treated Hodgkin's disease patients. These data are summarized in Table 24.

264. It is appropriate to note that effective chemotherapy is relatively recent and that in the past, with only x-ray therapy available, survival was not sufficient to provide much information. Data are accumulating that may permit the statistical isolation of the independent effect of chemotherapy, but this will not be feasible until some years at-risk have accumulated. There are no systematic dose-response data as yet.

265. The cumulative relative risk of acute non-lymphocytic leukaemia due to chemotherapy of various types is high, sometimes in the hundreds; for other leukaemias it is about 10 and for solid tumours, about 3-4; individuals older than 40 at treatment are at higher relative risk than those who were younger, a somewhat unexpected finding [C7, G4]. The risk of solid tumours appears to be increased about 3-4 times by combined therapy. In a study of United States patients, Boivin and Hutchinson report that the risk of a solid tumour was 1.8, relative to the expected number, for those with no intensive therapy (raising a question about the appropriateness of the expected rates), 2.1 for radiation only, 1.8 for chemotherapy alone, and 3.3 for combined therapy [B10]. After a latency period of 10 or more years, the relative risks were statistically significant only in the case of the radiotherapy-only group, for whom the relative risk of solid tumours was 25 (95% CI: 8.1-58.4).

266. An increased risk of acute non-lymphocytic leukaemia has been seen in children treated for Hodgkin's disease [T4]; however, there was no case in children treated with radiotherapy alone [M28]. While it is conceivable that individuals with Hodgkin's disease have a natural proclivity to develop leukaemia, the evidence suggests that leukaemias occur after chemotherapy and in all stages of Hodgkin's disease, but very rarely in the absence of chemotherapy [B10, B11, G4].

267. A recent study of Hodgkin's disease patients treated with chemotherapy and radiotherapy at the United States National Cancer Institute has been reported [B35]. As had been found in other radiogenic leukaemia studies, the excess risk in this small series of 192 patients was expressed in only a small window of time, in this case from three to 11 years. Of course, such second tumours, all acute non-lymphocytic leukaemias, were only observed in survivors of the original disease; moreover, the authors did not isolate the effects of radiation therapy from those of chemotherapy. Two other studies appear to confirm this report [P12, T19], although there are reports of elevated risk in comparable groups for a period of up to 20 years [K26].

268. Recently, another indirect risk of treatment of Hodgkin's disease has emerged. In a study of female patients in Houston, Texas, United States, cervical conditions resulting from infection by human papillomavirus were found to be considerably more prevalent than expected in a general population of the same ages. A twofold to fivefold increase in the risk of carcinoma in situ and of invasive carcinoma of the cervix and anogenital region is seen in these women [K8]. Although this has not been reported in other Hodgkin's disease series, the connection is plausible but will require longer follow-up to be ascertained (this study entailed a detailed retrospective review of Papanicolaou tests of the patients over many years). In an immune-suppressed individual, vulnerability to human papilloma virus infection appears heightened (paragraph 302); this infection progresses over a period of years, in some individuals to carcinoma [K8]. It is not possible, from the available data, to identify radiation effects independently, because most patients received combined therapy.

(b) Treatment for cervical cancer

269. Cervical cancer is commonly treated by external beam therapy and/or by the implantation of intracavitary radium sources in the vagina and the uterus for extended periods of time. In the cohorts that have been studied, radiotherapy has usually involved two 36-hour applications of 2.6 10^9 Bq of radium, followed by 20-70 Gy from external beams delivered in 2-Gy fractions over several weeks [B12]. Both orthovoltage (200-400 kV cobalt-60) and megavoltage (2-33 MV photons from betatrons or other devices) external radiation have been used, with a typical dose being 20-70 Gy to the pelvis, delivered in fractions of several Gy over a 4-8 week period. Risk analysis was initially carried out by classifying the tissue sites into three groups based on general level of dose. The greatest dose is received by the entire pelvic area and the lateral lymph nodes; other heavily exposed organs include the bladder, rectum, endometrium, colon, ovaries and bone. The kidney, gallbladder, stomach, pancreas, and liver are exposed to an intermediate level of radiation. Finally, remote sites such as the buccal cavity, lung, breast, brain, salivary gland and thyroid receive little radiation. Nearby sites have doses in the tens of Gy; intermediate sites, 1-10 Gy, and remote sites, tenths of Gy [B12].

270. The largest study of second tumours in cervical cancer patients is the International Radiation Study of

Cervical Cancer Patients (IRSCCP) [B12, D9]. Over 182,000 women from eight countries (Canada, Denmark, Finland, Norway, Sweden, United Kingdom, United States and Yugoslavia) have been followed from the time of diagnosis of cervical cancer. Over 1.3 million person-years of observation have accumulated, including 623,798 person-years to women who were treated by irradiation and 178,243 person-years more than 10 years after irradiation. The average follow-up was 7.6 years. A high fraction of cancers was confirmed histologically, and it is believed that the participating registries were able to ascertain the vast majority of cancers that occurred; data from the separate registries and details of the investigation are reported most fully in [D9]. Since most other studies of cervical cancer have as their subject the same individuals included in [D9], only the latter results will be reported in detail.

271. In addition to classifying the sites according to distance (close, intermediate or remote) from the radiation source, the IRSCCP study divided the women into those with invasive cancer who were treated with irradiation, those with invasive cancer who were treated without irradiation, and those with in situ cancer of the cervix treated without irradiation. Relative risks for women followed more than 10 years are given in Table 25 (the table contains data from another study, to be discussed below). Interestingly, the pattern of relative risks was similar among the three groups. Of 5,146 second cancers, at most 5% (162) were attributable to the exposure to radiation. Pelvic irradiation with high doses seemed to be associated with some increased risk of cancer to the exposed organs, including the bladder, rectum, bone, connective tissue, ovary, small intestine and kidney. The risk of bladder cancer was also increased in women with similar diseases who had not been irradiated; however, the relative risk increased, over time post-exposure, only in the irradiated group, suggesting a radiation effect [B12]. The risk of cancer of the uterine corpus was consistently below expected levels. Figure IV provides the probabilities (relative risks) and confidence intervals for various second cancers in irradiated women at least one year after irradiation.

272. The risk of leukaemia was elevated in these data, but the elevation was similar for exposed and non-exposed. The dose to the bone marrow is estimated to have ranged between 3 and 15 Gy, and hundreds of radiogenic leukaemias would have been expected. However, fewer than 100 were observed, suggesting that cell sterilization was an important factor. The relative risk of non-lymphocytic and acute leukaemias was small, 1.4 (95% CI: 1.1-1.8), and may reflect lower levels of exposure at more remote marrow sites, where cell sterilization was not important. In one of the studies of which the IRSCCP series was composed

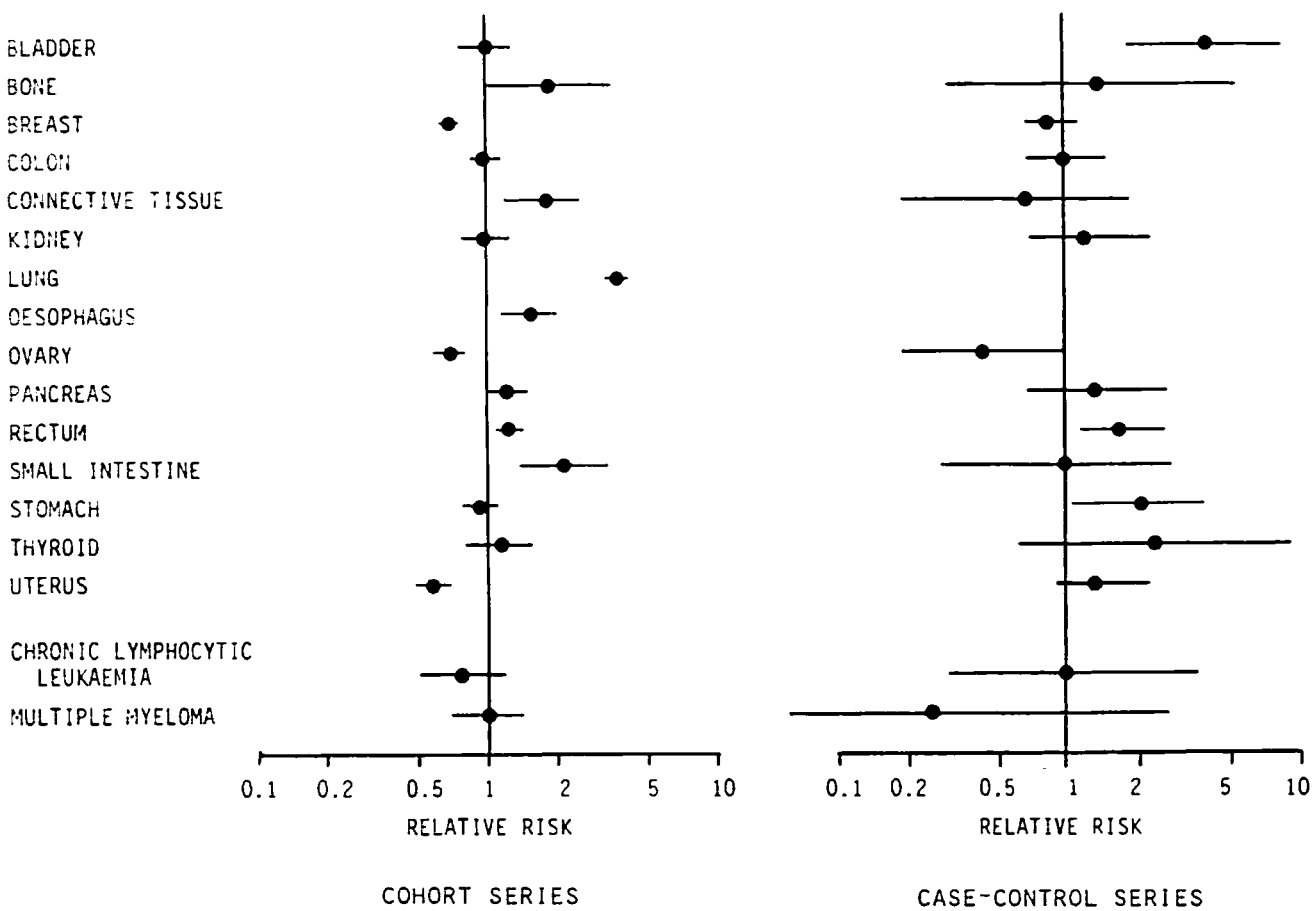

Figure IV. Relative risks of malignancy at specified sites after radiotherapy for cervical cancer based on a cohort series [B12] and a case-control series [B38].

[S34], doses to the marrow of about 7.8 Gy did yield a relative risk of 2.5 for leukaemia; however, although the sample base was 25,718 women, this was not significant. Leukaemia was slightly elevated in the Surveillance, Epidemiology, and End Results (SEER) programme patients studied in the United States by Curtis et al. [C8], and the relative risk of leukaemias in another large case-control study in the United States [B26] was 2.3 (95% CI: 0.2-24.4).

273. To understand this apparent leukaemia deficit better, a case-control study of leukaemia in a sample of the IRSCCP patients has been done [B36], using individual patient records from 196 leukaemias and 750 matched controls (cervical cancer patients not developing leukaemia) to estimate the doses to each of 14 skeletal components of bone marrow. Using a marrow-component-weighted dose-response function, the best-fitting models included a negative exponential (cell-sterilization) term. Linear and quadratic terms, relative to the effects at low doses, could not be distinguished statistically in this study, although the latter yielded higher risk estimates at low doses and were preferred by the authors. This suggested to the authors that in therapeutic doses, cell killing is responsible for the deficit in leukaemias noted above. The risk of chronic lymphocytic leukaemia (CLL) (RR = 1.03) was not elevated, but the overall relative risk for radiogenic leukaemias was 2.1 (90% CI: 1.0-4.3); risk increased until 4 Gy, with a 0.88% excess relative risk per 0.01 Gy, and at 1 Gy the relative risk was 1.88, based on the linear term of the dose-response function. If the exponential term is included, the relative risk at 1 Gy reduces to 1.7 in this study. Women treated over age 55 experienced no excess risk. The difference between the relative risk in this sample and the relative risk of 2.3 for the entire cohort was attributed to increased sample sizes in the more recent study [B36]. The estimated excess number of cases per 10^4 PYGy was 0.48..

274. The authors [B36] concluded that this analysis, incorporating both cell sterilization and local marrow exposure considerations, not only explains the deficit in leukaemia in the IRSCCP group relative to other groups receiving smaller mean doses (e.g., women treated for benign gynaecologic problems and atomic bomb survivors) but also provides an estimate of the maximum leukaemogenic effect of radiation. This is a relative risk of about 5; doses sufficient to generate greater relative risk values will, by virtue of the cell sterilization effect, fail to do so. Radiotherapy was judged to be a much weaker leukaemogen than chemotherapy.

275. For unknown reasons, colon cancer occurred at similar rates in irradiated and non-irradiated women. This was unexpected, since other studies (e.g., [D11] and [S3]) have found evidence for radiogenic colon cancer, and the rapidly dividing cells of the mucosa of the colon should be susceptible to radiation. However, a study of Swedish patients, while revealing excess cancers of the bladder, endometrium, ovaries, and rectum, also did not find an excess of colon cancer relative to the general Swedish population [P17]. On the other hand, there is a report of teenagers sterilized in German concentration camps during World War II who are now manifesting colon cancer in areas surrounded by radiogenic tissue damage [R15]. The lack of excess colon cancer in cervical cancer patients (at a statistically significant level) could be the result of cell sterilization [B12]; however, rectal cancer was increased in the same IARC series only among irradiated women, implicating the therapy [B12]. Since non-irradiated cervical cancer patients exhibited a risk pattern similar to that of those who were irradiated, other interacting factors must be suspected.

276. There was no excess of cancer of the stomach, pancreas or kidney, all of which organs had received intermediate doses in the IRSCCP patients. The results for pancreas and kidney are not surprising, because in other major series these sites were not highly susceptible. However, the stomach received substantial doses and would have been expected to show elevated risk.

277. For organs receiving low levels of exposure, no excess risk was seen other than to the lung and oral cavity. Younger as well as older irradiated women showed a reduced risk to the breast, for which the authors offer the explanation that radiation-ablation of the ovary has a protective hormonal effect; however, the effect was also seen in women irradiated post-menopausally [B12].

278. The deficit in uterine sarcomas is interesting, because an excess of such tumours occurs in women irradiated for benign gynaecologic disorders (see below); the deficit in the cervical cancer group may be due to a higher proportion of the women having undergone hysterectomy as part of their cervical cancer treatment, leaving fewer women at risk of uterine cancer [B12]. Excess risk to the uterus has appeared in a study in China, where 8,704 women treated for cervical cancer received 5.5-12 Gy to the uterus; in this group, 12 uterine cancers occurred five to 19 years after exposure [Y4].

279. Smoking-related sites (lung, oropharynx, bladder, oesophagus) have shown elevated risk in the IRSCCP series. Some excess was also seen in patients not irradiated. There may be an interaction between smoking and other factors to produce cervical cancer (see section V.A).

280. The IRSCCP study population has been used for a case-control study [B38] to provide more precise relative risk data than is possible with the more heterogeneous total cohort. The case-control study included 4,200 patients with second cancers for which histological confirmation was possible and 10,200 matched controls (patients without second cancers). These relative risk values and estimated excess cases per 10^4 PYGy are shown in Table 26 [B38]. Details of the dose-response patterns are provided in the original study [B38] but are too extensive to be given here; the results are broadly consistent with the full-cohort study [B12, D9].

281. In the case-control study of the same series [B38] it was found that the dose-response pattern

increased with dose, even for high doses, for most pelvic organs other than the colon. Indeed, rectal, bladder, uterine, vaginal and ovarian cancers all showed dose-response increases up through doses in excess of 100 Gy. These tissues should also have experienced a cell-killing effect, as indicated for the colon, and the fact that they did not casts some doubt on the explanation of the lack of colorectal cancer excess in this series.

282. Figure IV presents comparisons of the relative risks based on both the whole cohort and the case-control analyses of the cervical cancer series. There are some substantial, difficult to explain differences, highlighting the importance of using appropriate control or referent data.

283. A prospective study in Japan of 1,572 women treated with radiotherapy for cervical cancer (1,478 cases) and ovarian cancer (94 cases) revealed eight cases of leukaemia (five non-lymphocytic, one acute monocytic, and two chronic myeloid leukaemias) where 0.45 would have been expected, based on the rates for the general population [M26]. The period of follow-up ranged from six to 20 years. The relative risk was 11.2. The average mean marrow dose was 1.2 Gy, and the absolute risk coefficient was 0.45 excess cases per 10^4 PYGy to the bone marrow. Four of these individuals were in a high-dose-rate group treated with both a linear accelerator and remote afterloading using radium, and a fifth had been treated with the linear accelerator alone.

284. In a subsequently published series from Japan of 19,384 women with uterine carcinoma of whom 12,729 had been given either radiation alone (4,310) or radiation combined with surgery (8,419), statistically significant increases in leukaemia and cancer of the rectum were observed [A14]. The relative risks were 3.9 and 2.9, respectively.

(c) Treatment for ovarian cancer

285. Two groups of patients from the United States initially affected with ovarian cancer have been studied by Reimer et al. [R11]. In all, 45,903 person-years of risk were observed, with a mean initial age of 56 years. Patients had been given varying treatments. There was an overall relative risk of cancer of 1.4; the relative risk was 1.5 in the irradiated patients and 1.1 in non-irradiated patients. Sites included the endometrium, colon, bladder, breast and haematopoietic system. In the first two years of follow-up, the risk was greatest for endometrial cancer. Since the rates of hysterectomy were not known for the computation of expected and observed rates, this finding may be spurious. However, the uterine corpus of post-menopausal women is known to be susceptible to rapid carcinogenesis after the administration of exogenous hormones (e.g. [S29]). Reimer et al. found a non-significant excess of breast cancer, raising the possibility that other hormonal or therapeutic factors interacting with treatment may be involved. Their data are summarized in Table 27. Values given in the table are relative risks. The study reported relative risks after less than two years, 2-4 years, 5-9 years and more than nine years of follow-up, but there were no time patterns. The relative risk values were roughly constant from two to 9+ years, which is too soon after exposure to draw meaningful conclusions about projection effects. Dose information was not available.

286. In one of the groups, the non-irradiated patients had a relative risk of 0.3 of developing leukaemia, but irradiated patients had a corresponding risk of only 1.3 (not significant); however, in the other group, who had been given chemotherapy only, the relative risk was a significant 9.3 (95% CI: 5.2-15.3). In 1,399 ovarian cancer patients, Greene [G3] evaluated the risk of subsequent acute non-lymphocytic leukaemia as a function of treatment. In women treated by radiation alone, no acute non-lymphocytic leukaemia occurred; in those treated with both radiation and chemotherapy, the relative risk was not different from that in women treated with chemotherapy alone (combined therapy: RR = 120, 95% CI: 44-261; chemotherapy: RR = 100, 95% CI: 37-218) [G3].

287. Greene also reported an excess of colon, but not rectal, cancer after at least five years of follow-up, whereas, as noted above, in the cervical cancer group, rectal cancer was significantly elevated but colon cancer was not.

288. Lymphoma and bladder cancer were increased only in the irradiated group. No excess of leukaemia occurred in one of the series; however, in the other, there was a total excess of leukaemias (acute non-lymphocytic leukaemias), with RR = 171.4. Most of these cases had received radiotherapy, but all had also received chemotherapy. This study [G3] was the first report to raise the possibility of a causal role for radiation in bladder cancer and in lymphomas in ovarian cancer patients.

289. Another large study (9,726 cases) from the United States [C8] found the following relative risks of leukaemia: 10 for radiotherapy with no known chemotherapy and 9.5 for chemotherapy, both significant at the 1% level. When only acute non-lymphocytic leukaemia was considered, these relative risks were even higher (21.1 and 22.2, respectively). These results are not consistent with the minimal radiation effects in the absence of chemotherapy in other studies described earlier, and there is at present no clear explanation for the difference. However, chemotherapy is used increasingly to treat ovarian cancer and radiotherapy decreasingly; therefore, the estimation of the cancer risk from radiotherapy may be more relevant to radiobiology than to practical problems in human health. The Japanese investigation of cervical cancer, cited earlier, reported an excess of leukaemia in a population of 1,572, of whom 94 had ovarian cancer [M26]; whether this suggests a special risk only, or mainly, in the small subset cannot be determined adequately.

(d) Treatment for breast cancer

290. In a series of second breast cancers in women irradiated for a primary cancer of the breast, Hankey et al. [H13] found no significant evidence of a

radiogenic risk to the second breast (from scatter) but did find a relative risk of 3.2 for all second primary breast cancers, when compared to the overall population. For breast cancer patients treated only with surgery, the relative risk was 1.2-1.4, perhaps reflecting a generally elevated susceptibility of patients with a primary breast cancer. This study involved a five-year follow-up of 27,175 patients in the state of Connecticut, United States. In a comprehensive study of the Connecticut Tumor Registry for the period 1935-1982, Harvey and Brinton [H20] found the relative risk of a second tumour to be 2.0 (95% CI: 1.9-2.1), compared to 1.5 (95% CI: 1.5-1.6) for women treated without radiation; 20 years after irradiation the two risks became equal (at RR = 1.7, based on the Connecticut population). The relative risk for a second breast cancer was 3.9 for irradiated and 2.8 for non-irradiated patients. Relative risk values for other second tumours are given in Table 28; the treatment data refer only to initial treatment, and there may be some false negatives. Note that an excess of chronic lymphocytic leukaemia was reported; this is not thought to be a radiogenic tumour and suggests that these results may confound other sources of variability.

291. Among 14,000 British women treated between 1946 and 1982, 194 developed a second tumour in the contralateral breast more than one year after the initial diagnosis [B28]. There was no evidence for radiation induction in the second breast, based on a comparison with a matched control group.

292. In another study of initial treatment for breast cancer [C8], the relative risk of leukaemia associated with surgical treatment alone was 1.0, for radiation-only 1.7, and for chemotherapy alone a significant 3.8 (Table 29). The risk for radiation alone, when all types of leukaemia were considered, was not significant, but the relative risk for acute non-lymphocytic leukaemia alone was 3.7 (95% CI: 1.6-7.2); compared with the significant RR of 6.7 (95% CI: 4.5-32.3) for acute non-lymphocytic leukaemia following surgery or chemotherapy. The latency period suggested a causal effect of the treatment to the authors, because the excess risk did not occur until at least three years after irradiation (but, the same was true of the chemotherapy-only group). Patients with local disease had no excess leukaemias, but patients with regional stage disease exhibited relative risks of 2.7 for all leukaemias and 5.5 for acute non-lymphocytic leukaemia. The regional-local distinction was found for chemotherapy as well as radiotherapy. As this finding had not been reported in any previous systematic examination of breast cancer patients, the authors suggest that unreported chemotherapy could have occurred, though there may be some radiogenic effect [C8]. No dose-response data were available for these patients.

293. A study of 1,359 Japanese breast cancer patients showed a 1.29-fold higher risk of second cancer in patients without a family history of cancer, but with a family history of breast cancer the relative risk was 3; a radiation-associated relative risk of 1.62 was observed, but no increase was associated with chemotherapy [Y3].

294. A small number of the survivors of the atomic bombing of Hiroshima and Nagasaki have developed more than one primary cancer [M16, R6]. Specifically, three women have been identified, all of whom initially had breast cancer and subsequently developed a second malignancy. All had undergone radiotherapy. Two developed acute granulocytic leukaemia, one four years after radiotherapy and the other eight years after; their estimated atomic bomb doses (T65DR) were 5.94 and 3.64 Gy, respectively. The third woman (atomic bomb dose 0.31 Gy) developed cancer of the lung beneath the treated breast some eleven years after radiotherapy. While it is probable that the carcinoma of the lung was radiotherapeutic in origin, it is moot whether the two cases of leukaemia were attributable to the therapy or to the atomic bomb exposure. However, the time that intervened between first and second malignancy was in both cases consistent with a radiotherapeutic origin, and the treatment doses had been high.

295. A study undertaken in the United States of 8,483 women has found a significant excess of acute myelogenous leukaemia after irradiation for breast cancer, but not in those not irradiated [F11]. Many of these cases had also received adjuvant chemotherapy. The post-irradiation risk was $1.29 \pm 0.5\%$ after 10 years.

2. Adults exposed to radiation for immune suppression

296. Total-body immune suppression has been induced in numerous patients to treat leukaemia or Hodgkin's disease, to prepare the patient for a bone-marrow transplant following the eradication of leukaemia or to prevent host-versus-graft disease in recipients of kidney transplants. The treatment effects have been discussed above. The use of a bone-marrow transplant to replace leukaemic immune system cells has been successful in up to 60% of cases with acute myeloid leukaemia, and, to date, only two second malignancies have been noted [B14, L8, T8]; doses range between about 7 and 12 Gy. It is too soon to summarize the risks associated with this treatment, for there are as yet few surviving patients. However, one report has shown a 2% risk of second malignancies, over 2-5 years, in individuals treated with total body irradiation [T8]. Complicating these results is the fact that some chemotherapy before or accompanying the irradiation has probably been given to these patients.

297. Some credence is lent to these estimates of risk by Greene et al., who have scrutinized individuals treated for non-Hodgkin's lymphoma (NHL) [G3, G9]. Among 517 patients, in whom marrow dose could be estimated, there were nine acute non-lymphocytic leukaemia cases observed where essentially none (0.08) had been expected, a relative risk of 105 (95% CI = 48-199). This was after 2,203 person-years of observation, a mean age at diagnosis of 43.4 years and an average of 4.3 years of follow-up. For total nodal irradiation, the relative risk for acute non-lymphocytic leukaemia was 28.0, and for total-body irradiation, 7.0, both significant. Greene et al. found suggestions of a correlation between cumulative radiation dose to the marrow and relative risk of subsequent

acute non-lymphocytic leukaemia, independent of chemotherapy effects, but the numbers were small and no dose-response pattern could be derived from the data.

298. In this study, the relative risk after combined chemotherapy and radiotherapy was 6.0, also significant ($p < 0.05$). Controlling for chemotherapy, there was still an increasing acute non-lymphocytic leukaemia risk with increasing cumulative bone marrow radiation dose, which was significant ($p < 0.005$). The relative risk was 8.1 for doses greater than 7 Gy, compared to doses less than 7 Gy. When acute non-lymphocytic leukaemia patients were compared to a fourfold larger set of non-Hodgkin's lymphoma patients without subsequent acute non-lymphocytic leukaemia, a risk coefficient of 0.03 additional cases of acute non-lymphocytic leukaemia per 10^4 PYGy was estimated.

299. It may be that the lower doses generally given to treat non-Hodgkin's lymphoma patients had produced acute non-lymphocytic leukaemia, whereas the higher doses given to treat Hodgkin's patients has had a cell-killing effect and produced no acute non-lymphocytic leukaemia.

300. These patients received total-nodal, hemi-body or total-body irradiation or some combination thereof, exposing large volumes of marrow to a relatively low and fractionated dose, with single doses of about 0.1 Gy given over a period of months and totalling a few Gy. Hodgkin's patients, by contrast, typically receive 2 Gy per day, administered over a period of weeks and leading to a cumulative dose of tens of Gy [G3] with substantial cell sterilization, which may explain the absence of radiogenic acute non-lymphocytic leukaemia in them.

301. Danish patients treated for Hodgkin's disease and non-Hodgkin's lymphoma had the following combined relative risks of developing acute non-lymphocytic leukaemia: 8.4 within 10 years of initial treatment and 8.9 thereafter [S39]. Other patients in this registry developed an excess of lung cancer (RR = 1.8), female breast cancer (RR = 2.1), and bladder cancer (RR = 2.6); the Connecticut Tumor Registry did not find an excess of bladder cancer, but did confirm breast and lung cancer, along with thyroid and buccal cavity cancer [C14].

302. Patients immune-suppressed for renal transplant seem, like Hodgkin's patients, to be susceptible to infection by human papilloma virus and hence to a risk of cervical and related tumours [K8, S21]. In these studies, the effects of irradiation and of chemotherapeutic immune suppression cannot be separated; in any case, the effects are not dose-dependent, because the result is not a directly radiogenic cancer but a susceptibility to cancer clearly produced by unrelated agents.

3. Leukaemogenesis in cancer radiotherapy generally

303. Curtis et al. have reported the results of a survey of 440,000 patients in the United States treated for all types of cancer [C8]. They assessed the risk of leukaemia, specific to type of treatment administered for the primary cancers. A significant excess of leukaemias in general and of acute non-lymphocytic leukaemia specifically, for those patients given chemotherapy (Table 29), is described. Relative risks associated with exposure to ionizing radiation in the absence of chemotherapy were inconclusive: small, non-significant excesses were observed for cancers of the mouth, stomach, rectum, larynx, lung, connective tissues, breast, endometrium, ovary, prostate, testis, bladder, kidney and renal pelvis, and for multiple myeloma. The excess risk of bladder cancer associated with radiation for primary ovarian cancer agrees with the results found in the study of ovarian cancer patients discussed above [R11]. The overall relative risk of acute non-lymphocytic leukaemia among patients receiving neither radio- nor chemotherapy was 1.2 (not significantly different from 1.0); the corresponding risk was 2.5 with radiation and 4.5 with chemotherapy, both significant at the 1% level. All of the elevated risk was due to acute non-lymphocytic leukaemia. These patients may develop increasing relative risks with time, and solid tumours may also arise. Studies in the Connecticut Tumor Registry [C14] showed an increase in leukaemia after radiotherapy for cancers of the uterus or ovary, but chemotherapy as a joint cause was not examined in detail.

304. Although only three (3.9%) of the tests shown in Table 29 were significant at the 5% level, the overall result (not shown) was significant at $p < 0.01$ in both radio- and chemotherapy groups. Hence, the results do not appear simply to be statistical artefacts of multiple testing.

305. A case-control study of radiation-induced leukaemias in cancer patients has recently been reported, based on United States tumour registries from the states of California, Connecticut, Kansas, and Massachusetts [B26]. Cases consisted of individuals with two cancers, the second being leukaemia occurring more than one year after the diagnosis of the first tumour, and controls consisted of those with no second tumour. Controls were matched on sex, age at first diagnosis, site of first cancer, and survival after first tumour diagnosis; matching was 2 to 1. Chronic lymphocytic leukaemia was considered separately from all other leukaemias, which were pooled. This study involved 166 chronic lymphocytic leukaemia second tumours, 232 second leukaemias, and 781 controls.

306. As chronic lymphocytic leukaemia has not yet been shown to be a radiogenic tumour, it served as a data quality control. The relative risk of chronic lymphocytic leukaemia after radiotherapy was 0.7, that is, there was no significant difference from unity. For all other leukaemias collectively, the relative risk was 1.6 for all irradiated sites and 2.4 for only trunk sites. Both values are statistically significant. Among specific-site cancers, those of leukaemia after breast and uterine corpus were elevated, and in this and all other regards the results are similar to those of other studies of radiation of active bone marrow in adults. Results after cervical cancer were positive, with RR = 2.3, but not significant. No dose data were

available, and no information about the nature of the radiation treatments was given.

307. Study of the Danish tumour registry has found relative risks greater than 1 for acute non-lymphocytic leukaemia following initial irradiation to treat for head and neck cancers (RR = 1.1), genital cancers (RR = 1.9), female breast cancer (RR = 2.7) and lymphoma (RR = 8.4) [S39]; with the exception of lymphomas and breast cancers, for which the relative risk was 2.3, the risk for acute non-lymphocytic leukaemia 10 years after irradiation was not significantly different from 1.0.

308. The question of whether an interaction exists between radiation and chemotherapy in leukaemogenesis is of importance [see also G3, G9]. The incidence of excess acute non-lymphocytic leukaemia in Hodgkin's and non-Hodgkin's lymphomas is similar, implicating combined therapy or just chemotherapy with alkylating agents. Data from ovarian cancer patients, though inconsistent, suggest that chemotherapy is responsible, but those from non-Hodgkin's patients suggest an interaction. In the non-Hodgkin's patients, whole-body or broad tissue exposure is common, but the relative risks of acute non-lymphocytic leukaemia following total-body or nodal irradiation are both elevated (7.0 and 28.0, respectively). The results of the general study [C8] were inconclusive regarding radiation but agreed with other studies in showing a marked chemotherapeutic effect. Good dose-response data are not available from these general cancer-treatment surveys.

4. Adults exposed to radiation for treatment of benign conditions

(a) Haematopoietic tissue

309. The haematopoietic system, or some portion of it, is in the field of most radiation exposures. This system is actively mitotic throughout life and, with its own process of differentiation and cell division, is histologically distinct among tissues. It also behaves epidemiologically in a different manner from other tissues in regard to radiogenic cancer, in which respect it is similar only to bone cancer after brief exposures [C4]. Despite the sometimes negative findings of the above-mentioned studies on the effects of radiotherapy to treat cancer, where doses are often high, the haematopoietic system is highly vulnerable to radiation carcinogenesis. There are relevant haematopoietic data from most cohorts exposed to radiation; the results are remarkably homogeneous and permit a fairly unambiguous characterization of the risks.

310. *Leukaemia.* The bulk of our information comes from Japan and the British studies of ankylosing spondylitis patients, who received only short-term exposures. Before reviewing these data, the results of other exposures, in particular, those which occurred over long time periods, will be summarized. These results come from Thorotrast patients, from other patients given radium-224, from women exposed to radiation to treat gynaecological disorders, from radiation workers and from radium dial painters.

311. Thorotrast (thorium dioxide) is an alpha-emitter that was used from about 1930 through the early 1950s for a variety of diagnostic roentgenographic purposes. Two series of European patients have been followed in detail and have shown an excess of haematopoietic tumours [K16, M25]. In one, involving 3,772 Portuguese, Danish, and German patients [M25], the total cumulative whole-body dose was determined, 30 years after treatment, to have averaged 2.7 Gy following the use of 25 ml of Thorotrast, on average. The first appearance of leukaemias was eight years after treatment, with cases continuing at least to 1978. If, in fact, acute granulocytic leukaemia (AGL) can be induced at all by high-LET radiation, the small number of acute granulocytic leukaemia cases seen in this group of patients may be an indication of cell sterilization having occurred. Conjectures about the dose-response relationship in this and similar instances must be guarded, however, because of the wasted dose and the concentration of the isotopes in bone marrow. The long period of excess risk expression is not inconsistent with the results from single-exposure studies of radiogenic leukaemias, because the emissions in bone-resident nuclides persist indefinitely.

312. In another series of over 5,000 German patients [K16], exposures ranged from 0.5 to 4 Gy after similar Thorotrast dose levels. While the commonest resulting cancers were those in the liver (see paragraph 403), there were 27 leukaemias instead of the two that would be expected. The shortest time to appearance of leukaemia was five years. Most of these tumours were reticulosarcomas.

313. The effects of ^{131}I, a beta emitter, used to treat hyperthyroidism, do not appear to include leukaemia [H12, H14].

314. Radiologists who entered their profession between 1920 and 1939, had an increase in leukaemia, but those entering thereafter have shown no effects [C4, M18]. The doses received are difficult to estimate, but probably range from 6 Gy for those entering in the 1920s to 2.4 Gy for those entering in the 1930s. The extended time of exposure did not reduce the risk below that observed after single high-dose, high-dose-rate exposures [M18].

315. Some cases of leukaemia have been found in radium dial painters [C4, P19], but it is not obvious whether there has been a significant excess. Also not yet established is the possibility that these individuals have experienced an excess of myelomas. The data are reviewed in [R10]. One difference between the excess leukaemias associated with ^{232}Th and the apparent absence of leukaemia after exposure to ^{226}Ra and ^{224}Ra may be the length of time that the individual is exposed. The reasons for this difference require further study.

316. Table 25 shows that excess leukaemias were observed in several groups of women treated for benign gynaecologic disorders [W6]. In the benign disease patients, the risk of leukaemia declined as doses to the pelvic marrow increased. In a series from Massachusetts, United States, [B12], the latency period was similar to that in the Japanese survivors and the spondylitics; however, the numbers were quite small.

317. Detailed studies of the projection effects in Japan will be presented in chapter VI of this Annex; however, the T65 data on the Japanese atomic bomb survivors [Life Span Study (LSS)] and the British ankylosing spondylitis patients have been compared as well as analysed jointly [D11], based on the data described in [K7, S31]. The relative risks for leukaemia are given in Table 30. In both studies, the first manifestation of excess risk occurred within five years after exposure; the relative risk rose thereafter, and persists for at least 40 years [S49].

318. If one eliminates those survivors under 15 years of age at the time of the bombings and focuses on the risk within the first 20 years after exposure, the relative risk in the spondylitics (3.37) is much less than that in the Life Span Study group (13.50). Darby et al. [D11, D20] showed that in this restricted subset there was no trend in relative risk with age at exposure, in either group. There was no evidence in either study for an increase in chronic lymphatic leukaemia. There was no significant difference in relative risk between males and females in either study.

319. Age-specific relative risk models were fitted by Darby et al. [D11, D20]. For both groups of exposed individuals, the best model had the relative risk declining with time since exposure. In Japan, an age-at-exposure effect occurred, but it was due solely to the presence of individuals under 15 years old at the time of the bombing. The rate at which relative risks for acute leukaemias declined with time since exposure was a function of age at exposure, but the limited data on chronic leukaemias do not support such a difference. Figure III shows that there is a sensitive period for exposure under age 10, after which relative risk is roughly constant.

320. In Muirhead and Darby's analysis of model fitting to cancer data [M36, M37], in which the ankylosing spondylitis and Hiroshima data were studied separately, the constant relative (but not the constant additive) risk model was found consistent with the ankylosing spondylitis data; however, the constant additive (but not the constant relative) risk model provided a satisfactory fit for the Japanese data. Intermediate models provided somewhat better fits to both series, although not statistically significant, but the best-fitting parameter values for those models were markedly different for the two sets of data.

321. There are dose-response data of a general kind for women irradiated for benign or malignant gynaecologic conditions [W6]. Table 31 provides relative risks of leukaemia as a function of total pelvic marrow dose and mean marrow dose from several studies. In support of the cell-killing argument, raised earlier to explain the level of leukaemias in cervical cancer patients, it appears from these data that higher local doses are associated with lower relative risks of leukaemia.

322. Figures II and V provide a graphic summary of the relationship between single doses (or short-term exposures) and leukaemogenesis; Figure V has been used frequently to summarize the leukaemia findings in Japan. It is based only on the Japanese data [17] and on earlier dose estimates. The pattern has persisted in recent reports from Japan [O3, P6], but a thorough analysis of the revised dose estimates (DS86) is not yet available. For all acute leukaemias pooled, there is a distribution of excess cases which is a function of age at time of exposure. The younger the age at exposure, the shorter the latency. These patterns have been fitted empirically to a log-normal distribution.

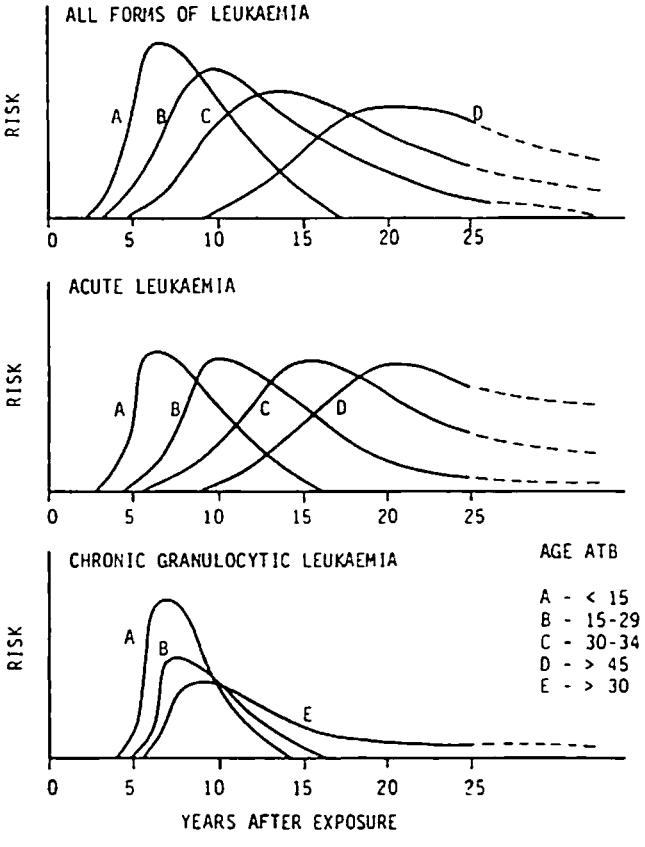

Figure V. Schematic representation of the relationship between age at the time of the bombings and time of occurrence of leukaemia in Japanese atomic bomb survivors receiving more than 1 Gy.
[17]

323. Figure V shows that for chronic granulocytic leukaemia, there is less variation, with age at exposure, in the projection pattern following radiation exposure [14, 15, 16]. Excess risk virtually disappears after about 20 years [O3, P6]. As with acute leukaemias, a log-normal distribution adequately fits the chronic granulocytic leukaemia data. Chronic and acute leukaemias may have a similar pathobiology, but they differ in the absolute effects produced by a given exposure level, so that the two types of tumour should not be considered jointly. The most recent data from Japan suggest that there may be some residual excess risk, even 40 years after exposure [S49].

324. Figure VI provides a different view of the T65 Japanese data, comparing them with the relative risk for leukaemia in the spondylitics from Darby et al. [D11]. Two variables were modelled, age at exposure and time since exposure. Interaction between these is modelled by standardizing each in terms of the other. The figure shows no apparent trends in susceptibility

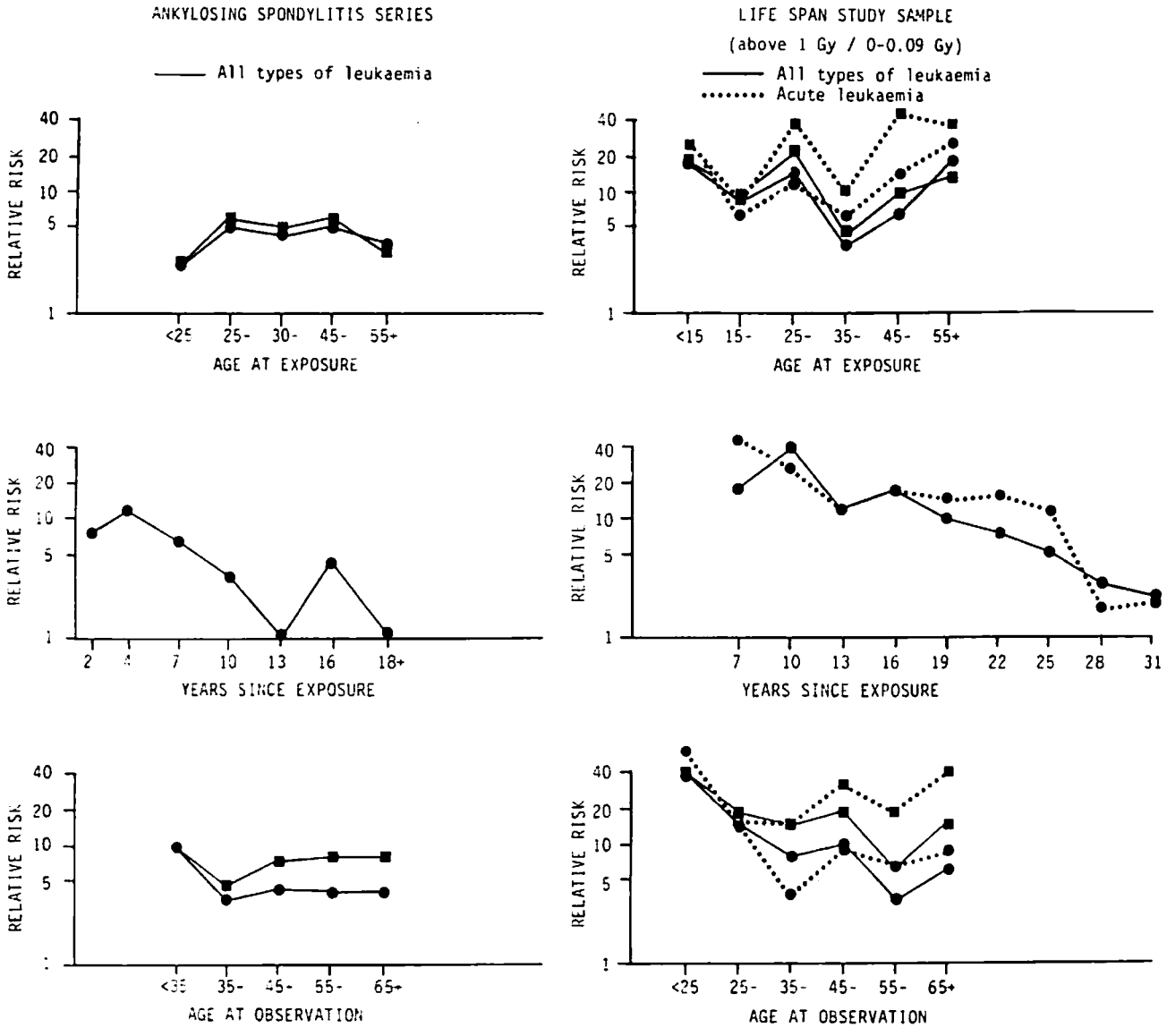

Figure VI. Relative risk of leukaemia in relation to age at exposure, time since exposure, and age at observation for the ankylosing spondylitis series and for the Life Span Study sample (T65DR doses). Lines marked with circles are for unadjusted data. Lines marked with squares are adjusted by including a linear trend in log (relative risk) with time since exposure in the model. For the plotted points, time since exposure has been chosen so that the initial point coincides with the unadjusted value.
[D11]

(RR) with age at exposure, other than the excess susceptibility in those under age 15 (children) in Japan, discussed above. Darby et al. found evidence of a linear trend in log RR with time since exposure, so the data were analysed both including and not including this trend (see figure). In neither series was there evidence that the rate at which the excess risk declined with time since exposure was a function of age at exposure.

325. Relative risk is higher in the Life Span Series, even after eliminating childhood exposures; this could be due to cell-killing in the spondylitics or to the effective amount of marrow exposed. The difference between acute and other leukaemias can be seen in Figure VI; when adjusted for a trend in time since exposure, the pattern of relative risk for acute leukaemias differs substantially from the unadjusted pattern. The most recent spondylitis data [D21] show that leukaemia excess RR does not disappear completely by 25 years after exposure.

326. It should be kept in mind that the point of reference for the discussion in this section is the exposure of non-cancer patients in view of the possibility that cancer patients are more cancer-prone than the population at large. Indeed, the regular pattern of effects seen in non-cancer patients is less perceptible in patients treated for cancer. In particular, individuals irradiated for leukaemias and lymphomas have not consistently manifested leukaemia as a second radiogenic cancer [G3].

327. *Multiple myeloma.* Multiple myeloma remains one of the most enigmatic candidates for the list of radiation-induced malignancies. The Japanese atomic bomb survivors have been studied in regard to multiple myeloma by Ichimaru and colleagues [H5, I2]. Data were based on 29 cases occurring in the period 1950-1976. The age-standardized relative risk increased with absorbed dose in the bone marrow, with no differences between cities or sexes. Based on the T65 doses, the excess risk estimate is 0.48 cases per

10^4 PYGy. As in other series, latency is 15-25 years, with a long-lasting period of excess risk (at least 30 years). A longer latency after younger ages at exposure follows a carcinoma-like pattern. The spontaneous incidence of multiple myeloma increases with about the 5-6th power of age, which is similar to the behaviour seen for carcinomas.

328. Darby et al. [D11, D20] found evidence of elevated myeloma risk in their combined analysis. An increase in risk has also occurred among radiologists who entered their profession since 1940 [M18], and the Hanford radiation workers, as well as other exposed cohorts, appear to have experienced a small excess of multiple myelomas [C10, G12, H16].

329. Many chronically exposed occupational groups were reviewed by Cuzick [C10]: nuclear workers, radium dial painters, uranium millers and miners and radiologists. The overall relative risk is between 1.4 and 2.9. Multiple myeloma was also more common in radiation workers in the United Kingdom, based on a recent study of the Sellafield nuclear workers [S54], though not in another of the employees of the United Kingdom Atomic Energy Authority [B22].

330. Cuzick [C10] reviewed the effects of diagnostic and therapeutic exposures on the induction of multiple myeloma. The exposed groups included Thorotrast patients, spondylitics, and groups exposed during fluoroscopy or for treatment of gynaecologic disorders. Results were similar: although many studies yielded confidence limits that included a value of 1.0, there was, overall, a small increase in the relative risk, about 1.6 (range 1.1-3.3).

331. Cuzick also assessed the relative risk according to type of radiation to determine if the effects of internal alpha-emitters (radium dial painters, Thorotrast patients and nuclear workers) had been different from those of gamma-emitters or x rays (atomic bomb survivors, radiologists, nuclear workers, spondylitics, fluoroscopy patients and gynaecologic therapy patients). His summary is given in Table 32. Uterine cancer patients are an exception to the pattern of excess risk, and for them the risk is higher with high-LET internal emitters than with low-LET sources.

332. The IRSCCP cervical cancer study [B12] found only marginal support for an excess of multiple myeloma; however, the general deficit of leukaemias in this group, attributed to cell sterilization, may be important. Both leukaemias and myeloma are B-cell diseases, as are some cases of chronic lymphocytic leukaemia, which has never appeared to be radiation-related.

(b) Non-dividing tissue

333. Many tissues in adults are composed of cells that do not normally or frequently divide. These include muscle and the neuronal tissue of the brain and central nervous system (CNS). As reviewed earlier, tumours of the central nervous system are known to occur following exposures of the head and neck in children; rhabdomyosarcoma may also occur, rarely, as the second tumour in retinoblastoma or other childhood cancer patients. However, some of these tissues are still growing in early childhood, and it would be worthwhile to determine whether normally non-dividing tissue is radiosusceptible.

334. In general, spontaneous adult-onset tumours in non-dividing tissue appear to be very infrequent. The most recent comprehensive reviews of radiation carcinogenesis, including the UNSCEAR 1977 Report, have either ignored radiogenic cancer at these sites or judged it to be rare or even absent [B6, C4]. Heavily irradiated parts of the central nervous system, excluding the brain, in ankylosing spondylitis patients have manifested excess cancer, although this finding is based on only four cases (0.5 expected) in 14,000 patients, two of which may already have been present at the time of irradiation [S28, S31]. The spinal cord is heavily irradiated in such patients. The paper by Smith et al. [S31] appears to be the first substantial report suggesting that these tissues are radiosusceptible. In the atomic bomb survivors, there is a similarly elevated relative risk of cancer associated with heavy irradiation of the spinal cord and nerves [S23], suggesting that the data in the spondylitics may be reliable. The cell type of these cancers was not given [S23, S28, S31].

335. Other individuals who may have received heavy doses to the central nervous system include the radium dial painters, who did not, however, experience an increase in brain tumours [R10]. Swedish patients treated with ^{131}I for hyperthyroidism exhibited only a slight, non-significant excess (cell type not specified) [H14]. Radiation workers, including radiologists, have shown similar results [e.g., M18].

336. A case-control study in Los Angeles, California, United States, of women irradiated for medical and dental diagnostic purposes found a relative risk of 4.0 for all forms of meningiomas after exposure under the age of 20 and of 2.1 for patients irradiated before 1945, both of which values are statistically significant. The majority of tumours arose after age 50. The authors think that there is an early age susceptibility, although these women, albeit aged less than 20, were not all irradiated as children [P18].

337. In sum, many individuals have received irradiation to considerable areas of muscle, nervous, and other connective tissue. The fact that tumours have only rarely arisen in these areas is in general agreement with the requirement that a tissue be mitotic to be radiosusceptible. If it can eventually be shown that radiation induces mitosis, or if these tumours are actually in mitotic cell types (e.g., glial cells), radiation may increase tumours in these tissues proportionately to their natural incidence. However, these tumours are so rare naturally in adults that it is difficult to detect a small increase from the available data.

(c) Dividing non-epithelial tissue

338. While much of the nervous and connective tissue of the body is not normally mitotic in adults, this is not true of all tissues. Notable exceptions are glial cells and the cells involved in the remodelling of

bone. These divide, at least in response to stress or to demands for repair. As has already been noted, these tissues are radiosusceptible in childhood, and osteosarcomas and neuroblastomas of various kinds are among the most consequential childhood tumours.

339. The data on risk for actively dividing mesenchymal tissue are strongest, and clearest for cancers of the tissues in the periosteum, i.e., osteosarcoma and other bone cancers. Indeed, ionizing radiation is the only well-documented risk factor for such cancers.

340. There are, basically, three different kinds of exposure for which data on bone cancer exist: persons exposed to bone-seeking radionuclides, internally deposited, often of high-LET alpha-emitters; persons exposed to high doses of external irradiation; and adults irradiated to intermediate doses during a single exposure in Hiroshima and Nagasaki. Internal emitters have been of two types, long and short half-life isotopes, with differing epidemiological results.

341. Individuals receiving exposure to bone-seeking internal emitters include watch dial painters whose mastoid and other cranial sinus epithelial linings were exposed to radon decay products and whose bones were exposed to ^{226}Ra and ^{228}Ra and patients who were given radioactive x-ray contrast medium (i.e., Thorotrast). Underground miners, who are exposed to radon gas, have not exhibited an excess of osteosarcomas. There is much literature on this subject from animals (see Annex B of the UNSCEAR 1986 Report) [U1]. Osteoblasts are the most common cells of origin of osteosarcomas. The internal high-LET alpha-emitters, ^{224}Ra and ^{226}Ra, are among the best-documented radiogenic causes of bone cancer. Radium-224, a short-lived isotope, emits radiation on the surface of the bone where the active target cells are located, whereas ^{226}Ra, a long-lived radioisotope, is distributed more evenly throughout the bones, and its emissions are more effectively shielded from these cells.

342. Radiogenic osteosarcomas tend to occur in the same locations of the skeleton in which spontaneous osteosarcomas occur, especially near the epiphyses of rapidly growing long bones; risk is highest in the knee joint and lowest in the vertebrae. Even with high spinal doses, there has been only a single vertebral osteosarcoma in 14,000 ankylosing spondylitis patients [E3]. Bone sarcomas have been observed in individuals first exposed to ^{224}Ra at ages ranging from 2 to 56 years [M22].

343. The effects of exposure to long-lived alpha-emitting radioisotopes is largely known from the experience of the radium dial painters and other individuals totalling about 3,000 in number; this work is summarized in [R10]. An excess of osteosarcomas in various bone and head/sinus carcinomas has been observed; for example, Polednak and his colleagues [P19], in a study of 634 women who worked in the radium dial painting industry from 1915 to 1929, observed 22 deaths from bone cancer where only 0.27 had been expected on the basis of age/time/cause-specific death rates for United States females. Most, if not all, of these cases were probably due to radiation, since the tumours are otherwise quite rare and the alpha-emitters provided continuous exposures. The excess occurred over an extended time, from seven to 59 years. The radioisotopes ^{226}Ra and ^{228}Ra have long half-lives (1,600 and 6.7 years, respectively) and are removed slowly from the bone. It is not possible to quantify the latency period of these tumours in terms of time after exposure, since the exposure was continuous. In such individuals exposure is not measured in terms of Gy but in terms of Bq, the inferred total systemic activity; however, the total exposure of the bone ranged from about 0.1 to 500 Gy. In 2,135 patients injected with Thorotrast, which contains ^{232}Th, a long-lived alpha-emitter, there were three bone cancers when one had been expected. Tumours of the sinuses of the head in these individuals may be due to the presence, for appreciable amounts of time, of radon gas (^{222}Rn) in the sinuses [e.g., R13].

344. The experience with persons exposed to short-lived alpha-emitters is different, because the exposure can be dated and the dose and dose-response relationship more easily quantified. The most important cohort is a group of 898 German patients given injections of ^{224}Ra to treat ankylosing spondylitis, bone tuberculosis and other diseases [M22]. These individuals experienced an increase in osteosarcomas, with onset from 3.5 to 25 years after the initial injection, which is very similar to the onset observed in radiogenic leukaemias (Figure VII). Such an early onset period contrasts with the continuing occurrence of bone cancer after ^{226}Ra exposure; presumably the shorter half-life makes the ^{224}Ra exposure more like a brief exposure. All ages were affected.

345. The mean dose to these patients was 11.0 Gy in children, administered over 11 months, and 2.05 Gy in adults, over six months. The distribution of induction times was the same in adults as in children. As noted earlier, this is not consistent with causation being a function of the number, or proportion, of actively dividing cells, which should be greater in children.

346. Unlike in groups exposed to radioisotopes having longer half-lives, there have been no sinus (paranasal, mastoid air cells) cancers in this group. This may be due to the fact that the decay products of ^{224}Ra do not include long half-life gases [M22].

347. The BEIR III Committee attempted to summarize the dose-response pattern, and their table of risks is reproduced as Table 33. Original dose-response patterns, developed by Rowland, were modified to remove exponential terms of the form $\exp(-cD)$ because these were numerically close to 1.0 [C4]. Both linear and quadratic forms have been given because it was judged impossible to differentiate confidently among the models based on the available data. For protracted exposure to alpha emissions from ^{224}Ra, Mays and Spiess have estimated 200 bone sarcomas per 10^4 PGy of average skeletal dose. They estimated a ratio of 7.5 for the effective endosteal dose to a given level of average skeletal dose; based on this, the risk coefficient is 27 per 10^4 PGy, as shown in the table [C4, M22]. As most of the risk experienced by the series of patients given ^{224}Ra in the Federal Republic

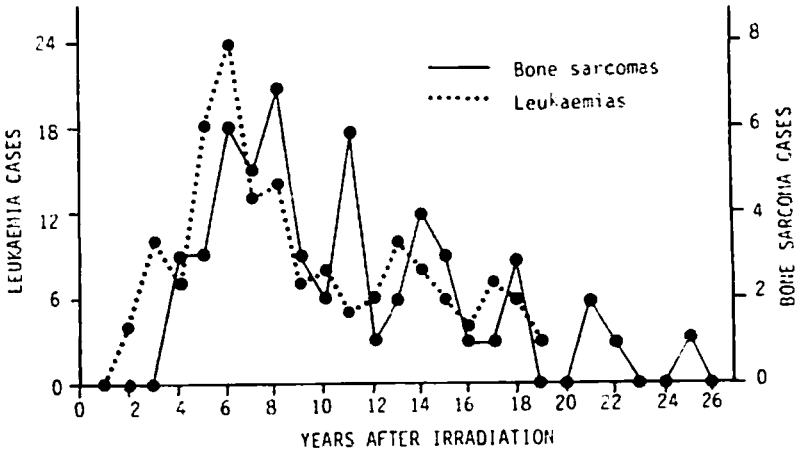

Figure VII. Bone sarcoma incidence in radium-224 patients and leukaemias in the atomic bomb survivors. The distribution of appearance times is remarkably similar for leukaemias, following prompt radiation, and for bone sarcomas, following relatively brief radium-224 irradiation.
[M22]

of Germany seems to have passed, this estimate should be close to the final risk for this series [M22]. Dose-response curves are difficult to compute because of the different types of radiation involved in exposed cohorts, but linearity cannot be excluded [C4].

348. Thomas and McNeill have fitted dose-response patterns for bone and head sinus cancers to a model with cell-killing:

$$RR = (1+bD^c)(0.5)^{D/d}$$

where b, c and d are constants to be evaluated in fitting the model [T11].

349. For bone cancer, these authors developed a relative risk estimate based on data from the watch dial painters and the radium-injected patients in the Federal Republic of Germany. They estimated the absolute excess risk to be 6.4 per 10^3 PY and MBq (2.36 per 10^4 PY and μCi) from exposure to long-lived ^{226}Ra and ^{228}Ra, where PY are counted after a five-year post-initial-exposure latency period and the dose is in terms of systemic intake. For short-lived ^{224}Ra, the estimates are 1.8 cases per 10^4 PYGy for juveniles and 1.0 for adults, measured in terms of skeletal dose. The authors found some evidence for non-linearity and cell-killing. These data are summarized in Figure VIII. The highly curvilinear nature of the dose-effect relationships should be noted when deriving risk coefficients in the low dose range.

350. For head carcinomas the authors used a 10-year minimum latency period and found that a linear model with cell-killing fit the data best. They estimated the risk of additional cancer deaths to be 5.4 per 10^4 PY and MBq (1.98 per 10^5 PY and μCi). The dose-response pattern fitted by them is given in Figure IX.

(d) Epithelial tissue

351. The epithelial tissues form the interactive surface of the respiratory, digestive, genito-urinary and secretory systems. As such, these tissues are the exposure interface between the inside of the body and the environment. In radiation exposures, unlike many chemical exposures, the protective normal mechanisms, such as buffering layers of mucus, are ineffective. Epithelial tissues are all characterized by layers of stem cells, which normally divide throughout life to produce the differentiated functional cells that are the basis of the organ systems. The number of stem cells, their

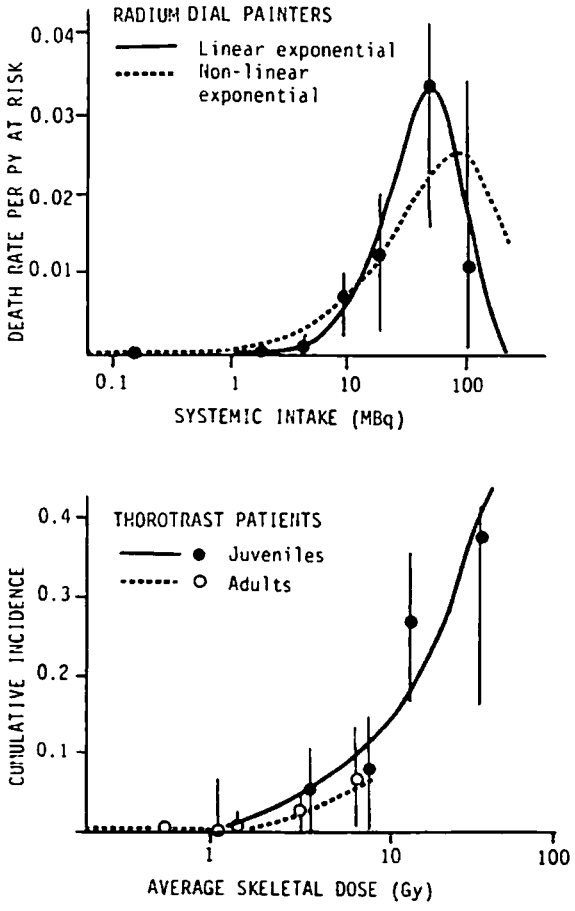

Figure VIII. Bone sarcomas in radium-exposed persons in relation to systemic intake or average skeletal dose.
[T11]

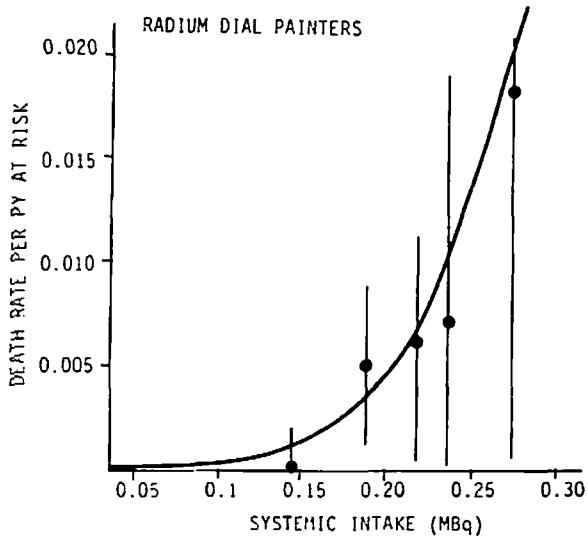

Figure IX. Death rates from head carcinomas in radium dial painters.
[T11]

architecture, turnover rates and metabolism differ for different organs, providing different size targets for radiation. In addition, epithelial tissues may be stimulated to divide or grow by a variety of agents, including hormones, irritation, cell damage and so on. Despite these differences, it would be valuable, from the standpoint of biological knowledge, to determine whether any generalizations can be made about the susceptibility of epithelial tissues.

(i) Skin

352. As a large and widespread epithelial organ, the skin is irradiated during most radiation exposures, yet the data on radiogenic skin cancer are fragmentary and inconclusive. That an effect exists has been known since shortly after the discovery of x rays. However, expected rates are difficult to determine since reporting is unreliable and skin cancers are not usually fatal. Most reports have centred on individual cases rather than on larger populations. The Japanese data reveal no radiation effect, and there are no estimates of risk available from diagnostic or therapeutic thoracic exposures. Occasional studies report excess cases, but the doses have usually been over 10 Gy.

353. The relative risk of skin cancer for radiologists in the United States has been between 2.4 and 3.3 over the past 65 years [M18]. Exposures have varied greatly over this period. In a series of about 2,200 children irradiated for tinea capitis, the relative risk was 7.1 [C4, S16, S27], and a relative risk of 5.4 was found in children who were thymus-irradiated [H1, C4]. Other studies of skin cancer following various types of radiotherapy have found similar results.

354. Table 34 provides data on the relative risk of skin cancer among whites exposed to treat tinea capitis, in terms of age at exposure and time since exposure [C4]. The expected numbers of cases were derived from national data from the United States; given the unreliability of such data for skin cancer, the excess could be attributable to closer follow-up or other biases. However, trend analyses with respect to both age and time since exposure are significant at $p < 0.0001$ [C4]. Tumours arose in irradiated areas of the scalp in 41 of the 2,200 irradiated children, many of whom had multiple lesions, but in only three controls [S27].

355. A study of Czechoslovakian uranium miners has shown a relative risk of about 4.6 for skin cancers, primarily basal cell cancer of the face; alpha-radiation doses are estimated to have been about 1 Gy [S30]. However, since alpha penetration to the basal cell layer is doubtful, this finding is not universally accepted [R7]. A recent study of six groups of Czechoslovakian miners, updating the earlier data, has again confirmed an excess of basal cell carcinoma of the skin in uranium (but not in coal) miners [S51].

356. A total of 6,405 patients treated for benign diseases of the head and neck in the Netherlands were ascertained 19 to 48 years after treatment. Thirty skin tumours in 21 patients were diagnosed, and a dose-effect relationship of 40 carcinomas per 10^4 PYGy was estimated [V4].

357. Dose-response patterns have not been accurately estimable for radiogenic skin cancer. If any conclusion is warranted, it is that the skin is susceptible to radiation, but that excess cases are not common, especially at doses of less than 5-10 Gy. There is no apparent plateau after which risk subsides. Since skin cancer is rarely fatal, is often not reported and is associated with exposure to sunlight [S27] and other factors, it is difficult to generalize about the latency period. In the tinea capitis series, the latency appeared to be 20 years or more [S27], and the risk persisted for at least 45 years ([C4]; see also [V4]).

358. Doses in the tinea capitis series were 3-6 Gy [S27]. From the tinea capitis and the thymus-irradiated children, the BEIR III Committee estimated risk to be 1.02 and 0.44 excess cases per 10^4 PYGy, respectively [C4]. However, several chest-fluoroscopy studies have reported fewer cases than these estimates predict. This deficit may suggest a non-linear pattern, e.g., a threshold, but satisfactory or comprehensive risk estimates for radiogenic skin cancer do not currently exist.

359. In a study of the "soft" x-rays (Grenz, or Bucky, rays) used in Sweden to treat a variety of dermatological conditions in 14,237 patients from 1949 to 1975, Lindelof [L15, L17] found a RR of 1.45, significant at the 5% level, of non-melanotic skin cancer. Malignant melanoma was not elevated (RR = 1.07).

(ii) Breast

360. In this century, large numbers of women have received irradiation to the chest to treat a variety of medical conditions. Among these are chest fluoroscopy administered to follow the progress of artificial pneumothorax treatment, radiotherapy for various non-malignant breast disorders, including post-partum mastitis, and the radiation received by the atomic bomb survivors.

361. Although there are difficulties in estimating doses and in other aspects of these studies, risk appears consistently to increase with increasing dose and to decrease with increasing age. The bulk of the information suggests that the dose-response pattern is linear, although one Canadian fluoroscopy series has obtained a better fit with a quadratic model [H6]. Radiogenic breast cancers occur at the same ages at which breast cancers occur naturally; elevated risk appears to persist throughout life, after an initial latency period. Latency is rather long (> 10 years); it may also vary, being an inverse function of age at exposure. In general, cases of exposure at post-menopausal ages have not been studied in numbers sufficient to allow a reliable assessment of effects, and there may be a decreased susceptibility with increasing age [T14]. However, exposed Japanese of this age have a relative risk of 3.1 [L6, T6, T14]. The possibility of a cohort effect, associated with the increase in breast cancer in Japan since 1945, should be considered. As discussed earlier, it has now been shown that exposure at ages below 10 leads to a substantial risk of breast cancer.

362. The details of the relative risk of breast cancer from incidence data collected in Hiroshima and Nagasaki for the period 1950-1980 are summarized in Table 35 [T14]. A trend of increasing susceptibility with decreasing age, within a given dose level, can be seen. Figure X shows the decrease in relative risk with increasing age at exposure, for the 0-0.09 and the 0.5+ Gy (T65) groups [T14].

363. The Japanese results can be compared with those of two other studies from the United States, a Massachusetts tuberculosis fluoroscopy and a Rochester series of post-partum mastitis patients [B2, C4, L6, S47]. Howe has also reported fluoroscopy data from most provinces in Canada [H6]. Total relative risks, for doses from 1 to 4 Gy, are consistently between 2 and 3, with values of 4-6 for those exposed at younger ages. At doses higher than 4 Gy, most studies have only small samples; however, the largest of these has found a relative risk of 14.6 at high doses [H6]. Incidence data from Japan suggest a corresponding relative risk of about 4 [W5], indicating perhaps that survival from breast cancer depresses the true relative risk estimated from mortality data. Relative risk data from these four study populations are summarized in Table 36. Table 37 provides details on relative, as well as absolute, risk differences for three of the major investigations, subdivided according to age at observation and age at exposure; the similarities in the different data sets can be seen.

364. A recent case-control study of breast cancer following irradiation to treat tuberculosis in Denmark has found no significant increase [S53]. While the study was too small to rule out an effect, it was large enough to confirm that other studies in the literature are not underestimating the risk. A similar negative result, and interpretation, has also been reported by Davis et al. [D27] based on a study in Massachusetts. Doses were smaller than in other series (0.66 Gy) and the average age at exposure higher (28) than in other studies.

365. Acute post-partum mastitis patients have now been followed for up to 45 years, with an average follow-up time of 29 years [S47]. Relative to controls and female siblings of patients, the RR value for breast cancer in the irradiated breast, age- and interval-adjusted, is 3.2 (90% CI: 2.3-4.3). The risk increased by 40% per Gy with an essentially linear dose-response except for a diminution at doses above 7 Gy, with no fractionation effect. A multiplicative projection model was a better fit than an additive one, and the RR did not change with time since exposure.

366. The absolute risk in Japan has been estimated to be between 3.0 and 4.0 ± 0.7 cases per 10^4 PYGy, with a pattern that is roughly linear and no inter-city difference [T9, T14]. Risk coefficients in the various fluoroscopy and mastitis series range from 6 to 8.5 cases per 10^4 PYGy [C4]. As previously noted, with the exception of the Nova Scotia series, these data are consistent with a linear dose-response pattern (see Figure XI and Table 38). The New York mastitis data for uni-lateral breast exposure suggest that doses of 4-14 Gy have a cell-killing effect [B2, C4, L6]. However, for bilateral breast exposure, even at higher doses (in some instances tens of Gy) no downturn in the dose-response curve was observed. The fluoroscopy series, especially in Nova Scotia, were highly frac-

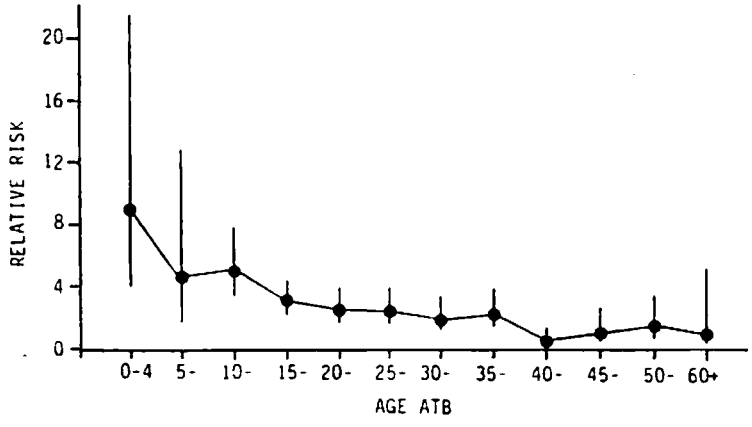

Figure X. Relative risk of breast cancer in atomic bomb survivors for the 0.5 or more Gy relative to the 0-0.09 Gy dose group (T65DR kerma doses). [T14]

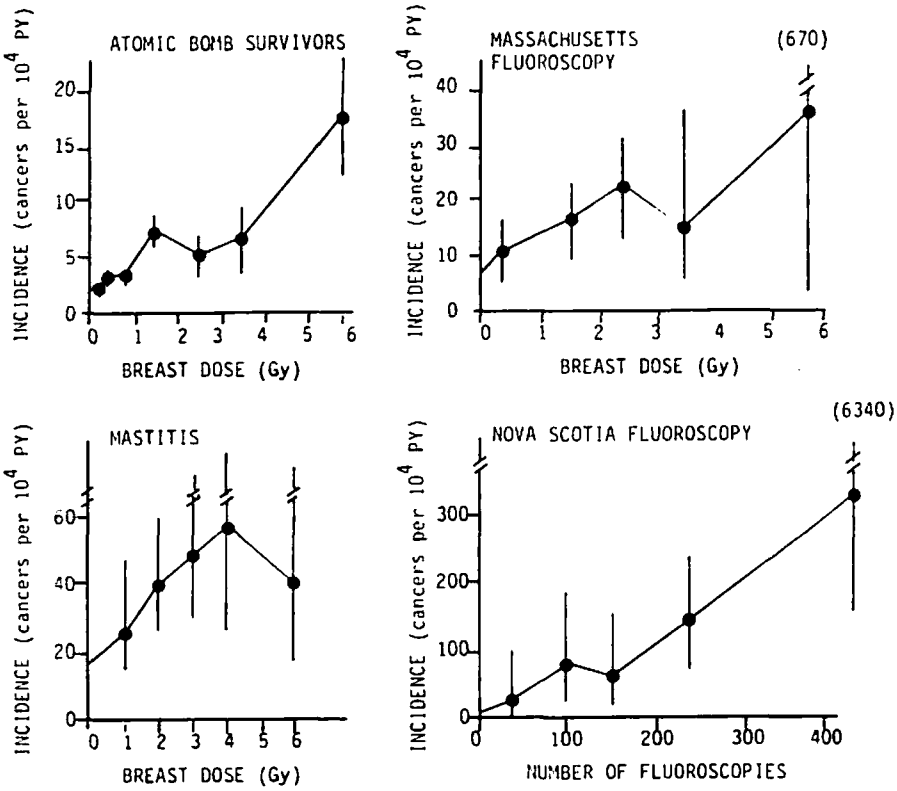

Figure XI. Breast cancer incidence in relation to radiation dose in atomic bomb survivors (T65DR doses), mastitis patients, and fluoroscopic studies in Massachusetts and Nova Scotia.
[B2]

tionated, and this may make a difference at high doses. Mastitis may also have its own biological relationship to breast cancer after irradiation [L6].

367. Besides the slight indication of non-linearity at high doses in [S47], the other exception to a simple linear model is from a record-linkage study of data from nearly all of Canada. A pure quadratic model appears to fit these data best, though a linear-quadratic model fits almost equally well [H6]. The departure from linearity is evident in the lower response per unit dose of women in the Canadian provinces other than Nova Scotia, where the doses ranged up to much higher values [H6]. In Nova Scotia, the patients were examined in the anterior-posterior position (facing the x-ray tube) whereas in the other provinces the patients were mainly examined in the reverse position, resulting in doses per fraction about 20 times smaller. The absolute risk on a linear basis in the range 0-2 Gy, which contains the major fraction of the cancer cases, appears to be about three times smaller for those provinces than for Nova Scotia alone. Based on the evident lack of excess cancer for the lower dose range (0 to 0.99 Gy) in the Canadian study as a whole [H6], this factor would be considerably greater at low doses. This one series contributes the bulk of the data above 4 Gy. Howe argues that because in other instances high-dose information is relatively sparse, and because it is in the high dose range that non-linearity is expected to be most apparent, a linear-quadratic model is the prudent model to adopt in the establishment of breast-cancer dose-response patterns [H6].

368. Two studies have examined the possibilities of synergism between several other risk factors in women irradiated for post-partum mastitis [B30, S37]. These factors include family history of breast cancer, late age of parity, oral contraceptive use, menopausal hormone use and various ovarian-related factors. Women with benign cystic breast disease and those irradiated at the time of their first childbirth were at increased risk, but other women were not.

(iii) Lung

369. Most of the exposures to the breast or chest also involve the lung, and there are several cohorts of individuals who received internal exposures specifically to the lung, principally underground miners who inhaled radioactive radon gas. Exposures to the lung from therapeutic radiation have been experienced by patients with ankylosing spondylitis. These and the other groups have experienced most of the kinds of exposures needed to understand the radiosusceptibility of the lung. In particular, the miners were exposed to moderate doses of high-LET radiation over long time periods, the atomic bomb survivors were exposed to a single dose, and the radiotherapy patients received fractionated, moderate-to-large doses over a short time period.

370. There appear to be no risk differences between males and females, after accounting for the effects of smoking. Most of the available information, however, comes from males; data on both sexes come primarily from Japan and clearly suggest no difference except that which is due to smoking [S49].

371. Relative risks from exposures to brief external x- and gamma-irradiation are 1.2-2.0 [K7, S28, S31, W5]. In the miners, who had variable levels and durations of exposure to inhaled alpha radiation, because relative risks are dose- and duration-dependent and because there may be interaction between the exposures and smoking, this aspect of the data will be discussed in section V.A. The mining data come from uranium miners in Czechoslovakia [S19, S51], the United States [C20, I11, S20] and Canada [C4, G10, H15, M19]; from Swedish metal and Canadian gold miners [A9, D12, E1, M19, R5, R7]; and from a few other reports [C4, R7, T11, T20]. Thorotrast patients were exposed to thoron (^{220}Rn) gas, also an alpha-emitter, as an exhalant; these patients manifest an excess of lung cancer (40 cases vs. 34 expected) after doses ranging from 0.3 to 14 Gy [K16]. The smoking habits of these patients do not differ from those of the general population of the Federal Republic of Germany.

372. Radiogenic lung tumours appear preferentially in the epithelium of the upper bronchial tree, unlike in experimental animals given radioactive inhalants or intratracheal instillation. One mechanism for the upper bronchial effect of natural exposures to radon daughters is the adsorption of the free-ion fraction, that is, ions not bound to dust particles (see Annex G of the UNSCEAR 1977 Report [U2]). Most data suggest that the cell types do not significantly differ from those in non-radiogenic lung cancer [C4, C12, S20].

373. The ages of onset of radiogenic lung cancers are similar in general to those of spontaneous lung cancer; there is little evidence for excess risk before age 35 [C4]. This suggests that the latency period is a function of age at exposure; however, not all of the data are consistent. The minimal latency period has usually been at least 10 years, roughly independent of age at exposure in spondylitis patients and in Swedish [R5] and Canadian miners [C4]. In other mine studies and in the Japanese atomic bomb survivors, latency has been dependent on, and negatively correlated with, age at exposure: early exposure has led to longer latency and, perhaps as a result of increased years at risk or increased years at observation, higher absolute or lifetime excess risks. The data from the United States are not entirely clear. In one study of Colorado miners (where dose rates may have been higher than elsewhere), there was a shorter latency period in exposed smokers than in non-smokers, but doses may have been overestimated due to the way in which exposures were sampled [C4, R5]. Moreover, the follow-up times for individuals initially exposed at younger ages may be insufficient. Excess risk is known to persist for at least 50 years after exposure began.

374. The overall data suggest that the relative biological effectiveness (RBE) of alpha-radiation to the lung relative to gamma-radiation, is 20, although there is much uncertainty in this estimate, largely ascribable to the difficulty of converting data on WLM to absorbed doses in Gy [C4]. A reference conversion is 6 mGy per WLM for mean bronchial dose and usual conditions in mines [I11]. This results in unit risk of 1.0 per 10^6 PY-WLM corresponding to 1.67 per 10^4 PYGy.

375. Thomas and McNeill have fitted the dose-response data to additive and multiplicative models for exposure to alpha-emitting radionuclides [T11, T20]. Results are provided in Table 10. The models fitted were linear cell-killing models of the form (using their notation)

$$R = (a + bD^c)(0.5)^{D/d}$$

where a, b, c and d are the parameters estimated from the dose-response relationship, fitted by weighted least-squares, and R refers to both additive and relative risk (in the case of additive risk, a was set at 0.0, and in the case of relative risk, to 1.0). The second exponential term models cell-killing effects. A linear dose-response is modelled by setting c = 1.0. For details on the justification of this dose-response model, see [T11, T20]. Thomas and McNeill found some evidence of a departure from linearity in the dose-response patterns of the mining data (Figure XII).

376. An extensive analysis of lung cancer in miners exposed to radon daughters has been published, reporting on results from four studies of six miner groups in Czechoslovakia [S51]. The lung cancer rate increased as a function of exposure. Excess risk appeared about 5 years after the onset of exposure, peaked at 20 years, and, though excess persisted, it was no longer significant after 30 years (the approximate limit of follow-up to date in these subjects). Unlike some other studies of miners who began exposure under age 30, there was a detectable excess risk before age 40. However, relative risks were higher with greater age at onset of exposure. The data from the Czechoslovakian uranium miners appear to be essentially complete for group S (miners first exposed between 1948 and 1957) [S51, K28]; the total lifetime risk can thus be calculated directly without the use of a projection model, suggesting an average lifetime risk of approximately $4.5\ 10^{-4}$ per WLM. Other findings of importance were: (a) a documented excess of lung cancer at total exposures less than 50 WLM; (b) an approximately additive effect of smoking; and (c) possible evidence for a cell-sterilizing effect at high doses for small cell lung carcinoma, but not for epidermoid cancers.

377. Further data on the Ontario miners have also become available [M40, M42]. These too indicate that the minimum latency period for appearance of excess lung cancers after first exposure to high concentrations of radon daughters is 5 years, not 10 years as previously assumed. This conclusion is substantiated by studies of the Eldorado uranium miners in Canada [H25, H31]. It also appears that excess lung cancers in these uranium miners reached a maximum about 10-15 years after first exposure and decreased towards zero about 20 years after last exposure [K28, M40, S51]. The risk coefficient derived from the Ontario miners study suggests an average lifetime risk of about $1.7\ 10^{-4}$ per WLM for miners exposed to 1 WLM per year from age 20 to 55.

378. The range of risk coefficients derived from various studies of uranium miners is very broad but is

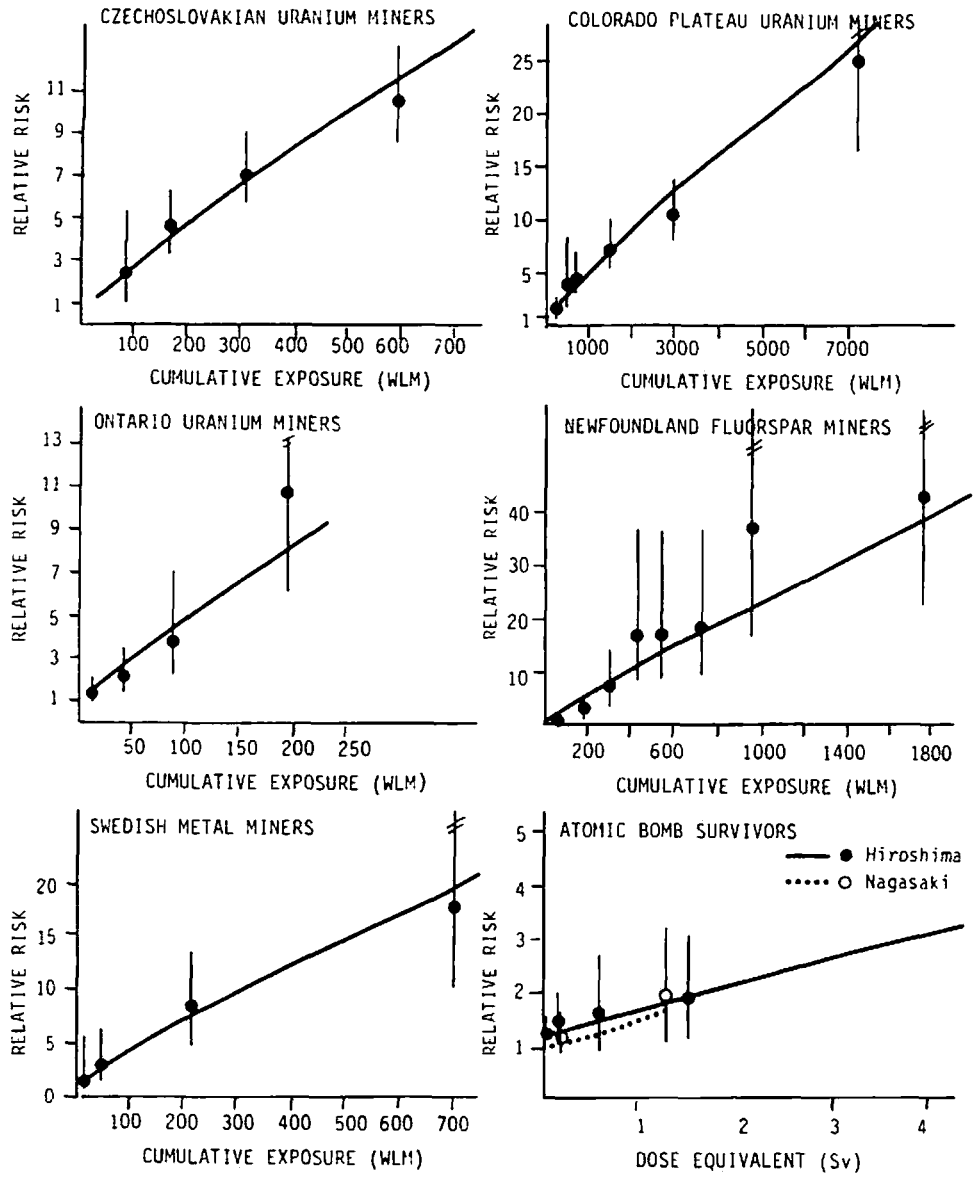

Figure XII. Lung cancer risk in five groups of miners and in atomic bomb survivors (T65DR doses).
[T11]

in general compatible with the central value of about 10 excess cancers per 10^6 PY and WLM (additive risk model) or about a 1% increase in normal incidence (as suggested in ICRP 50) of lung cancer per WLM (multiplicative risk model). When applied to the adult male population of North America, these risk coefficients suggest an average lifetime risk of about 3 10^{-4} per WLM for uranium miners age 20 to 55 at the time of exposure [M40]. Recent data from those studies in which most attention was given to reassess exposure data are compatible with the range of 1.5-4.5 10^{-4} per WLM for adult male miners, as was estimated in ICRP 32.

379. As mentioned briefly in chapter I, in many areas of the world houses have been built with or on materials which contain ^{226}Ra from which radon gas is released into the air of the living space. Exposure to the alpha emissions from the radon daughters is a potential risk factor for lung cancer, as demonstrated in miners, but the risks from exposures in homes have only recently begun to be estimated. Extensive indoor survey results are becoming available, but there are, as yet, few studies of the lung cancer risks associated with living in such environments for long periods.

380. In an initial study from Sweden, Svensson et al. reported on a case-control study of the association between lung cancer and radon in houses in the area around Stockholm [S52]. Study subjects had lived in the area for 30 years or more. There was a statistically significant relative risk of 2.2 (95% CI: 1.2-4.0), and 4.1% of cases in this group appeared to be attributable to the exposure. There was an indication of a dose-response pattern, with increasing cumulative exposure, as seemed similar to results from miners in the United States and in Czechoslovakia.

381. Other data have not shown an effect of domestic radon daughter exposure on lung cancer. A recent study by Gjorup and Hansen [G20] compared Denmark to Sweden, which has 2.1 times the radon exposure levels

in homes. There was no evidence for an excess of lung cancer in Sweden. Potential differences in other risk factors, such as smoking, were not studied in this report.

382. The ICRP issued in 1987 a summary of the risks associated with exposures to radon [I11]. This study covered the existing literature in detail up to about 1986, and considers many aspects of exposure of the lung to high-LET radiation. Its conclusions and lifetime risk projections are given in chapter VII.

383. The BEIR IV Committee [C20] has recently issued an appraisal of the effects of radon exposure. This report was received too late for review by UNSCEAR, and only a brief Secretariat review is considered here. The BEIR IV Report reviews all of the high-LET data available to it through 1987, for all types of exposure, and provides extensive dose-response modelling and statistical fitting, as well as lifetime risk projections.

384. After reviewing the literature on radon, the BEIR IV Committee considered the best way to obtain a single numerical estimate of the risk from radon exposure is with the following equation:

$$r(a) = r_0(a)\,[1 + 0.025g(a)(W_1 + 0.5W_2)]$$

where r(a) is the lung cancer mortality rate at age a; $r_0(a)$ is the baseline lung cancer mortality in the United States 1980-1984 population; g(a) is a coefficient equal to 1.2 for ages less than 55 years, 1.0 for ages 55-64 years, and 0.4 for ages 65 years and over; W_1 is the cumulative radiation exposure in WLM from five to 15 years before age a; and W_2 is the cumulative exposure 15 years or more before age a. This is a relative risk model which accounts for age at risk.

385. The BEIR IV Committee [C20] considered only occupational data. On the assumption that the occupational results can be applied to radon exposures in houses, BEIR IV estimated that 1 WLM per year would increase the number of lung cancer deaths in both sexes by a factor of 1.5 with current patterns of cigarette smoking. Occupational exposures to 4 WLM per year from ages 20-40 were estimated to increase the male lung cancer deaths by a factor of 1.6, most of the cases being in smokers. Note, however, that the exposure estimates for two of the studies used for the calculations done by the BEIR IV Committee, notably the Beaverlodge data [H25] and Swedish iron miners [R5], have been questioned by Frost, and Swent and Chambers (see [C20]). It has also been argued that a large part of the difference in risk estimates for the general population is due to differences in the assumed lung cancer rates in the reference populations rather than to differences in the risk coefficients in BEIR IV [C20] and ICRP 50 [I11]. The BEIR IV Committee modelled the smoking data as interacting multiplicatively with radiation, but acknowledged that a sub-multiplicative (but not additive or sub-additive) model was consistent with the existing data.

(iv) Thyroid

386. The best data on thyroid cancer are from children irradiated for a variety of conditions; these have already been reviewed (paragraphs 206-216). Adults have been treated with radioactive iodine for hyperthyroidism, without showing any documented excess of true thyroid cancer [C4, H12, H14]. In adults as in children, the anaplastic, and highly dangerous, form of thyroid cancer apparently has not occurred following irradiation.

387. A recent report has examined thyroid cancer in adults as well as children exposed to fallout from the Nevada, United States, atomic test site [Z3]. No excess was observed, and it is apparent that very large samples would be required to detect such an excess. The doses received by the Nevada population are in the range 0-1.5 Gy, usually below 0.4 Gy in adults. Based on these and other data, including the risk to the thyroid from external x rays, the authors estimated the absolute excess risk to be between one and four cases per 10^4 PYGy. The BEIR estimate was four carcinomas per 10^4 PYGy, including some occult carcinomas [C4]. There is insufficient information on which to base estimates of the effect of age at exposure.

388. Two reports from Sweden have examined thyroid cancer in adults and to a smaller extent in children following the administration of diagnostic amounts of ^{131}I which delivered doses to the thyroid gland of 0.5-1.5 Gy at dose rates of 2-6 mGy per hour [H27, H28]. In the first and preliminary study [H27] the incidence of thyroid malignancies in about 10,000 patients receiving typical administrations of 2 MBq was compared with the expected number of malignancies computed from the age- and sex-specific incidence in the Swedish Cancer Registry. Eight cases were found in the patients after a follow-up of 17 years compared with 8.3 expected. The authors estimated that an excess of at least 16 would be expected based on risk estimates for adults in the Japanese atomic bomb survivor population (external acute low-LET irradiation). This study was analysed further in a report of the United States National Council on Radiation Protection and Measurements [N5] which concluded that a risk reduction factor of at least 3 was applicable to iodine-131 irradiation compared with high dose rate external irradiation. Another review of this study is contained in the UNSCEAR 1986 Report [U1] which points out several factors which might account for the failure to observe the predicted excess.

389. The above study has recently been expanded [H28] to 35,000 patients receiving diagnostic ^{131}I administrations with a mean absorbed dose of 0.5 Gy, followed for an average of 20 years. Again the incidence of thyroid malignancies was compared with the expectation based on Swedish Cancer Registry data. Record linkage identified 50 thyroid cancers occurring 5 or more years after the initial ^{131}I administration compared to 39.4 expected based on general population rates. Patients who were examined for a suspected thyroid tumour received the highest doses and were at the highest risk. Patients given ^{131}I for other reasons were not at increased risk and neither were those who were observed for 10 years or more. An expected excess of 41 thyroid cancer cases was computed from the age- and sex-specific risk

coefficients estimated by the United States National Institutes of Health, Committee on Radioepidemiologic Tables for external high dose rate irradiation by x or gamma rays [U4]. The authors concluded that the thyroid cancer risk from irradiation of the thyroid gland by ^{131}I might be up to four times lower than with acute external low-LET radiation.

390. An excess of thyroid cancer has occurred in Japan [C4]. The approximate estimate for both cities, based on T65 doses, is 0.92 male and 2.40 female cases per 10^4 PYGy [C4]. Relative risks have been about 4, with the excess appearing 15 years after the bombing and persisting thereafter: whether risk has begun to decline is not certain. Generally, the adult pattern is similar to the pattern in children, with latency and subsequent risk behaving as they do for other adult epithelial tumours. Despite the difference in the absolute risk of spontaneous thyroid cancer between males and females, the 3:1 female to male case ratio is about the same as that in the unexposed population.

391. A recent summary of thyroid cancer risks issued by the National Council of Radiation Protection and Measurements [N5] expressed risk as follows:

$$\text{Risk} = R \times F \times S \times A \times Y \times L$$

where R is the absolute risk per 10^4 PYGy for both sexes in ethnically similar populations of children exposed to external x-irradiation after a minimum induction period of five years. For the United States population, based on estimates derived from externally irradiated children, the report [N5] takes this value to be 2.5. F is a dose-effectiveness factor equal to 1.0 for external x- or gamma-irradiation and for ^{132}I, ^{133}I and ^{135}I and equal to 1/3 for ^{131}I and ^{125}I. S is a sex-correction factor equal to 4/3 for females and 2/3 for males, assuming that females are twice as susceptible as males and that the value R is based on populations comprising equal numbers of males and females. A is an age-susceptibility correction factor equal to 1 for exposure at ages under 18 and 1/2 for exposure at older ages. (If sex-specific R values are used, then S = 1.0). Y is the average number of years of post-exposure risk in the group being evaluated. L is lethality, equal to 0.10, assuming that only 1 case in 10 is lethal (this factor is to be used only when estimating the lifetime deaths due to radiogenic thyroid cancer).

392. The risk can be calculated for any study group using this formula. As an example, Table 39 provides risk estimates for an exposed United States population [N5]. The report of the National Council on Radiation Protection and Measurements compared absolute and relative risk models and found little difference in lifetime estimates. This model was also tested against the Marshallese data, from which direct estimates of risk are not reliable, and it gave an adequate fit [N5].

(v) Other epithelial tissues

393. The literature on lung, breast, and thyroid cancers has been reviewed separately because, of all epithelial cancers, these are the ones for which the best data are available. There are, however, many other epithelial tissues in the body. Data on cancer in these tissues come mainly from three groups of individuals; namely, the atomic bomb survivors, the ankylosing spondylitis patients and women irradiated for malignant and benign gynaecologic disorders.

394. For many years it had been thought that some organs were relatively insusceptible to radiation carcinogenesis. This notion stemmed from the lack of evidence for a statistically significant excess risk or to the low background risk of the malignancy itself. It now appears that most (indeed, probably all) organs are vulnerable to radiation-induced cancer, given the right conditions of exposure. In Japan, data still do not support an excess risk or dose-response for cancers of the buccal cavity, rectum, pancreas, small intestine, uterus or malignant lymphoma [P15]. These sites may achieve significance as the exposed cohort passes through the years of greatest background risk, since in the last decade several sites not previously thought to be affected have shown a dose-response relationship. In patients irradiated for benign gynaecologic disorders, tumours of the buccal cavity, as well as of the kidney and urinary bladder, have relative risks of about 2, which are comparable to the relative risk in high-exposure Japanese (> 1 Gy) [W5] and in spondylitics (average exposure 2 Gy).

395. In their comparison of the data from Japan and the spondylitis patients, Darby, Nakashima and Kato have suggested that there may no longer be any truly radio-insusceptible epithelial tissues. This opinion is set forth in [D11] and [D20]; the latter contains the data on which the computations were based. Their conclusion was arrived at only when the data from the two groups were combined and analysed jointly, increasing the sample sizes sufficiently to show statistically significant excesses. A summary of the risks based on this joint analysis is given in Table 40. For example, gallbladder cancer was significantly more frequent than expected in the combined series than in either series alone. Darby et al. also described an excess of central nervous system tumours in their combined analysis, but see [P15]. This joint analysis will be referred to in the following paragraphs. However, it should be noted that these estimates, while they are the only joint estimates currently available and the only estimates based on a sample size large enough to detect significance for some sites, are based on obsolete doses and a shorter follow-up than is now available. The estimates have been revised recently, and while no new joint analysis is available, the revisions will not reduce the qualitative evidence for excess risk at the sites reported by Darby et al.

396. In addition to the cervical cancer patients, several other cohorts totalling about 14,000 women exposed to pelvic irradiation for a variety of benign gynaecologic conditions have been followed [B6, C4, S3, W6]. While these women add information on epithelial sites, they also pose further questions and uncertainties. Relative risk data for them were presented in Table 25. Both radium and x-ray treatments were involved [B12, D9], and the exposures were external, low-LET (x ray) and internal, high-LET [W6]. Doses ordinarily ranged from 20 to 70 Gy, given in fractions of a few Gy over periods of 4-8 weeks [B12].

397. In women treated for benign disorders, uterine sarcomas were increased about eightfold and female genital and urinary organ tumours about twofold. Exposure to radiation may lead to relatively advanced, aggressive uterine tumours when the original reason for pelvic irradiation is to treat a malignant, rather than a benign, condition [M35]. An elevated risk of uterine sarcomas was seen in one ovarian cancer series [R11] but not in another [C8] nor in the cervical cancer series [B12].

398. The joint analysis of the Japanese data and the spondylitis data [D11, D20] (see Table 40), serves to summarize the available literature on a variety of exposed sites. A multiplicative projection model describes the combined data reasonably well. Age-specific relative risk is roughly constant as a function of age once the latency time is over. For heavily irradiated sites, both sets of data show a positive correlation between the excess risk and the baseline prevalence of the tumour (Figure XIII). This correlation suggests that radiation magnifies processes already at work multiplicatively.

399. In their analysis of the Life Span Study data for the years 1950-1978, Kato and Schull [K7] concluded that the mortality experience of this cohort supported a relative risk model more strongly than the additive one. This assessment has been further supported by the more formal adoption of the relative risk model in the Life Span Study Reports 10 and 11 [P15, S49]. Muirhead and Darby reached similar conclusions [M36, M37]. The excess deaths from all cancers other than leukaemia and bone cancer increase with age at death for the same age cohort in proportion to the age-specific death rate from cancers in the population of all Japan and do not show a constant excess value by age at death for the same age cohort, as predicted by the absolute risk model.

400. Darby et al. also examined the post-exposure risk for a pooled series of selected epithelial sites for which data are available from both the spondylitis series and the > 1 Gy group in Japan [D11, D20]. These sites, which the authors referred to as "selected sites", include the pharynx, oesophagus, stomach, pancreas, larynx, lung, ovaries, skin, and bones. They

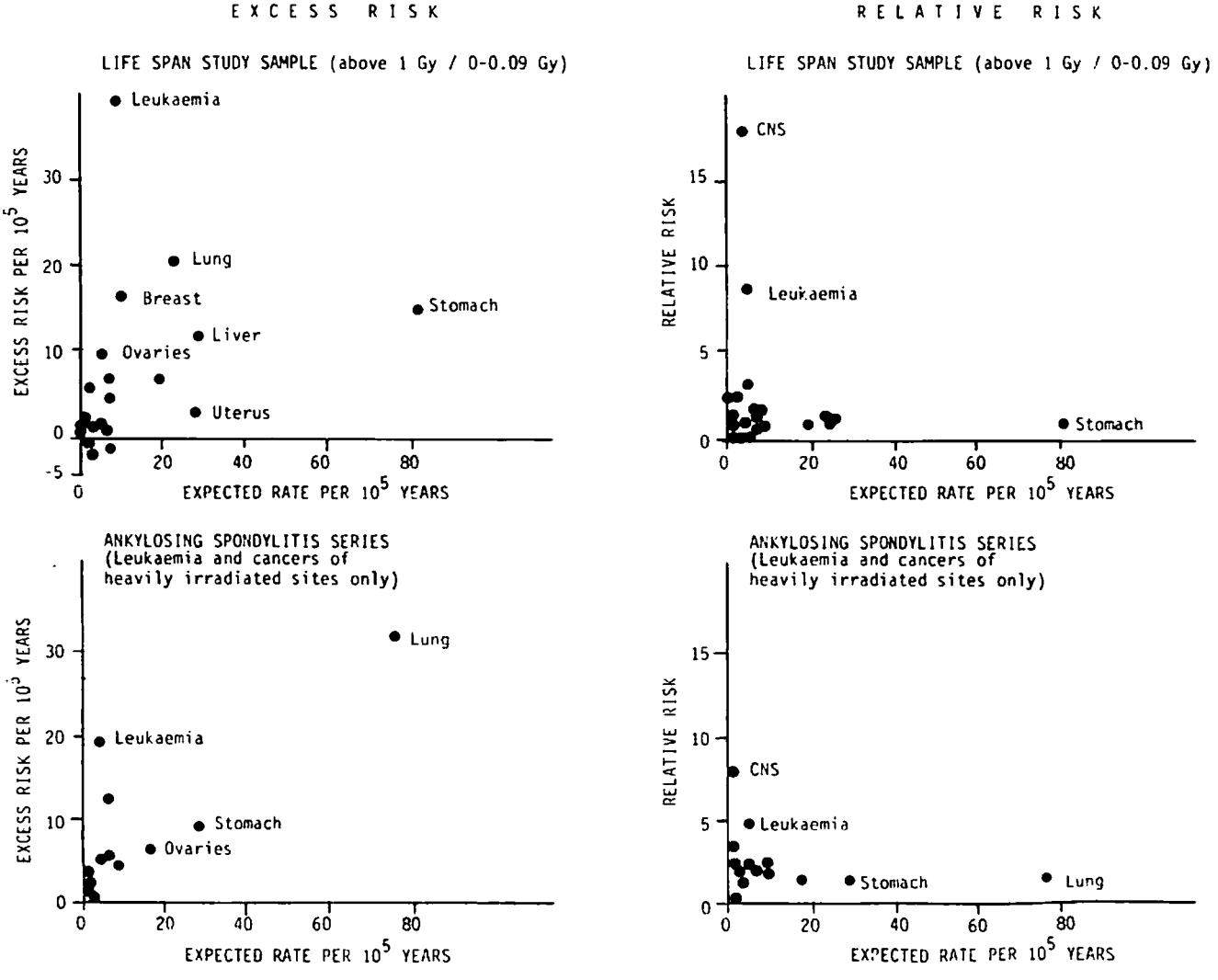

Figure XIII. Excess and relative risks of cancers in the Life Span Study sample (T65DR doses) and in the ankylosing spondylitis series for various cancer sites compared to expected population incidence rates.
[D11]

also found that the relative risk model describes the data on the pooled sites.

401. However, the latest report from the ankylosing spondylitis series [D21] differs somewhat from the other data in regard to epithelial cancers other than colon cancer. The authors have found that 25 years after irradiation, the RR values return approximately to normal, contrary to the essentially permanent effect seen in other studies. Age at irradiation did not significantly affect the subsequent relative risk for these tumour sites (no patients where such an effect has been seen were under age 15).

402. The relative risk is higher in females than males in Japan for many epithelial sites (it is lower for leukaemia). This is shown in Table 41, taken from Life Span Study 10 (T65). For oesophageal and lung cancer, the difference is probably due to different smoking habits.

403. Because differences have appeared between the Japanese and spondylitis study, and the doses have been revised, one must interpret the parallel analysis of Darby et al. with caution. However, the new data seem unlikely to change the support for the relative as opposed to the absolute risk model for excess solid cancer risk, even if the relative risk is found to be a function of sex, age at exposure, and time since exposure, as suggested by Muirhead and Darby [M36, M37]. Similarly, Darby et al. used pooled data to demonstrate excess cancer risk at many sites for which an excess could not be documented in either study alone. This is probably a reliable indicator that those sites are susceptible to cancer from exposures to ionizing radiation.

404. In analysing the available data on these epithelial tumour sites, especially those of the digestive system, a variety of observations are worth summarizing.

405. *Digestive system.* Little data exist on salivary gland tumours from Japan and the spondylitis series, partly because of the low exposure levels. However, from the essentially consistent results of eight studies of medical therapeutic exposures, Land [L11] estimates that the absolute risk of salivary gland tumours is 0.26 ± 0.06 cases per 10^4 PYGy after the first five years post-exposure, with little evidence of an association between response and age at exposure. The data are summarized in Table 42. Most of these exposures are in children, including two tinea capitis series [M13, S16], and head and neck exposures in five studies [H1, J4, M11, S15, S40], or in middle-aged women treated with radioactive iodine [H21]. In the Japanese data, dose estimation is complex, but the risk is estimated as 0.056 ± 0.036 per 10^4 PYGy for malignant salivary gland tumours and 0.063 ± 0.035 per 10^4 PYGy for benign ones [O4, T15]. Recent data have established the existence of a dose response for oesophageal cancer and for stomach cancer, but it is still difficult to obtain accurate estimates of the lower bounds of the effects [O3].

406. No single data set supports an excess for gallbladder cancer; the main risk factor for this very rare tumour is gallstones, which are relatively rare (but becoming more common) in Japan and more common in women. Most of the spondylitics were men; the gallbladder was given little dose in the cervical cancer patients. There is little statistical power in the available data, although the recent report [P15] from Japan estimated a small effect (relative risk at 1 Gy about 1.15).

407. The pancreas seems to be of uncertain susceptibility [L11]. Risk cannot be assessed from the available data, and expected rates are complicated by problems in late and sometimes difficult histologic diagnosis. In many countries pancreatic cancer is a common cancer, and one might therefore expect the evidence to be more clear-cut; this is not the case at present.

408. Cancer of the small intestine is generally rare, and it is still difficult to know if there is a radiogenic effect. The data come mainly from some cervical cancer patients, but, as noted earlier, both irradiated and non-irradiated subjects had similar excesses. Colon cancer has already been discussed in the context of the cervical cancer patients, where inconsistent results were obtained. In Japan, mortality data show an increase in the Life Span Study sample using the T65 doses [K7, P15] and the new DS86 doses [S48]. Only a non-significant increase was reported in the spondylitics; however, a possible association between spondylitis and ulcerative colitis casts doubt on that result [D11]. Rectal cancer seems to be a consequence of exposure to ionizing radiation, but a dose-response pattern is not estimable and the dose may need to be more than 1 Gy to produce a detectable effect.

409. *Genito-urinary system.* The mortality data from Japan still do not support a dose effect for uterine or uterine cervix cancer, and the evidence comes almost exclusively from those women irradiated for gynaecologic disorders. The only evidence of the inducibility of prostate cancer comes from the Nagasaki Tumour Registry; considering all cases, including those discovered only at autopsy, the absolute risk is 2.1 cases per 10^4 PYGy based on a linear model [L11, W5]. This has not been confirmed, at least as yet, in the mortality data [S48]. Prostate cancer is a disease of advancing age, and most cases are not discovered clinically and would not be reflected in mortality data. Land speculates that a small radiogenic risk would be even less detectable in the much higher background prostate cancer rate in Europeans and North Americans [L11].

410. A recent study in Japan [T21] has shown a statistically significant dose-response pattern for both malignant and benign neoplasms of the ovary, with a latency period of at least 15-20 years.

(vi) Liver

411. Somewhat more detail is available for liver cancer after radiation exposure. The liver had been regarded as being relatively radio-insusceptible. However, the Japanese data have now revealed a slight increase in liver cancer, when "not otherwise specified" cases are included [P15, S49]. It bears mentioning that the liver is a common site of metastasis for other radiation-induced cancers, e.g., those of the lung, stomach and breast, and that death certificates will

commonly fail to distinguish between a primary and a secondary malignancy, particularly in the absence of supportive pathological information. The increasing use of radioisotopes for diagnostic liver scans or other radiotherapeutic purposes makes more data available and also underscores the importance of a better knowledge of the liver's susceptibility. The best data come from the Thorotrast patients (indeed, Thorotrast use was stopped in about 1955, when its liver carcinogenicity was discovered [M26]). Most cancers caused by this agent are bile duct carcinomas, hepatocellular carcinomas or angiosarcomas [C4].

412. Thorotrast data are reviewed in [C4], and details specifically from the Federal Republic of Germany series are in [K16]. The average dose to the liver from the 25 ml of alpha-emitting substance injected was about 0.25 Gy per year; about 65% of the amount injected was deposited in the liver. From these exposures, the risk estimate was about 300 liver cancers per 10^4 PGy [C4], projecting cumulative risk to the lifetime of the exposed cohort of individuals. For an average of 23 years at risk beyond the first 10 years in this group, the estimated risk rate coefficient was 13 liver cancers per 10^4 PYGy. Complicating this assessment were the conceivable effects of Thorotrast toxicity, on the one hand, and radiation-produced cell sterilization, on the other. Tumours began to appear about 10 years after initial exposure, and the period of elevated risk may have extended beyond 40 years [K16].

413. The cumulative incidence of liver tumours in the Federal Republic of Germany series is presented in Figure XIV, for different liver dose rates measured by x-ray film and whole-body counter assessment. Because deposited radioisotopes can be visualized and quantified on x-ray film, the dose-response pattern has been estimated for liver cancer. Risk as a function of time since exposure rises more steeply in the more heavily exposed [K16].

414. Data are also available from Japanese military patients treated with Thorotrast to diagnose war injuries [M29, M31]. In these patients the risk for hepatic cancer was 40.0 relative to a military control group and 22.2 relative to population-based controls; the relative risk in both cases was 1.3 (not significant) for other tumours, which included a variety of sites. After 35-43 years, there have been 50 hepatic tumours in 254 subjects, a cumulative incidence of 19.2%. Based on autopsies from these individuals, the mean dose rate for the individuals with hepatic cancer was estimated to have been 0.29 Gy per year, low-LET equivalent, with a mean total dose of about 9.20 Gy [K18] of this high-LET exposure, after a mean 36.1-year latency period.

415. Other individuals have been exposed to alpha-emitters deposited in the liver, particularly ^{239}Pu, in the case of nuclear workers. The available data do not show an effect but are compatible with an effect no greater than 10 times that of Thorotrast [C4].

5. Occupationally exposed adults

416. As was noted earlier, studies of the effects of ionizing radiation on adults exposed in the course of their employment or military service have focused largely on radium dial painters and radiologists in the United States and the United Kingdom or on individuals engaged in nuclear weapons research and fabrication, in the activities of nuclear power stations, in the maintenance and outfitting of nuclear-powered naval vessels, primarily submarines, or in nuclear weapons tests. The findings on the radium dial painters and radiologists have been described elsewhere in this document; this section summarizes the findings of one large case-control study of radiological technicians [J5] and of the other studies of occupational, including military-service-related, exposure.

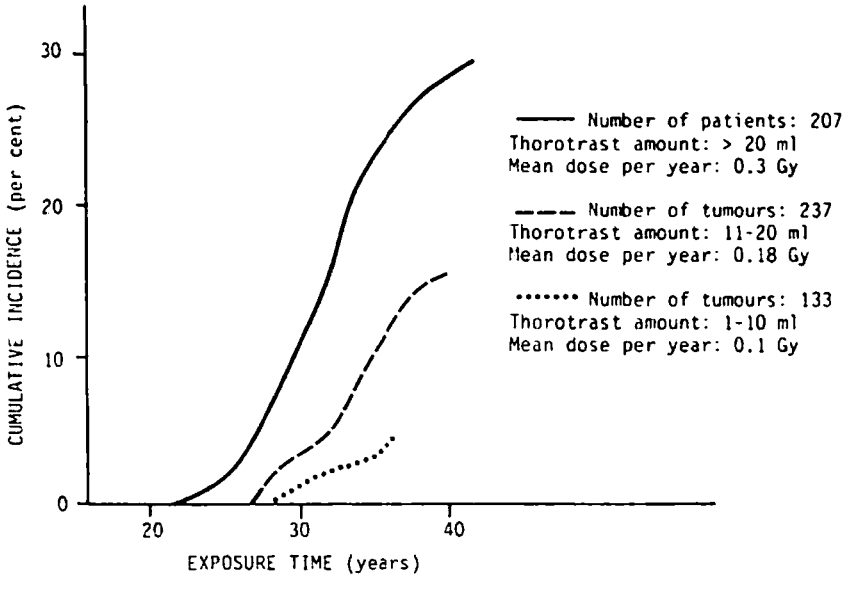

Figure XIV. Cumulative incidence of liver tumours in Thorotrast patients. [K16]

417. Jablon and Miller [J5], in a study of 6,560 radiology technicians in the United States Army in the Second World War, found no statistically significant differences between them and a control group (6,826 medical, laboratory and pharmacy technologists) with respect to the frequency of individual sites of cancer or deaths from other causes. More specifically, for 174,500 PY of risk, they observed 12 leukaemia deaths (including one case of chronic lymphocytic leukaemia) among the radiological technicians and 7 among the controls ($P = 0.12$, one-tailed test). While the doses are uncertain, their exposures may have been 0.05-0.15 Gy per year, based on the experience of similar technicians at the Cleveland Clinic (United States) in 1953. Most of these radiology technicians did not pursue the same kind of work after they had left the Army, where their average stay had been less than 3 years.

418. Efforts have been made to determine whether individuals employed in the nuclear industry do or do not have increased risks of cancer. In 1978, for example, Najarian and Colton [N6], in a study of 1,722 death certificates for a variety of workers at the Portsmouth Naval Shipyard (New Hampshire, United States), found six deaths from leukaemia among the 146 former workers presumed to have been involved in activities where exposure could have occurred, whereas 1.1 deaths were expected. A subsequent retrospective cohort mortality study [R17] of all the workers at this shipyard failed to confirm the finding. Among three cohorts, (a) 7,615 nuclear workers (doses 0.01-0.91 Sv; mean 0.03); (b) 15,585 non-radiation employees; and (c) 1,345 with no measurable exposure, on whom vital status could be ascertained in 96% of cases, Rinsky et al. found no increased mortality for the exposed groups as contrasted with the other two groups, nor did they find evidence of a dose-response relationship within the exposed cohort. The standardized mortality rate (SMR) for leukaemia was 84 (95% CI: 34-174). As is true in many occupational settings, some uncertainty surrounds the actual doses involved: for years prior to 1974, the estimates are based on film badges, but for subsequent years, they are based on calcium fluoride dosimeters. A study of the employees of all of the nuclear shipyards in the United States, government and private, is presently under way. While it has not yet reported its findings, the study may eventually clarify the issue. Similarly, in 1981, Austin and his colleagues [A16] reported a threefold increase in the frequency of malignant melanoma among the employees of Lawrence Livermore National Laboratory (United States). Again, a substantially larger, later cohort study of the workers at Los Alamos National Laboratory (United States) [A15] failed to support this. Among 11,308 employees, only six cases of melanoma were ascertained where 5.69 would have been expected based on age and sex-specific mortality rates (SMR = 105). In neither of these studies was evidence presented that the cases had received higher exposures than other employees.

419. The situation with respect to the employees at the Hanford Facility in the state of Washington, United States, is equally perplexing. Kneale, Mancuso and Stewart [K20] purported to show that a variety of malignancies, including multiple myeloma, are elevated among the workers at this laboratory, but a more thorough and statistically sounder study [G12] does not bear out their contentions, although it does find a greater frequency of multiple myeloma than expected. It should be noted that even this result rests on three cases. Whether this increase is, indeed, a consequence of exposure is therefore moot, but multiple myeloma has been found to be elevated among the atomic bomb survivors, presumably as a result of their exposure, and the effect could be real. More recently, Beral and her colleagues [B22], using standardized mortality rates, examined the causes of death among 39,456 individuals employed by the United Kingdom Atomic Energy Authority (UKAEA) between 1 January 1946 and 31 December 1979. They found mortality to be increased for only four causes of death, namely, testicular cancer (SMR 153; 10 deaths), leukaemia (SMR 123; 35 deaths), thyroid cancer (SMR 122; three deaths), and non-Hodgkin's lymphoma (SMR 107; 20 deaths), but in no instance was this increase statistically significant at the 5% level. The SMR for myeloma was 83 (95% CI: 36-163). Cumulative dose estimates are available for approximately half of these employees; few (84) had received a cumulative dose in excess of 0.5 Sv. Among the workers for whom there were dose estimates, prostatic cancer was the only cause of death clearly related to exposure (SMR 594 for employees with exposures exceeding 10 mSv; four deaths). Although the numbers are small and the evidence is perforce weak, the data suggest a greater risk among workers exposed to tritium than among workers exposed to other sources of ionizing radiation (SMR 889; 6 deaths). Beral et al. [B22] estimate excess mortality for leukaemia and all cancers were 2.2 and 10.5 deaths per 10^4 PYSv, respectively. Neither of these estimates is significantly different from zero, but at face value they both agree reasonably well with the Japanese and other studies. It is interesting to note that when the UKAEA findings and the Hanford findings are combined, neither the increase in prostatic cancer seen among the former nor the increase in multiple myeloma seen among the latter is any longer significant [D24]. This suggests that both individual findings could be due to chance.

420. Possibly the most thoroughly studied of these special cohorts has been the plutonium workers, particularly those individuals who were involved in working with this element at the time of the Manhattan Project, when the potential hazard associated with the inhalation of plutonium particles was poorly recognized. Some 37 years of follow-up have failed to disclose an increased frequency of any malignancy; the number of workers involved is small, but their exposures were undoubtedly large [V2]. Studies of plutonium workers at the Los Alamos facility [V3] as well as of workers at other installations in the United States [W18] have also failed to find a significantly elevated risk of malignancy, generally or site-specifically. Although the number of years at risk are already large, these studies continue, and it is conceivable that an effect could still emerge.

421. In 1979, a preliminary report [C16] indicated that eight cases of leukaemia had been identified

among 3,224 former servicemen who had participated in the nuclear weapons test code-named SMOKY, one of a series known as PLUMBBOB, conducted at the Nevada Test Site, United States, in 1957. Only 3.5 cases would have been expected on the basis of age- and sex-specific population rates (RR = 2.3). Subsequent studies of this same cohort [C17] extended the observations to the incidence of all types of cancer and other specified causes of death. No increase in other cancers was seen, but 10 cases of leukaemia (including one of chronic lymphocytic leukaemia) were found where 4.0 were expected (RR = 2.5). Similar claims, based largely on scanty epidemiological evidence, have since been made for Australian and British participants in weapons tests carried out by the United Kingdom [K21].

422. Stimulated by these reports, the Medical Follow-up Agency of the United States National Research Council launched an investigation of the participants in five series of tests occurring in the years 1951 through 1957 [R16]. This investigation embraced a cohort of 46,186 individuals. A total of 46 deaths from leukaemia were ascertained (52.4 expected on population rates). No significant excess was found among the participants at any test series other than PLUMBBOB or among PLUMBBOB participants not represented at SMOKY. The earlier findings of Caldwell and his colleagues with respect to the SMOKY test were confirmed. No other form of cancer was consistently elevated; overall, only 1,046 deaths from malignant neoplasms were identified where 1,243 were expected based on population rates (SMR = 0.84). While the doses of the individuals involved in all of these tests are poorly known, film badges suggest that the highest dose received by any one of the participants in SMOKY who subsequently succumbed to leukaemia was 0.036 Sv (most received doses of less than 0.005 Sv). At the present, then, there is no consistent or statistically significant evidence for an increase in either leukaemia or other malignant neoplasms in nuclear test participants.

423. Darby et al. [D26], updating the study of Knox et al. [K21], have summarized the cancer mortality and incidence among 22,347 men who participated in the United Kingdom's atmospheric nuclear weapon tests and experimental programme, and have compared these findings with those on 22,326 individuals matched with the participants for age, type of armed service, rank (officers and other ranks; socioeconomic class for civilians), and the date of entry to the study. The latter individuals were drawn either from among servicemen who did not participate in the weapon test programme, or, for the civilians, from the roster of employees of the Atomic Weapons Research Establishment who had not visited a test location or attended tests in the United States. Thirty-eight causes of death were examined separately.

424. Mortality from leukaemia and multiple myeloma in the participants was slightly greater than would have been expected from national values, but it was substantially lower in the controls. However, the rates of leukaemia and multiple myeloma showed very little difference between groups characterized by recorded doses from external radiation. These authors cautiously concluded "Participation in the test programme did not seem, in itself, to have caused any detectable effect on the participants' expectation of life, apart from possibly causing small risks of developing leukaemia and multiple myeloma".

425. Rinsky et al. [R21] have described the results of a case-control study of lung cancer in civilian employees at the Portsmouth Naval Shipyard (United States). Their study involved 405 cases and 1,215 controls drawn from the roster of civilian employees matched on age, year first (last) employed, age at date first (last) employed, and length of employment. The distribution of cumulative radiation doses among the cases differed little from that among the controls save in the percent exposed to 0.01-0.05 Sv where the radiation-related excess was statistically significant. However, when exposures to asbestos and welding fumes were taken into account, the radiation-related risks at all levels of exposure were reduced, suggesting a greater exposure to these factors. This confounds the observed association between radiation and lung cancer. Analysis of mortality by time since exposure revealed no pattern of increase as latency increased. Data on cigarette smoking and socioeconomic status were not available. These authors conclude that their study does not preclude an association between lung cancer and exposure to ionizing radiation (at the levels obtaining among nuclear shipyard workers) nor does it provide evidence in support of such an association.

426. Checkoway et al. [C21] have described a historical cohort mortality study of 6,781 white male employees of the nuclear materials fabrication plant known as Y-12 at Oak Ridge, Tennessee, United States, in the years 1947-1979. Among monitored workers, the mean cumulative alpha-radiation dose to the lung was 0.082 Sv, and the mean cumulative external whole-body penetrating dose from gamma-radiation was 0.0096 Sv. Mortality excesses were seen for cancers of the lung, brain and central nervous system, but not for other sites of cancer nor other causes of death when the rates among workers were compared either to national or state rates. No dose-response trend was observed for mortality from cancer of the brain and of the central nervous system, but the rate ratio for lung cancer, based on contrasting workers receiving 0.05 Sv or more with workers receiving less than 0.01 Sv, was 4.60 assuming zero-year latency and 3.05 on a 10-year latency. These rate ratios are, however, based on only three deaths and one death, respectively. Thus, the evidence of an increase in lung cancer mortality at these dose levels is far from compelling.

427. Workers for British Nuclear Fuels at the Sellafield plant have been studied [S54]. Among 14,327 known to have been employed at the plant from 1947-1975, 572 of 2,277 deaths were due to cancer, 5% less than expected based on death rates for England and Wales (overall mortality was also slightly less than expected). Radiation workers had deficits of liver and gallbladder cancers, lung cancer, and Hodgkin's disease, and excess deaths from myeloma and prostate cancer. Neither excess was significant, and there was no excess in leukaemias. Dosimetry showed positive

associations between accumulated dose and death rates from bladder cancer, multiple myeloma, and haematopoietic neoplasms. These were significant in regard to doses accumulated up to 15 years prior to the time of death, but not if doses up to two years before death were included.

6. Exposures to elevated cosmic and terrestrial radiation

428. Although levels of exposure to cosmic radiation vary as a function of altitude, and although some correspondence exists between cancer rates and altitude, there have been few convincing studies to show whether cancer rates at high elevations are substantially different from those elsewhere [C4]. Many correlated factors could explain the data that are available (see [A5] for a review). Studies designed to assess firmly whether cosmic radiation itself causes cancer would require prohibitively large samples.

429. A large-scale investigation of background radiation has been undertaken in China [H24, Z4], where cancer mortality levels in a high-background area in Yangjiang country were compared to those in a control area with one third the exposure levels. After age adjustment, there were no significant differences, even though chromosomal and other indications of radiation exposure did differ.

430. A separate study has compared the effects of radon alpha-exposure in high-background areas of Guangdong Province of China [H24] with a control area. High-background area exposures were about 0.38 WLM per year, and control-area exposures were 0.17 WLM per year. No difference in age-adjusted lung cancer rates was found.

431. A study of total background radiation in Japan [U5] found an effect only for male liver cancer. This effect fitted a linear dose-response model, but it is difficult to determine if there were other factors correlated with background exposure or if the result is a statistical artefact of some kind, as one due to multiple testing. Liver cancer is not usually reported as a radiogenic site, unless doses are also high enough to induce excess cancer at most other sites as well. In Connecticut, United States, where there is a tumour registry and an airborne gamma survey of the entire state was taken, there was no association between terrestrial radiation and cancer in the period 1935-1974 [W16]. The authors concluded that even a population currently in excess of 3 million persons is too small to detect the level of excess risk which might be associated with the observed level of background radiation.

7. Summary of exposure effects in adults

432. In respect to the radiation exposure of adult human subjects (see also [B21]), several generalizations seem permissible. Regardless of the reason for the initial exposure, it is evident that (a) a single exposure can be carcinogenic if the dose is large enough; (b) there is no uniquely radiogenic cancer cell type; (c) though most, perhaps all, of the common cancers probably can be caused by ionizing radiation, until now the data have not shown a risk for chronic lymphocytic leukaemia, squamous-cell cervical cancer, or Hodgkin's disease; (d) the breast, thyroid, and bone marrow are particularly susceptible; (e) leukaemia, especially acute non-lymphocytic leukaemia (ANL), can be produced by radiation; it has a latency period of less than 5 years, peaks rapidly thereafter and then declines, but some excess risk persists for at least 30-40 years; (f) solid tumours have an age-onset pattern similar to that of non-radiogenic tumours at the same sites after a latency of about 10 years. For many sites, the latency period is a function of age at exposure. In the existing studies, relative risks have been between 1 and 3 for many epithelial sites after many different kinds of exposures of about 1 Gy; risk persists for 30 years (for life, in some studies); (g) age at exposure is the most general host susceptibility factor, with higher risk associated with younger ages at exposure; (h) atomic bomb survivors and most other study cohorts have yielded comparable results, with a few notable exceptions, and the latter appear explicable in terms of the exposure regimens used and other factors; (i) some individuals may be genetically more susceptible to radiation-induced cancer than others, but good data to demonstrate this unambiguously are very limited.

IV. HOST FACTORS THAT MODIFY RISK

433. There are many biological differences among human beings that may affect their susceptibility to radiation-induced cancer. These biological variables are commonly known as host factors, referring to the risk that the exposed individual will become a host to a tumour. There are many possible host factors for which some data exist. Among these are (a) sex; (b) age at exposure; (c) genetic constitution; (d) health status; (e) life-style; and (f) ethnicity. Since the publication of the UNSCEAR 1977 Report, some information has become available on the potential role of each of the factors listed.

A. SEX

434. Current data generally suggest that sex has little or no effect on radiation carcinogenesis. Tumours in an irradiated population exhibit a sex ratio very similar to the same tumours in a non-irradiated population. There is a strong preference for females in thyroid cancers produced by radiation, but the sex ratio is similar to that observed for spontaneous thyroid cancer. So far, breast cancer following radiation has essentially been found only in females and this corresponds with the extreme rarity of male breast cancer. Cancers in the organs usually manifesting adult onset occur in the typical sex ratios, which for many sites (for example, the lung) show a male preference and there is no evidence that the radiation-related relative risk is higher. The male excess of lung cancer is probably a temporary one related to the history of smoking habits. Squamous cell carcinomas and adenocarcinomas of the lung in Hiroshima and

Nagasaki, for example, seem to develop more rapidly in males than in females, but no difference appeared after smoking habits were taken into account [H10, K22]. Adenocarcinomas are more frequently slow-growing than are lung cancers of other histology. The evidence of a slightly higher relative risk for females than for males in Japan at many epithelial sites was reviewed earlier (see Table 41); most of the difference is probably due to interaction with other sex-associated risk factors rather than to a radiation effect.

435. Radiogenic leukaemia in Japan has a similar relative risk in both sexes, although the excess risk per 10^4 PYGy is significantly less in females (1.95 in males and 1.20 in females). Background incidence of leukaemia is two times more frequent in males. For other fatal cancers, shown in Table 41, only multiple myeloma has a higher background incidence in females; however, the excess risks are, from a statistical standpoint, not significantly different in the two sexes. Since the background rates are higher in males, the relative risks are somewhat greater in females [P15]. Sex may influence tumour growth and, indirectly, survivorship.

B. AGE OF ONSET OF TUMOURS IN EXPOSED ADULTS: SINGLE AND CHRONIC EXPOSURES

1. Exposure to the atomic bombings in Japan

436. The carcinogenic effects of single exposures of external radiation are known almost exclusively from data in Japan. There, the relationships between dose, age at exposure and age at expression of excess risk have been studied in detail [K7, P15, S18, S48, S49, W5]. Because the results have been reviewed extensively elsewhere in this document, only those facts that relate to tumour sensitivity as a function of age at exposure and age of onset will be examined here. For most tumours, there is a decreasing sensitivity with increasing age at exposure, in terms of subsequent risk of excess cancer. The solid tumours of adult onset increase in frequency only at the ages at which they naturally appear in non-irradiated individuals. For leukaemia, the additive risk rises with age at exposure, while the relative risk declines rapidly with age over 10 [K7]. Leukaemia, a classic radiogenic tumour with other special characteristics, is usually treated separately. The characteristic pattern for breast cancer has already been discussed in relation to childhood exposures (see paragraphs 240-242). Basically, all tumour risks seem to decline as age at exposure increases. The same reports also show the positive correlation between risk and dose at all ages. There is an unexplained difference between the data in Japan and uranium miners, the latter showing increased risk with increased age at exposure [S51].

437. In addition to the fact that susceptibility to radiogenic tumours decreases with increasing age at exposure, the characteristic latency periods are related not so much to age at exposure as to the tissue involved. The leukaemias have the most definite pattern, with a latency of 2-5 years and a decline in additive risk after about 25-30 years; a similar pattern exists for bone cancer. However, the solid tumours of adult onset have latency periods of a decade or more, and the excess risk persists indefinitely, probably throughout life, although this will be determinable only after the entire cohort experience in Japan is known.

438. The subsequent risk of cancer fits a multiplicative risk projection model. The delayed latency period, the persistence of risk throughout adult years, and the age pattern of excess cases are all consonant with multi-stage carcinogenesis, as for example, the model proposed by Moolgavkar [M1, M2], which posits two stages with selective proliferation of partially transformed cells.

439. Under such a model, increased risk should be seen soon after exposure to a single dose, if (as seems likely) radiation acts as an initiating agent (see section III.A.1). However, if this effect is small in the contrasted groups, the excess risk will not be detectable for some time after it has actually arisen, given the available sample sizes. It can be shown that, under such a multi-stage risk model with multiplicative projection effects, because of the nature of the change in the risk function and the effects of competing causes of death (which effectively terminate the observational experience at age 85 or so), the mean age and the age distribution of cases in exposed adults will be similar to those in the population at large [M4]. For these reasons, the age patterns of risk for those exposed to single doses are in agreement with a multi-stage model, and no special life-history or tissue sensitivity characteristics are required, other than in those cases, e.g., breast, bone and leukaemia, where tissue life-history plays an obvious role in sensitivity.

2. Exposures to nuclear tests and fallout

440. The estimated dose-response pattern and its possible significance for those who have been exposed to fallout from a single nuclear detonation (adults in the United States and British military service, Japanese fishermen and Marshall Islanders) are discussed elsewhere in this document. In sum, the numbers of exposed persons were too small and too heavily weighted towards young adults to provide good data on age effects. Adult Japanese fishermen, although few in number, and Marshall Islanders [C6] seem not to have exhibited an excess risk. Non-haematopoietic tumours do not appear to have arisen in these groups in detectably higher frequencies.

441. For adults exposed to chronic doses of radiation either occupationally or through the ingestion of radioisotopes for therapeutic purposes or as a result of nuclear testing, the duration of exposure is too long and, typically, the dose too low to provide useful information about special effects of age at exposure.

C. GENETIC CONSTITUTION

442. Given the genetic variability that exists between individuals within a group and between groups, it is

reasonable to presume that the risk of cancer may vary among individuals of the same sex, age and apparent life-styles when exposed to the same amount of ionizing radiation. A number of relatively rare, largely recessive disorders are known in which fibroblasts from trait-bearers are deficient in the repair of some radiation damage in vitro; it is also known that these individuals are at increased risk of a variety of malignancies, especially malignant lymphoma and leukaemia [K11]. Cell lines from patients with one such disorder, xeroderma pigmentosum, in which UV light is a mutagen, did not disclose a cross-sensitivity in regard to cell-killing with gamma radiation, but enhanced sensitivity to cell-killing has been reported in vivo in irradiated children. Some cell lines from patients with heritable diseases, including cancer-prone ones, have shown cell-killing sensitivity after such radiation [A7, G5, P10, P11], but this is not always found [W10]. One study [F7] has reported that cell cultures manifesting a variety of chromosomal aberrations have shown similar low-dose response estimates.

443. The carriers of one major disease, ataxia telangiectasia (AT), may have been subjected to irradiation in numbers sufficient, eventually, to show an excess of cancers, if it exists. The gene for AT is relatively common in Israel, where it is expected that at least some children irradiated for tinea capitis would be carriers of the gene. The latest report [R22] suggests that Moroccan children, who have a high frequency of AT carrier status, are especially susceptible, among the total Israeli tinea capitis study series. This study did not report thyroid cases. However, in another report from the Israeli series, the thyroid cancer pattern may also reflect this fact, although genotyping has not been done on the cohort and only some of the children were given ionizing radiation as opposed to UV therapy.

444. The cells of these individuals carry two copies of the "susceptibility" allele, one on each parental chromosome, i.e., they are homozygous for an abnormal allele (form of the gene). However, in their families there will be many heterozygous individuals whose cells have only one copy of the abnormal gene, the other being normal. Indeed, not only in these families but also, by virtue of the frequency of the susceptibility allele, in the larger population, there will be a substantial number of individuals, perhaps several per cent, who are heterozygous "carriers" of the abnormal allele. For them, affected relatives will be unlikely and would arise only when a heterozygote and another carrier marry, a relatively rare occurrence. Substantial speculation has centred on the likely cancer risks of individuals heterozygous for these genes. Little direct information exists as yet since there is no simple, easy test for heterozygotes. However, on the presumption that the parents of affected individuals must be carriers (except for very rare instances in which a mutation occurred in the child), some testing of the radiosensitivity of fibroblast cultures from these parents has been carried out. This work, though still tentative in nature, suggests that there is an intermediate level of radiosensitivity, measured by cell survival, between affected and normal homozygotes [P10]. While it is unlikely that excluding very small subsets of abnormally radiosensitive individuals would alter population risks importantly, their existence will require separate estimation.

445. The Li-Fraumeni syndrome is a dominantly inherited genetic susceptibility to cancers of many organ sites [L13]. Normal fibroblasts from Li-Fraumeni family members are resistant to killing by radiation; Chang et al. [C18] found that in non-irradiated cultures of such cells, the c-myc oncogene has a threefold to 18-fold increased expression and that c-raf-1 expression was also elevated. Kasid et al. [K23] have found that elevated expression of the c-raf-1 oncogene is associated with a cell line of laryngeal carcinoma which is radioresistant (in vivo and in culture). Why this oncogene should apparently be associated with carcinoma risk and radioresistance is not clear.

446. In retinoblastoma when the individual is an obligate heterozygote for a cancer-related region on chromosome 13, the subsequent risk of radiogenic tumour is a reflection of the susceptible genotype.

447. Less has been written, indeed less is known, about the role of genetic predisposition to specific malignancies and the relationship, if any, of this predisposition to radiation-induced risk. Are, for example, women who come from "breast-cancer families" more prone to develop breast cancer after irradiation of the breasts than women who do not come from such families? One study in Japan may begin to provide an answer [Y3]; there was an increased risk of second tumours in women with a family history of breast cancer relative to those without such a history, and there was evidence suggestive of an interaction with radiation in producing this risk. A substantial fraction of colon cancer cases is familial, although to date no study has looked for an excess susceptibility in irradiated persons from high-risk families. A variety of shared environmental factors could lead to a familial appearance of cancer, so that a definitive answer to the question of carrier susceptibility must await the development of practical tests for genetic susceptibility.

448. As reviewed in detail above, many second tumours following radiotherapy occur in individuals whose primary tumour was of a heritable kind. In some of these families there is an excess of cancers of other types in the relatives of the probands. Such relatives do not suffer the index disease, but they may be carriers of some modifier allele at a different genetic locus or, for some other reason, predisposed to develop cancer. Retinoblastoma and childhood sarcomas have both been involved in such studies. If these probands are from cancer-susceptible families, or if their cancer reflects a cancer-proneness, dose-response estimates for them may be of little value to the population as a whole, but the identification and characterization of susceptible genotypes may be extremely important in their own right.

449. Data relevant to these issues are sparse, but those that are available suggest there may be a small

but non-trivial fraction of the human population that is prone to develop cancer and, as a consequence, may be much more liable to develop radiation-related cancers. This may mean that the average dose-response pattern is a relatively poor indicator of individual risk. It may be too low for those individuals especially predisposed, and too high for those individuals who are not at special risk. However, to improve the estimation of risk, one must be able to identify the susceptible individuals, which is not practicable at present.

450. In ankylosing spondylitis patients there is strong association with specific genes at the histocompatibility loci, in particular an allele known as B27. Individuals with at least one copy of this allele are much more susceptible to spondylitis than are those with other alleles. It is thought that this may involve the development of auto-antibodies in such individuals after exposure to some agent, possibly an unidentified virus. At present there is no evidence that the HLA type is related to cancer or to cancer induction, so that from this point of view the ankylosing spondylitis patients may be thought of as representative of the general population.

D. ETHNIC CHARACTERISTICS

451. Shore et al. [S16, S27] have suggested that radiation-induced skin cancer is functionally related to the degree of pigmentation of the exposed individual. No increase in skin cancer with exposure has been seen among the 2,226 blacks who made up 25% of their tinea capitis population, but 41 cases occurred in the white children who made up the remaining 75%. Nor has an increase been seen among the Japanese survivors of the atomic bombings of Hiroshima and Nagasaki [C4, O2]. These results correspond to the prevailing skin cancer rates in blacks and in Japanese. This suggests that partially transformed cells, exposed to the DNA damage of UV radiation in light-skinned individuals, may become fully transformed. However, no UV-radiation cross reaction has been found in cell lines carrying UV-sensitive genotypes (reviewed above).

E. HORMONAL EFFECTS

452. A deficit in breast cancer has been observed in the cohorts of women who had been treated for gynaecologic disorders (malignant and non-malignant) [B12, L11, W6]. At the same time, ovarian cancer was seen to be reduced in women who had been treated for gynaecologic cancer [B12]. These observations agree with a cell-killing effect at the ovary, depriving the breast of oestrogenic compounds that may contribute to carcinogenesis in partially transformed breast cells. However, breast cancer was also reduced in women treated for benign conditions, who did experience elevated ovarian cancer [W6], the explanation is therefore not obvious at present, and non-representative subjects (i.e., subjects who present an inappropriate expected risk) may be responsible.

453. A study specifically designed to detect interaction between various hormone-related variables and breast cancer risk in women irradiated for postpartum mastitis was conducted in 571 patients and 993 controls in Rochester, New York, United States [S37]. This study found that women with benign cystic breast disease were at excess risk following radiation, but because the benign disease occurred after irradiation, a causal relationship could not be established. Oral contraceptive use, family history of breast cancer, late age of parity, menopausal hormone use and ovarian-related factors (cysts, missed menstrual cycles) were tested, but no interactive relationship with radiation was detected. Another United States study reported similar results [B1].

454. In the Japanese data [T14], breast cancer risk seems to be less if the radiation exposure occurred after menopause. However, this may be a cohort effect, attributable to different levels of hormonal stimulation in the United States and Europe on the one hand, and Japan on the other. Japanese-American women are developing age-specific breast cancer frequencies much like those of other Americans, and breast cancer is becoming more frequent at older ages in Japan. If this is a cohort effect, and if radiogenic breast cancer is related to the hormonal stimulation that appears to be responsible for these international differences, post-menopausal radiogenic breast cancer may increase in Japan [T14].

455. Tokunaga et al. found a slight but non-significant excess risk among women who had borne their first child after age 30 [T14]. Nulliparous women may also have elevated radiogenic risk [B1]. Similarly, irradiation after the first childbirth seemed to lead to elevated risk; age at first childbirth is certainly related to the occurrence of breast cancer, but how this interacts with radiation is not known. This and other studies seem to suggest an age effect on breast tissue susceptibility.

F. OTHER DISEASES

456. It has already been discussed whether individuals irradiated for the treatment of cancer suitably represent the general population in terms of their susceptibility to radiation-induced cancers. In general, most studies find similar patterns of radiation risk. An exception to this is excess sensitivity among children who have genetic predispositions, which suggests that children exposed for cancer treatment should not be considered for general risk estimates. Presumably some adults also have greater genetic susceptibilities for cancers, but these individuals cannot be identified in general populations.

457. Although the non-random HLA genotypes in ankylosing spondylitis patients (reviewed above) may not affect their radiation susceptibility, they clearly suffer from proportionally different causes of death than the general British population [S28]. Their pattern of relative risk for a variety of causes is given in Table 1 of [S28]. In addition to the colorectal cancer which may be confounded by the higher risks

of colitis and its associated cancer risks in these patients, many other causes of mortality are different from a general population. Even if this does not apply to their general cancer susceptibility, it clearly affects the interpretation of relative risks derived from the United Kingdom baseline cancer and general mortality rates.

V. ENVIRONMENTAL FACTORS THAT MODIFY RISK

458. It has been asserted, based on world-wide cancer data, that 85-90% or even more of all cancers are avoidable; that is, that they are due to exposures to environmental carcinogens, e.g., smoking, diet, personal habits and the like [D13]. This assertion is based on the observation that age-standardized incidence rates for virtually every type of cancer vary greatly among the populations of the world. Doll and Peto, in their review of cancer patterns in the United States, have estimated that 1-1.5% of all cases in that country are radiation-induced (non-occupational sources of radiation) [D13]; some of these cases will be due to natural background radiation, but the remainder will be due to man-made sources of exposure.

459. The most systematic data on variation in site-specific cancer incidence, by age and sex, is the series of volumes published by the International Agency for Research on Cancer (IARC) [W17]. They provide the best available baseline cancer risks and, while they include the effects of radiation on a population basis and cannot be used specifically to identify environmental causes, they can be used in computations of risk assessment, as discussed in chapter II. Radiogenic effects will be functions of the baseline risk. In addition, as seen in chapter III in regard to second cancers in patients irradiated to treat cancer, radiation risk is highly dependent on the effects of other risk factors.

460. The observed pattern of world-wide cancer variation is not highly correlated with ethnicity in any simple way that would suggest that this variation has a genetic basis, so it is generally assumed that environmental factors must be responsible. It is usually also assumed that in any population a large fraction of the baseline cancer rate, for any tumour site, is determined by local exposures to various agents (including radiation). Smoking and diet, pollutants in the environment, endogenous as well as exogenous hormones, and many other agents are known to be associated with variations in cancer risk.

461. Among the more elusive of the host factors and environmental factors that can influence the occurrence of a radiogenic cancer are the health status and life-style of the exposed individual. Their roles have been difficult to assess, for the variables themselves are difficult to define and to measure adequately. Smoking habits are more than just the sum of the cigarettes one smokes daily, weekly, annually or in a lifetime of smoking; moreover, these habits change with age and perception of risk. Data are beginning to be available that are relevant to three questions, namely: (a) what roles do smoking, diet, and the like play in cancer risk? (b) are life-style attributes related in any way to the absolute or relative risk differences in the irradiated? (c) can an understanding of the interaction of risks lead to an understanding of the action of radiation?

462. Regardless of the answers to these questions, relative and absolute excesses of cancer attributable to radiation must at present be evaluated in the context of local baseline patterns. For the cohorts of individuals who have been exposed to large doses of radiation, the levels of environmental risk exposure have been changing in the populations in which they live. Without adequate cognizance of these changes, it will be difficult to identify the effects of radiation, *per se*, or to say whether the effects of radiation are the same in populations with vastly different socio-cultural environments.

463. In addition to changes in mean levels of exposure within a population over time, there will be inter-individual differences in exposure that are relevant both to the analysis of specific cohorts and to the estimation of future risk. Although the data are limited, in many study cohorts it has been possible to make observations, or plausible inferences, about the role of certain environmental co-variates. These will now be reviewed.

A. SMOKING

464. Clearly, cigarette smoking is one of the most definite, and easily assessed, risk variables; it applies to cancer of the lung in smokers whose lungs are exposed to radiation, as well as to certain other tumours. However, Kato and Schull [K7] found no evidence that smoking increased the radiation-induced lung cancer incidence in Hiroshima and Nagasaki disproportionately. Prentice et al. [P7] and Kopecky et al. [K22], in a more elegant and broader analysis of the same data, reached a similar conclusion. Persons heavily exposed to both cigarette smoke and radiation had significantly lower cancer mortality for all cancers except leukaemia, stomach cancer and digestive cancer other than stomach cancer than predicted with a multiplicative risk model. The lung cancer relative risk function could not be distinguished as either a multiplicative or an additive form, though there might have been a slight preference for the latter.

465. Women treated for gynaecological disorders have experienced an increase in the incidence of cancer at smoking-related sites, including lung, bladder, oropharynx, and oesophagus [B2, B12, W6]. This has been observed in irradiated and non-irradiated patients. Since smoking is a well-established risk factor for cancer of the cervix, which typically selects for women of lower socio-economic status, some of the excess risk of smoking-related cancers in these women may be due to their smoking habits; alternatively, it may be that they were susceptible to cervical carcinogenesis for a variety of reasons; among them, smoking. They may have been more susceptible to smoke than the

population from which their expected rates were computed [B12, W6]. In a case-control analysis of a subset of the cervical cancer series, Boice et al. [B38] were able to show an association between smoking and smoking-related cancers, as well as for other known risk factors and their associated cancers (i.e., nulliparity effects on breast cancer risk, and obesity and menopausal oestrogen therapy for endometrial cancer).

466. The relative risk of lung cancer in the international cervical cancer study of women under age 40 was more than 12, a much greater risk than can be accounted for by the effects of smoking in the patients [B12]. For women who had not received irradiation, the relative risk was the highest of any group in the study, about 3, and a similar excess of bladder cancer was found in the women treated for benign conditions [B12, W6]. A different series, of 2,500 French women treated for cervical cancer, observed 10 cases of bronchial carcinoma, with only 28 months, on average, intervening between the diagnoses [S36]. This latency time is too short for radiation to have induced cancer in the lung. The possibility that the excess lung cancer, at least, reflects the misclassification of metastatic cervical cancer has also been considered [B12, D9, S36], although these authors argue that the persistence of excess risk in the large international study after 20 years suggests some of the excess may be real. Given the low dose of radiation received by the lung, a truly radiogenic effect would be difficult to detect [B12].

467. One extensively studied potential interaction is that between smoking and radiation in occupationally exposed underground miners. The alpha radiation of the radon daughters present in the air of underground mines provides long-term, generally low-level exposure to the lungs, as does smoking. Some aspects of these studies have been mentioned earlier.

468. Most of the miner populations studied had a high percentage of smokers, so that the independence of the two effects has been difficult to evaluate accurately. However, Navajo Amerindian men working in mines in New Mexico, United States, also developed excess lung cancer and relatively few of them were smokers [S20]. None the less, the data on smoking and radiation with respect to miners are complex and inconsistent [C4]. One study of iron miners in northern Sweden indicated that smoking had, roughly, an additive effect in the exposed miners [R7], while another [D12] purports to find an interactive effect. In their recent study of six Czechoslovakian mining groups, Sevc et al. [S51] found smoking to have an additive effect. Thomas et al. [T20] have found an effect intermediate between additive and multiplicative. However, in an analysis of data from uranium miners in the Colorado plateau (an area including parts of the states of Colorado, Utah, New Mexico and Arizona, United States), Whittemore and McMillan [W12] found strong support for an interactive relationship between smoking and radiation exposure. It should be noted that the dose estimates were uncertain and may have been higher than in other series. This could explain some of the difference in results [C4]. Also, while earlier analyses of these data had not revealed an interactive effect [C4], the more recent study used proportional hazards analysis, a better method than categorical analysis because it compares risk within the exposed cohort and is not dependent on assumptions about control group risks. Whittemore and McMillan also demonstrated that the models fit the data better when cumulative exposure (to both smoking and radiation) was used, instead of average annual exposure. The best data on this topic show that the interaction between smoking and radon daughter exposure is intermediate between additive and multiplicative [S51]. This was also the conclusion of the BEIR IV Committee [C20].

469. There is clearly a radiogenic risk of lung cancer in the absence of smoking as a co-factor. In general, the difference between the absolute risk for miners who smoke and that for those who do not has been small, but the difference in the relative risk between the two groups is great [C4, R7]. As was true in some mining studies [C4, R7], data from the Japanese atomic bomb survivors fit additive and multiplicative models almost equally well [P7, K22].

470. The time from first exposure to lung cancer has commonly been about the same for smokers and non-smokers [C4, R7], implying that radiation, and not smoking, was responsible. Even in the Colorado miners, where an interaction was inferred, there was no evidence for an age of onset difference [W12]. This finding disagreed with earlier analyses of these data [W13], which seemed to show such a difference perhaps due to overestimation of the exposures [C4, R7, W12]. The latency period reported for Japanese non-smokers is longer than that for smokers. Although some of this difference could also be due to errors in the estimation of smoking and radiation exposures, some of it may reflect the dissimilarity between chronic mining exposures and the single exposures in Japan. There have been large differences in the exposure levels of the various mining cohorts, and dissimilar doses can have disparate effects (interactions not apparent in some situations may be detectable in others).

B. DIET

471. Stomach cancer is very common in Japan, and some dietary factors have been implicated (e.g., the high-temperature cooking of meat and fish and preservation methods for fish). Thus, radiation exposures in Japanese can be expected to have effects that are related to the high level of exposure to these other risk factors. In a multiple regression analysis of stomach cancer in Hiroshima and Nagasaki, Ikeda et al. examined the relative importance of various risk factors for stomach cancer [I8]. Their study, which had a total residual (unexplained) variance of less than 1%, showed a statistically significant association between stomach cancer risk and age, sex, consumption of milk and consumption of broiled (but not dried or pickled) fish. Radiation dose had a much smaller, non-significant effect [I8].

472. Stomach cancer in irradiated persons in Hiroshima and Nagasaki tends to occur more often in areas of intestinal metaplasia, and the frequency of the latter appears linearly related to dose [M15]. As noted earlier, intestinal metaplasia in the lower third of the stomach is a characteristic precursor of gastric carcinoma in Japan, suggesting that irradiation has interacted with predisposing dietary factors. Because gastric cancer rates are dropping in Japan as diet changes, it is imaginable that Japanese exposed to radiation at a future time would have fewer partially transformed cells and, hence, a lower risk of radiogenic cancer. Radiation may induce cancer by increasing the prevalence of the precursor state as well as by further transforming cells already in such a state.

C. INTERACTION BETWEEN THERAPEUTIC MODALITIES

473. The findings in patients given combined radiation and chemotherapy for cancers, especially adult cancers, were described earlier. Generally, the strongest effects were those of chemotherapy, and some studies found no specific radiogenic effect on solid tumours or on acute non-lymphocytic leukaemia (ANL), only a chemotherapeutic effect. Some applications of radiation have probably led to intense local irradiation and cell sterilization, which may actually weaken the combined effect of the two types of therapy. It is not known if either the presence of disease or the method of applying the therapies affects the nature of any interaction. As reviewed above, in several adult cancer series, the relative risk of persons who had not received radiotherapy was greater than 1.0, which shows how important it is to have proper reference groups when comparing the effects of different modalities of therapy. Even if those treated with combined therapy show greater cancer risks, if the patients do not represent the population at large, the inferences about the effects of therapy, including radiation, will not be precisely applicable to a general population of exposed individuals. In any case, chemotherapy is commonly part of the treatment in which radiotherapy is used, so that the potential effects of the latter alone cannot be readily assessed; this is particularly so in view of the constantly changing therapeutic schedules and agents.

474. One of the most interesting sets of subjects in which to examine the effects of combined radiation and chemotherapy is the set made up of patients who were treated for retinoblastoma. Since, as noted earlier, about 25% of patients with this childhood tumour have a genetic susceptibility to it and to other cancers also, this cohort provides an opportunity to examine the effects of the different therapies both inside and outside a well-delimited radiation field. For patients receiving no chemotherapy, there are good baseline data.

475. An analysis of British retinoblastoma patients has been reported by Draper [D15] (Table 43). The small sample sizes and the fact that most patients given chemotherapy were genetic cases limited the analysis to those with the genetic form of the disease. The risk of a subsequent cancer among patients receiving both kinds of therapy was greater than for those receiving only one kind, both at 12 years and (especially) at 18 years after initial diagnosis. These differences were significant at the 5% level, although Draper noted that since the modes of radiotherapy may have been different for those also given chemotherapy, there could not be too much emphasis on the specific numbers. Thus, in genetically susceptible individuals, combined therapy adds to the risk of cancer; the excess risk is apparently experienced both inside and outside the radiation field. The fact that the effect of combined therapy seemed much greater after 18 than after 12 years suggests that an even greater effect may be revealed as more follow-up time accumulates.

476. In the data on Wilms' tumour [L3], combined chemo- and radiotherapy was given to four of the nine radiation-associated second tumour patients. Combined therapy did not increase the risk of cancer relative to radiation therapy alone, when all cancers were considered. The relative risk of chemotherapy, among irradiated patients, was 1.02. However, if only tumours arising in the field of irradiation were considered, the relative risk of chemotherapy was 1.50, significant at the 5% level. These patients received orthovoltage therapy.

D. STATISTICAL REFLECTION OF HIGH POPULATION RISKS

477. In the parallel analysis of cancer data from ankylosing spondylitics and the Japanese series, Darby et al. [D11] plotted both the relative risk and the excess risk of various cancers against the expected risk in the two populations. These are shown in Figure XIII. Most of the tumours cluster in a small area on such a graph ($RR < 5$; risk per 10^5 and year < 25). The points for stomach cancer, which is very prevalent in Japan, and for lung cancer, which is very prevalent in the United Kingdom, are among the few outliers on the respective graphs. Both exhibit very low relative risk contrasted to their population risks. It appears that the pattern observed by Darby et al. is, with the exceptions of leukaemia and CNS tumours, a linear one, with the relative risk roughly between 1.5 and 3 for all epithelial sites.

478. Central nervous system (spinal cord and nerve) tumours were much elevated, relative to their normally rare occurrence. This is probably because these tissues are seldom exposed to environmental mutagens (blood-CNS barrier), with almost no mix of spontaneous and radiogenic tumours in the data sets. For leukaemias, the relative risks were different in the two groups (about 3 in spondylitics and 9 in the Japanese survivors), but the population prevalences were similar. The relative risks for both sites (CNS and leukaemia) were different from the relative risks for most carcinomas. The explanation for this is not clear, but the discrepancy highlights the difference between haematopoietic and epithelial tissues and perhaps serves as indirect evidence for the importance of environmental risk factors in the epidemiology of radiogenic carcinomas.

E. GENERAL CONSIDERATIONS ABOUT INTERACTIONS

479. The many factors discussed in the previous sections indicate that there are numerous ways in which radiation could have, or could appear to have, an effect on the irradiated individual. Clearly, it is very important to ensure that the expected rates with which the risk in irradiated subjects are compared are representative of their population. The 1980 BEIR III Report [C4] attempted to derive summary risk coefficients of the excess cancers to be expected per 10^4 PYGy.

480. The BEIR III estimates [C4] were developed by considering all the data available, in an effort to synthesize the information from a variety of heterogeneous data sets. There are, however, pitfalls in such an approach. One reflection of the uncertainties surrounding the use of these estimates shows up in a comparison contained in one of the reports by the international group studying the risk of second cancer in cervical cancer patients [B12, D9]. This comparison showed a poor correspondence between the number of excess cancers (based on the expected number of cases in the referent populations of the eight collaborating tumour registries) and the number of excess cancers that was predicted by applying the BEIR III risk estimates [C4] to the doses and exposure times in the cervical cancer data. The comparison is given in Table 44. It should be remembered that the expected cases were based on risk coefficients per 10^4 PYGy derived largely from the Japanese T65 dose estimates, and the comparison in Table 44 is based on the cervical cancer data in [B12].

481. The biggest difference is in the leukaemias; the authors attribute this difference to cell sterilization in the pelvis and hence to an overestimate of the effective dose [B12], and which their case-control study, taking marrow-weighted doses and cell-sterilization into account, showed to be an artefact of assuming uniform dose to all marrow (i.e., when marrow-weighting is done, the discrepancy largely disappears [B36]. Dose-fractionation may also have reduced the apparent relative risk per unit dose relative to that found in other studies [B36]. However, the difference is too large to be attributed solely to that factor. In addition, more excesses of lung cancer have been observed than were predicted and fewer excesses of most other cancers, notably breast, kidney and bladder. These have plausible explanations which were reviewed earlier (paragraphs 269-284). In general, however, it must be emphasized that (a) cancer patients are not representative of the population at large in respect of many risk-factor exposures; (b) risk-factor exposures may be relevant to more than one type of cancer; and (c) radiation itself may have direct or indirect effects on the risk of cancer in other organs. Thus, to understand the true risk associated with radiation may be difficult, since good background cancer risks are not usually available for the special subset of individuals exposed to ionizing radiation.

482. These facts stress the importance of taking other environmental and host factors into account. They also show that whole-population exposures may in many ways be more informative than exposures of selected population subsets, and that internal controls, rather than the whole population, may be the most appropriate comparison group. In the instance of the cervical cancer data, the appropriate comparison group is probably the benign gynaecologic disease group (to the extent that they in fact did not receive radiation therapy themselves). The data from Hiroshima and Nagasaki are as close to the ideal in this regard as any available.

VI. SUMMARIES OF RISK ESTIMATES IN MAJOR COHORTS

A. STUDIES PROVIDING SUMMARY RISK ESTIMATES

483. This chapter provides overall summaries of radiogenic cancer effects from the most comprehensive data sources currently available. Details from studies of tissue-specific exposures or susceptibilities were reviewed in chapter III. Here, results relevant to risk estimation and projection are given.

484. There are only three sets of data from which radiation effects can be estimated for a large variety of sites: those for the Japanese atomic bomb survivors, the ankylosing spondylitis patients and the cervical cancer patients. In all three, the sample was large, the individuals were followed for long time periods and an essentially similar kind of exposure was received by many parts of the body. Each set of data has its own characteristics. The Japanese data included internal controls, as did the cervical cancer data (i.e., patients treated without radiation). All the data are for short-term, low-LET exposures. In the spondylitics and the cervical cancer patients, groups of tissues receiving approximately the same level of dose were analysed jointly; the whole-body exposures experienced in Japan could not, of course, be included in this analysis. Only from Japan do we have effective comparisons of the exposure effects in males and females. The Japanese cohorts also provide the most comprehensive data from which to estimate dose-response patterns.

485. The most commonly used measure of the effect of exposure on a given site is the number of excess cancer cases per 10,000 persons exposed to 1 Gy after one year (10^4 PYGy), although some estimates count experience only after a five-year or 10-year latency period. The methods by which this number has been computed were discussed in chapter II.

486. Summary estimates of site-specific risk coefficients are available for the Life Span Study in Japan [K7], the Nagasaki tumour registry [W5], the ankylosing spondylitis patients [S31], the BEIR III Report [C4] and an older report from the ICRP [I9]. Land [L11] has collated some additional data. These risk estimates can now be revised in the light of newer dose estimates and longer follow-up times. In this chapter, the latest reports available are summarized.

B. PROJECTION OF RELATIVE RISK IN THE MAJOR STUDIES

1. Results from exposure to treat cervical cancer

487. For most sites, the international study of cervical cancer patients [B12, D9] found that the number of excess cancer cases was smaller, except for the lung, than would have been predicted based on: (a) the number of person-years at risk; (b) the expected number of cases in the appropriate registry populations; and (c) the BEIR Committee estimates [C4] of the excess number per 10^4 PYGy for the same sites. This difference has already been set forth in Table 44, at which point the question was raised of how well the irradiated subjects typified their larger populations.

488. *Pooled heavily irradiated sites.* To summarize the general effects of heavy irradiation such as was received to treat cervical cancer, the authors have analysed the joint manifestation of second primary cancers occurring in heavily irradiated sites; that is, sites close to the irradiation and likely to have received more than 1 Gy (stomach, small and large intestine, liver, gallbladder, pancreas, uterine corpus, ovary, other genital organs, kidney, bladder, bone and connective tissue). These will be referred to as heavily irradiated sites. In general, the pattern observed agrees with what has been found in Japan and in the ankylosing spondylitics [D11]. The details of the separate registries and site-by-site analysis may be found in [D9]; the results are summarized in [B12], from which the following is taken.

489. The minimum latency period for the heavily irradiated sites after cervical cancer was about 10 years; the excess risk thereafter did not diminish for at least 30 years. Figure XV compares these exposed patients

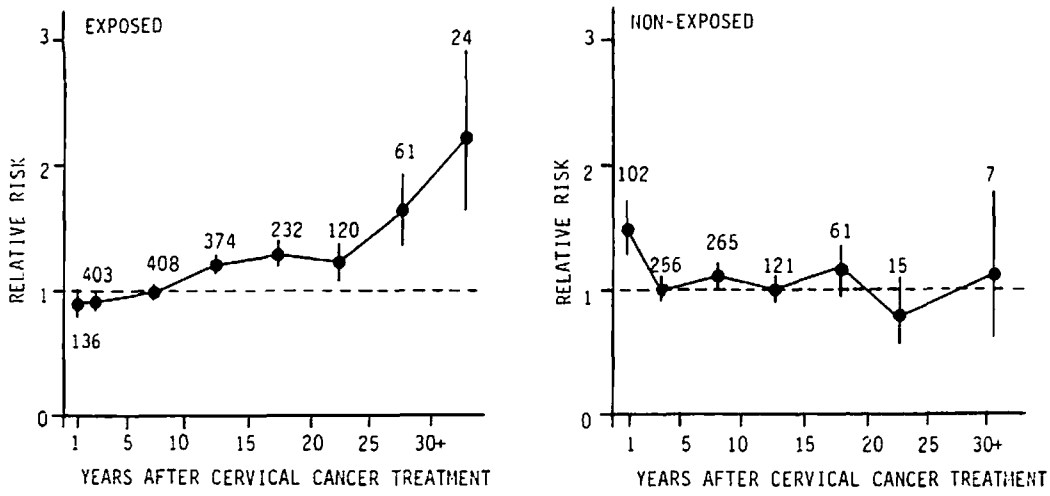

Figure XV. Risk of a second primary cancer occurring in or near the pelvis (close and intermediate sites), related to time of diagnosis of cervical cancer, for patients treated with and without radiation. The number of cancers are given above the 80% confidence bars.
[B12]

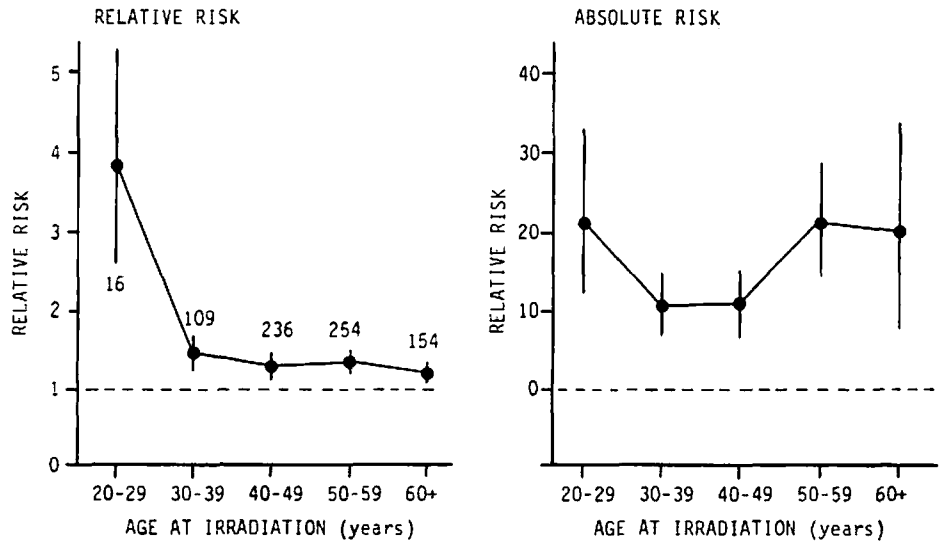

Figure XVI. Relative and absolute risks for second primary cancers occurring in or near the radiation field (close and intermediate sites), related to age at exposure, exclusive of the first 10 years of observation, for women treated with radiation. The number of cancers are given above the 80% confidence intervals.
[B12]

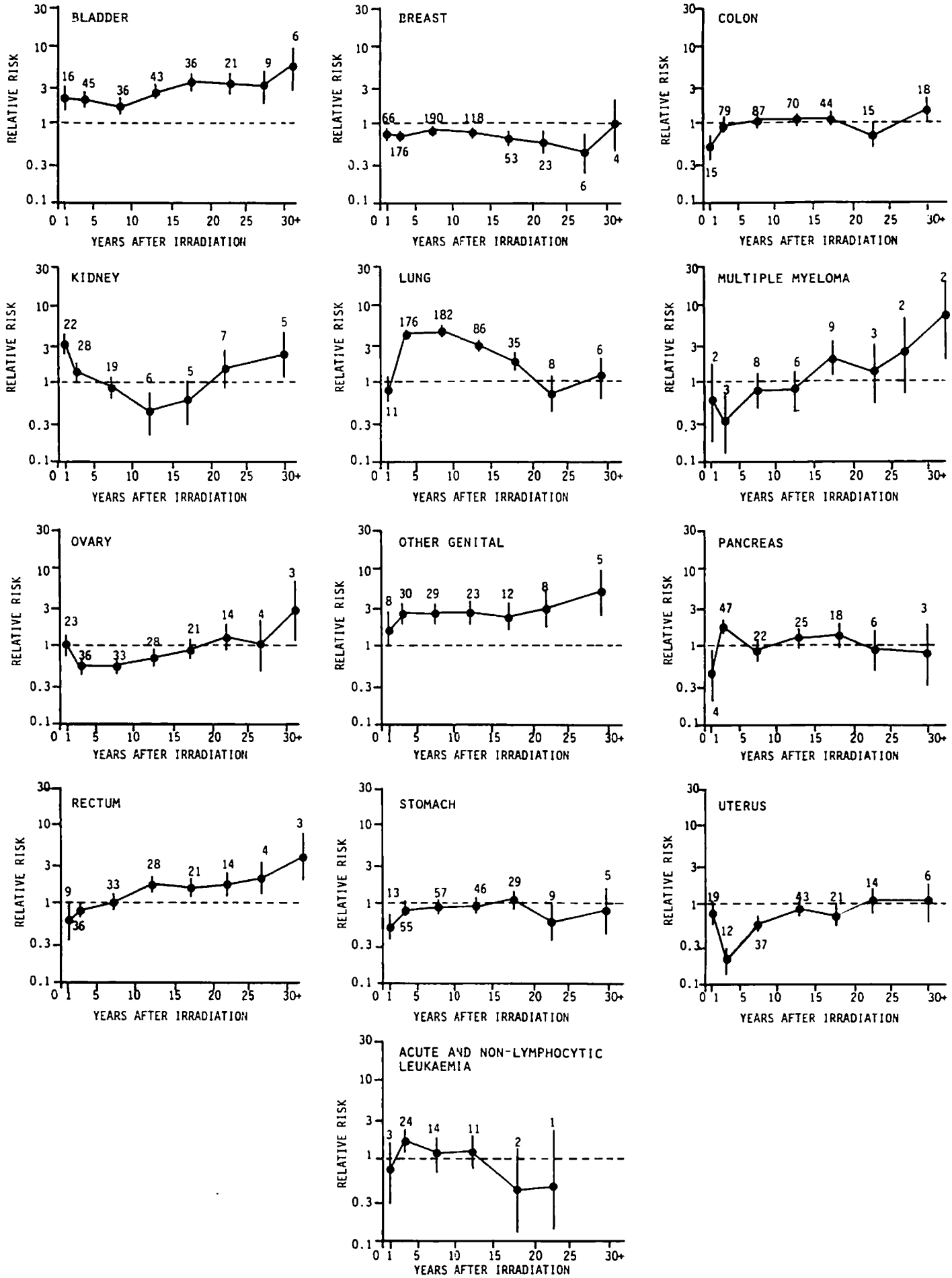

Figure XVII. Relative risks of second cancers in women irradiated to treat cervical cancer.
[B12]

with non-exposed cervical cancer patients, who manifested no increased risk when all the heavily irradiated sites were aggregated. Many of these women were followed for the rest of their lives (some lived into their nineties) and it was seen that risk apparently continues to remain elevated during all post-irradiation life [B12].

490. In terms of relative risk, women are more sensitive if irradiated under the age of 30, but thereafter age at irradiation appears to have little effect for these pooled sites. Since the background risk of cancer at these heavily irradiated sites increases with age, the absolute risk rises with age at exposure. This is shown in Figure XVI. The Japanese data have typically showed high sensitivity among those who were young at the time of the bombings.

491. *Individual heavily irradiated sites.* These same data have been analysed separately for each site [B12, D9]. Some of these findings are important enough to warrant review here. The patterns of relative risk following exposure at some of these sites are given in Figure XVII. Table 26 gives the most recent relative risk values for these projection effects, pooling data on all ages at exposure, based on a subset of the cervical cancer patients and a control group for comparison.

492. Figure XVII shows that bladder cancer increased gradually with time, with a relative risk of about 3.0. The projection effect appears to be radiogenic. As noted earlier, however, bladder cancer was among the smoking-related sites with similar patterns in the non-irradiated; but in the non-irradiated it decreased with time. While it is curious that rectal, but not colon, cancer was elevated, the projection pattern of rectal cancer is clearly the kind of pattern to be expected for radiation-related malignancies, and it obtained only among irradiated patients [B12]. Endometrial cancer (corpus uteri) had a relative risk of less than 1.0; this may be due, at least in part, to a high prevalence of hysterectomy. The increase in the relative risk 10 years after irradiation, with a levelling off afterwards, suggests a radiation effect. The marked and significant increase in "other genital cancers" refers mainly to an increase at the vulva, vagina, and unspecified sites; these sites probably share risk factors (e.g., HPV susceptibility) and may not reflect a radiogenic effect; they were at elevated risk in all groups in the study.

493. The bladder cancer projection is typical of a radiogenic epithelial cancer, except that it appeared in the first 10 years after irradiation. Boice et al. [B12] attributed this to mis-classified cervical metastases. After a 15-year latent period, there was no association between risk and age at exposure. The high level of risk and the increase over time suggest that more than the confounding effects of smoking are involved [B12].

494. In terms of sites remote from the source of exposure, an increase in smoking-related cancers was found, most dramatically in the lung. The incidence pattern (relative risk highest five to 10 years after irradiation and no excess cases after 20 years) is not typical of radiogenic lung cancer. The deficit of breast cancer is attributed to ovarian ablation by the radiation, which indirectly has a protective effect.

495. *Haematopoietic tissue.* Figure XVII shows the overall pattern of relative risk of leukaemias following irradiation. Pelvic marrow in these women received 3-15 Gy. While there was a marked deficit in leukaemias, relative to the BEIR III estimates (see Table 44), the pattern of excess leukaemias matches the pattern, with regard to projection effects, that is expected for radiogenic leukaemias (an excess beginning two to five years after exposure and diminishing after about 20 years). The relative deficit in excess cases is attributed to local cell killing [B36]; the dose received by the peripheral marrow is estimated to have been < 1 Gy, a dose which is leukaemogenic. The figure also shows the small excess of multiple myeloma and its persistent increase.

2. Results from exposure to treat ankylosing spondylitis

496. Results have recently become available that summarize some of the effects seen in the patients exposed to treat ankylosing spondylitis. The analysis presented here pertains only to the effects of single courses of x-ray treatment [D21, S28, S31]. The expected numbers were derived from British national mortality statistics. Table 45 presents the relative risks for major groups of sites as a function of time since irradiation [D21]. In these data the relative risk for leukaemia can be seen to rise rapidly and to persist beyond 25 years after exposure. The relative risks for the other sites remained approximately constant for the first 25 years after exposure, with values between 1.5 and 2.5, but then tended to disappear. Little effect, overall, was seen in lightly irradiated sites.

497. The site-specific projection effects are given in more detail in Table 46, also from [D21]. The authors found that for several sites the relative risk was elevated as early as up to two years after exposure as well as from three to nine years after exposure. They attributed this unexpectedly early appearance of excess relative risk to the possibility that tumours that had existed had been mistaken for ankylosing spondylitis, and they suggested that the relative risk at six to eight years' post-exposure was only slightly different from 1.0 [S31]. In their view, these data are consistent with the Japanese and other results for solid adult tumours in terms of the first appearance of excess risk, though not in terms of the disappearance of excess risk after 25 years.

498. Table 47 gives the relative risk values for the same series of patients as a function of their age at exposure, for leukaemia and for the heavily irradiated sites. The relative risk for leukaemia appeared to be slightly lower among those exposed at under age 25 and roughly constant afterwards, but none of the differences were significant. Also, the differences were not significant for all heavily irradiated sites combined.

499. A recent study has improved the dose estimates for the spondylitis patients [L16]. Earlier estimates, in

particular the BEIR III estimates, were very different from these new values, so that summary tables of the new values are included here for reference (Tables 48 and 49). This study was based on a sample of 934 patients (1/15 of the total series), for 903 of whom organ dose estimates are reported in the tables. Estimates were based on an Oak Ridge Laboratory program [W19] that models the process based on a mathematically defined human phantom. It is clear from the tables that there was great inter-individual variation in dose; thus, dose-response patterns from the entire series of over 14,000 patients are not based on precise individual exposure estimates. The new dose estimates are about 19% higher than prior estimates and very different from BEIR III [C4], although they are close to recent estimates by Drexler and Williams [D22].

3. Joint analysis of Japanese (T65D) and ankylosing spondylitis data

500. The detailed analyses of the risk effects from Hiroshima and Nagasaki, based on the revised (DS86) dose estimates, constitute the most important single data set on risk effects in existence. However, to augment the information from this and the large series of ankylosing spondylitis patients, Darby et al. [D11] analysed the Japanese and spondylitis data jointly. A summary of their results and of some of the basic data are presented. However, it should be noted that (a) the data from Japan apply to the old dosimetry (T65DR); (b) the spondylitis doses have been revised [L16]; and (c) the spondylitis risks have been revised [D21] since this joint analysis was prepared. Therefore, the joint analysis must be considered only rough and qualitative in nature, and it is to be read carefully in regard to carcinoma risks more than 25 years after exposure.

501. With these cautions in mind, Table 40 gives joint, summary relative and absolute risk values for the Life Span Study [W5] and the spondylitis patients [S28]. This paper [D11] provided some summary statistics on projection effects, with regard to a series of selected sites for which results between the two data sets could be meaningfully compared.

502. In the spondylitics, absolute risk standardized for time since exposure increased rapidly with age at exposure; in the Life Span Study, there was less evidence of such a trend. The result was similar for absolute risk by time since exposure standardized for age at exposure: a trend in the spondylitics but not in the Japanese. For the studies to be compared more directly, the observed Japanese risks were standardized to the same exposure-time distributions seen in the spondylitics, and this produced clear trends in the data from Japan for age at exposure and time since exposure. The joint estimate was an absolute risk of 31.7 per 10^5 PY (SE = 8.5) for every 10-year increase in age at exposure, and the studies did not differ significantly. In Japan, but not in the spondylitics, there was a statistically significant ($p < 0.05$) trend in time since exposure. The combined analysis showed no significant difference ($p > 0.10$), and the joint estimate was an average increase in absolute risk with time since exposure of 34.0 per 10^5 PY (SE = 15.7) for each six-year time period. This was significant at the 5% level.

503. Relative risk results are summarized in Figure XVIII, which shows that in the ankylosing spondylitis

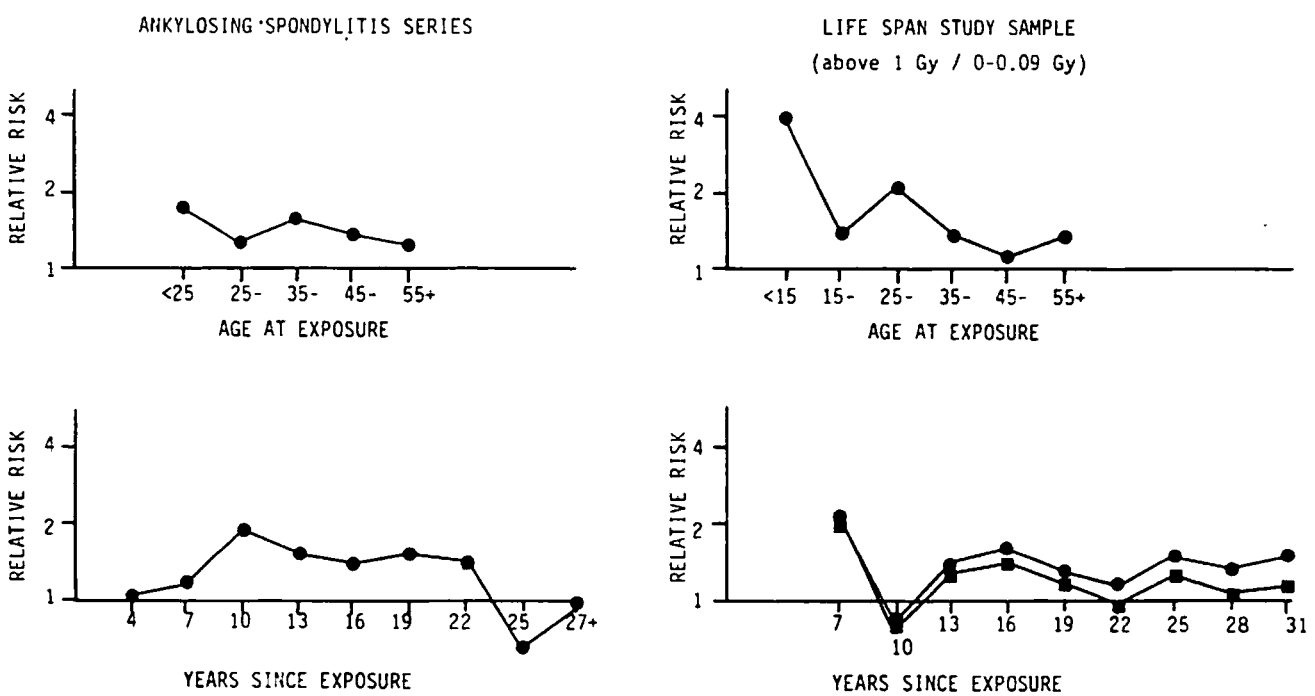

Figure XVIII. Relative risk of cancer in selected sites combined in relation to age at exposure and time since exposure for the ankylosing spondylitis series and for the Life Span Study sample (T65DR doses).
[D11]

patients there were no trends in relative risk for age at exposure, and only before 10 and after 22 years did relative risk fall below its generally flat projection pattern; there was no evidence that these two factors interacted [D11]. The data from Hiroshima and Nagasaki showed significant linear and quadratic trends in relative risk with age at exposure (declining with increasing age), and a log-linear trend in relative risk could be fitted to the data. The trend in relative risk with time since exposure was roughly level, both before and after incorporating a log-linear trend in relative risk with age at exposure. In analysing these jointly, Darby et al. found no trend in the relative risk in time since exposure; for five to 30 years after exposure, the relative risk remained about constant. Jointly there was a significant log-linear trend in relative risk for age at exposure, with the joint estimate of the trend in log (RR) with exposure age of -0.5 (SE = 0.13).

4. Recent results from studies of the Japanese exposed to the atomic bombings

504. The Atomic Bomb Casualty Commission and its successor, the Radiation Effects Research Foundation, have at various times used a variety of measures of individual exposures to the atomic bombings of Hiroshima and Nagasaki. These included distance from the hypocentre [M38]; the presence or absence of the symptoms associated with acute radiation illness [N10]; and, now, no fewer than three separate physical dosimetries, namely, the T57 [R19, A17], (for an application of these doses, see [I10]), the T65 [M24], and the new Dosimetry System 1986, or DS86 [M39, N11, R20]. Each successive method of expressing doses has necessitated a re-estimation of the risk coefficients associated with the various known effects of ionizing radiation and a re-examination of the dose-response relationships. With these changes there has emerged a better ability to estimate the risk coefficients in terms of organ absorbed doses or organ dose equivalents rather than in terms of kerma, whether free-in-air or in-house (shielded).

505. The newest system of dosimetry is an outgrowth of a series of events. In 1975, Preeg of the Los Alamos National Laboratory (United States) re-examined the gamma-ray and neutron spectra from the Hiroshima and Nagasaki atomic bombs using a one-dimensional model and discovered that they differed considerably from the spectra used in calculating the T65 doses. Simple calculations based on these spectra suggested that the T65 neutron dose was markedly overestimated for Hiroshima. Subsequently, Loewe and Mendelsohn at the Lawrence Livermore National Laboratory (United States) and Kerr and Pace at Oak Ridge National Laboratory (United States) independently calculated the air doses at Hiroshima and Nagasaki and reported that both the neutron and the gamma doses differed substantially from the T65 estimates. These findings prompted a complete re-evaluation of the atomic bomb radiation dosimetry (for a fuller account of the events that preceded this reassessment, see [R20]). The reassessment, begun jointly by the Governments of Japan and the United States, culminated in March 1986 in a consensus system known as the Dosimetry System 1986 (DS86). The principal differences between this system and the T65 dosimetry, the heretofore most commonly used system (see Table 50), are as follows [R20]:

(a) the yield of the Hiroshima weapon is now presumed to have been approximately 20% greater than had earlier been thought; that is, 15 rather than 12.5 kilotons;

(b) although the free-in-air (FIA) gamma doses are somewhat greater at distances of 1.4 km or more in Hiroshima, neutron exposures are less in both cities, and substantially so in Hiroshima, about 10% of their previously estimated value (30% in Nagasaki). Since delayed radiation from the fireball makes a relatively greater contribution to the total DS86 dose, the loss or gain of shielding as a result of the blast effect, particularly in the first several seconds following the detonation of a bomb, could substantially influence kerma in shielded areas and, ultimately, organ absorbed dose. Time-dose dependencies have not, however, been taken into account either in this new system of dosimetry or the old;

(c) attenuation of the FIA gamma kerma by wooden Japanese structures, houses and tenements is approximately twice as great under the DS86 than under the T65 dosimetry (the average transmission factors under the two systems are 0.90 (T65) versus 0.46 (DS86) in Hiroshima and 0.80 versus 0.48 in Nagasaki). However, attenuation of the neutron kerma by such structures differs much less strikingly (the average transmission factors are 0.36 (T65) versus 0.31 (DS86) in Hiroshima and 0.41 versus 0.35 in Nagasaki);

(d) transmission of gamma rays through tissue is significantly higher, at least for the deeply situated organs, than had previously been estimated [K14]. It must be borne in mind, however, that in the T65 system each specific organ transmission factor is a constant averaged over all postures, orientations and ages; whereas in the DS86 system, fixed values are not used for the proximally or "heavily" exposed (defined as those survivors within 1,600 m in Hiroshima and 2,000 m in Nagasaki), where detailed exposure histories are generally available. Their organ doses reflect the circumstances of their individual exposures, including posture, orientation and age at the time of the bombing. The increased tissue transmission for most organs tends to offset, wholly or largely, the changes in the shielding transmission factors;

(e) finally, for some 18% or so of the exposed members of the Life Span Study cohort (largely individuals surviving in concrete buildings or factories), doses cannot as yet be computed, and the new dosimetry improves but does not clarify all of the implausibly high exposures seen with the T65 system. There remain a number of survivors whose estimated whole-body shielded kerma exposures exceed 4 Gy or, in some cases, 6 Gy. These are doses at or above the recently estimated LD95 in these cities [F13]; given the virtual obliteration of the immune system at doses in excess of 7 or 8 Gy, survival under the

circumstances that obtained in these cities would be most unlikely. Better means are needed to address these incongruities than the simple truncation of dose, since their inclusion in analyses can affect the shape of the dose-response relationship as well as estimates of its parameters [G13, G18, J6].

506. As previously seen with the T65 doses, a statistically significant increase in the frequency of deaths with increasing dose is observed for leukaemia, cancers of the oesophagus, stomach, colon, lung, breast, ovary and urinary bladder and multiple myeloma. No significant increase is as yet observed for cancers of the gallbladder, pancreas, uterus and prostate or for malignant lymphoma. The most recent report was extended to include other sites of cancer, such as bone, pharynx, nose and larynx, and skin except melanoma, but none of these sites showed a significant dose-response relationship [S49]; however, mortality from tumours of the central nervous system other than the brain tends to increase with dose ($0.10 > p > 0.05$) (mortality from brain tumours alone does not).

507. While the excess in leukaemia mortality has declined with time, it none the less remained significantly elevated as late as 1981-1985, showing that the period of risk is at least 40 years rather than the commonly supposed 25. For cancers other than leukaemia, excess deaths continue to increase with time in proportion to the natural cancer rate for the attained age, and the relative risk remains unchanged over time for all specific age cohorts except the youngest, i.e., 0-9 years at the time of the bombings. For the latter cohort, unlike the older ones, the time from exposure to death is shortened with increasing dose for all cancers except leukaemia, and the relative risk decreases with time (Tables 51 and 52).

508. Tables 51 and 52 show the time course of excess risk in the DS86 subcohort data as a function of age at and time since exposure, for relative and absolute excess risk, in 10-year groups. The relative risk at 1 Gy changes significantly with time after exposure. The magnitude of this change is known only over a limited time period (about 40 years) since exposure.

509. Tables 53 and 54 give three summary measures of risk, namely, the excess relative risk at 1 Gy, excess deaths per 10^4 PYGy and the attributable risk, for all malignant neoplasms, leukaemia, all cancers except leukaemia, and eight specific sites of solid tumours based on the T65 and DS86 systems. These risks were derived by fitting a linear dose-response model to the data from both cities, both sexes and all ages at exposure within that subset of individuals in the Life Span Study sample for whom both T65 and DS86 doses are presently available (some 82% of all exposed individuals in the sample). Note that in so far as shielded kerma is concerned (Table 53), under the DS86 system, the excess relative risks are increased from 35% (stomach cancer) to as much as 53% (cancer of the ovary and other uterine adnexa). For excess deaths, the corresponding figures are 31% (multiple myeloma) and 61% (cancer of the ovary and other uterine adnexa). However, for no site or group of sites does the attributable risk change as much as 10%. For organ absorbed dose (Table 54), with the exceptions of cancer of the female breast or the ovary and other uterine adnexa, the risks are invariably lower with the DS86 doses and as much as 30% lower in the case of cancers of the stomach (excess relative risk).

510. Over the range of doses from 0 to 6 Gy, there is no clearly significant evidence of non-linearity (although other forms of response fit the data), so from a purely statistical point of view linear risk estimates are a reasonable summary of the dose-response. Moreover, when linear, quadratic, and linear-quadratic models (with or without provision for cell-killing) are fitted to the data on all cancers except leukaemia and on those five sites where a clear dose-response curve had previously been obtained (i.e., leukaemia, and cancers of the stomach, colon, lung and female breast), a simple linear model fits the data on leukaemia, cancers of the stomach, lung and female breast, and all cancers except leukaemia better than the quadratic model and as well as the linear-quadratic model, as judged by the deviance (that is, twice the difference in the log likelihoods under the full model, which exactly fits the data, and under the model based on the parameters that have been estimated). Inclusion of cell-killing does not significantly improve the fit, except in one instance where leukaemia mortality under either the linear or linear-quadratic model fits somewhat better with a cell-killing term. These findings hold true both for organ absorbed doses and shielded kerma.

511. Under the DS86 system, the neutron doses, although not wholly negligible, are so small that meaningful estimation of the neutron RBE is difficult, if not impossible. Reasonable RBE estimates cannot be derived directly through maximum likelihood estimation; however, some insight is possible if it is assumed that the small inter-city differences that still obtain reflect differences in neutron exposures. With the DS86 organ doses, assuming an equality of excess relative risk between Hiroshima and Nagasaki, the neutron RBE for leukaemia is 20-30; for all cancers except leukaemia, 30 or more; and for cancers of the stomach, lung and female breast, less than 1. Based on an equality of excess deaths between the cities, the neutron RBE for leukaemia or all cancers except leukaemia is 30 or more; for lung cancer, 10-20; and for cancers of the stomach and female breast, less than 1. The disparity between these estimates attests further to the difficulty of deriving meaningful estimates of the RBE with the new dose estimates for the survivors.

512. The differences between the cities are smaller under the new system than under the old for all sites of cancer, including leukaemia, and are no longer statistically significant. However, at face value, mortality in Hiroshima remains higher at most doses than in Nagasaki, for leukaemia as well as all cancers except leukaemia. This fact, when coupled with a similar consistent tendency for other indices of radiation damage (such as the frequency of chromosomal aberrations, lens opacities, and epilation), suggests that some explanation for the small inter-city difference in dose response is still necessary.

C. UNCERTAINTIES ASSOCIATED WITH RISK ESTIMATES

513. Even after many decades of study, the uncertainties that surround estimates of the carcinogenic effects of radiation are many and fundamental. Indeed, there is still no model of the underlying process that is clearly the correct one. The importance of a good theoretical model is greatest where the data are weakest, at low doses of low-LET radiation, so that our ability to estimate these risks is severely limited.

514. Because the majority of all cancers appear to have an environmental origin in the sense that avoidable exposure to environmental risk factors is involved [D13], much of this Annex has dealt with the problems in risk evaluation caused by the existence of multiple risk factors. In addition to environmental risk factors, there are also many host factors, such as genes, age, hormonal status, sex and the like, that affect risk.

515. It is probable that in any population exposed to ionizing radiation there is variation in the exposure to other risk factors. At low levels of radiation, this variation may be greater, perhaps much greater, than the risk produced by the radiation itself. It is not surprising that it is difficult to estimate risk at low doses or that different results are often obtained. The methods used to estimate confidence intervals tacitly assume that all exposed individuals in a given category (e.g., age, sex or dose) have equal risk, which seems unlikely to be true.

516. The most important documented other risk factor is smoking. Another source of bias is the "healthy worker effect": in occupational cohorts, workers are often healthier than the general population so that their baseline cancer rates may differ from the rates of the larger population, complicating the problem of determining the expected number of cases in these cohorts unless control groups from within the cohort are used.

517. The twentieth century has been a period of rapid change in levels of exposure to cancer-causing agents in all populations in which radiation exposure data are available. This is reflected in changing cancer rates within populations over time. All of the major cohorts used in radiation biology to estimate cancer risks have experienced changing exposure levels, though it has not been possible to account for this well in any study. Estimates of risk derived from cohorts that have been followed for the past half-century to the present will have inexact application to cohorts exposed now or in the future, and the degree of the inexactness is not known.

518. There is substantial variation in general mortality rates in different countries. Exposed individuals may be expected to experience somewhat different lifetime risks in a developing country as compared with an industrialized one. However, much of this difference in overall mortality occurs during childhood and would have little effect. Also, cancer rates for most sites are lower in developing countries, so that the absolute excess, and perhaps even the relative risks for the same dose, may be different. Most large population exposures studied to date have occurred in industrialized nations; there are few data and perhaps less exposure in the developing countries.

519. In addition to changing baseline cancer risks and the effects of other exposures, there is uncertainty over the dose-response pattern. Most current studies use a linear model for breast and thyroid cancer and a linear-quadratic model for other sites; these are the best-available models only, for the data do not really permit the validation of a specific model with confidence. It is unfortunately true that most estimates of low-LET, low-dose effects are based on extrapolations from high-dose data.

D. UNCERTAINTIES ASSOCIATED WITH RISK PROJECTIONS

520. The many uncertainties involved in the estimation of risk from observations on exposed cohorts have been reviewed in this Annex. The main limitations may be stated as follows: (a) no single large cohort has as yet been followed throughout its entire lifetime, so that the lifetime effects of exposure cannot be empirically determined; (b) the data that are available are most incomplete for those who were young at exposure: these individuals have not reached an advanced enough age for the bulk of their risk to be expressed, yet their lifetime risks may be the greatest; and (c) there are not now, and for the foreseeable future will not be, sufficient data on low doses to allow useful risk estimation.

521. In the face of these limitations, there are formidable problems in determining what model(s) to use in projecting risk forward into the unobserved future lifetimes of potential cohorts. Once the current cohorts have been completely observed, risk projection will have a sounder empirical basis.

522. The work of Muirhead and Darby [M36, M37], cited earlier, as well as models applied by the Radiation Effects Research Foundation (RERF) [S49], shows clearly that, depending on what covariates are considered and on how their effects are modelled, one can obtain strikingly comparable degrees of fit even to the best available data on the major exposed cohorts. In the Muirhead and Darby models, the parameter γ expressed, at values of 0 and 1, the 'pure' multiplicative and additive projection effects; however, the most likely value of this parameter was sometimes statistically indistinguishable from either of these models under certain combinations of covariates. With an intermediate value of γ, the intuitive biological meaning of the model becomes unclear, and the model is probably best thought of as an empirical one only. Given this, it is clear, as noted in Muirhead and Darby [M37] that a variety of models could be constructed with approximately equivalent goodness-of-fit.

523. Although the goodness-of-fit of several projection models to the empirical data may be comparable,

their projected lifetime risks may not be as close. Being, therefore, currently unable to make lifetime projections with much confidence, alternative models have to be presented, which, hopefully, bracket the true risks. Even so, there is no way of specifying quantitatively the degree of uncertainty in these alternatives.

524. Until very recently, it had appeared from experimental animal data and empirical data on humans that the relative risk projection model was the more appropriate of the two models for most solid carcinomas. However, if the excess risk of these tumours eventually declines with advancing time since exposure, as some data now suggest, then neither simple additive nor simple multiplicative projection effects will pertain, and none of the models, even hybrid models such as those of Muirhead and Darby, which describe the effects of time since exposure in a monotonic way, will be applicable.

525. These are fundamental problems, for it does not currently appear possible to discriminate among the various projection models based on their fit to empirical data. The only practicable solution to this problem may be to wait until the experience of the Japanese, the spondylitics and the other cohorts is more fully expressed than at present and to derive empirical projection models. Even so, changes in baseline risks, as well as confounding cancer risk factors, may make such projections inaccurate for future cohort experiences.

VII. RISK PROJECTIONS

A. GENERAL CONSIDERATIONS

526. In chapter II of this Annex, the various concepts related to risk projection, the kinds of data required, and past efforts to estimate lifetime risk from exposure to ionizing radiation were discussed. In this chapter, the most appropriate existing data will be used to compute estimates of lifetime risk for those cancer sites for which sufficient information exists to make meaningful projections. The purpose is to derive approximate estimates of the risk of exposure to low-LET radiation, taking into account sex, age at exposure and time since exposure for the lifetime of an entire exposed population.

527. Radiation-induced mortality in a population may be represented in a number of ways, most commonly as either the expected lifetime number of excess cancer deaths in the exposed population or the number of person-years of life lost because of cancer deaths, both per unit collective dose. Estimation of these expressions of risk remains formidable, as does a meaningful synthesis of the estimates that are already available. The task is complicated by one or more of the following main difficulties: (a) the unique nature of some of the samples from which risk coefficients have been derived; (b) the differences between studies in sample sizes and in the periods of follow-up; (c) the methods of case ascertainment that have been employed; (d) the poor knowledge of the doses received and their distribution over sites; and (e) the nature of the comparison groups used.

1. Whole-body risk coefficients

528. Some of the numerous studies described elsewhere in this Annex, although important in their own right, are of limited value for projection purposes; they provide the relative frequency of occurrence of cancer in an exposed group, as contrasted with a non-exposed referent one, but often at only one of the many sites of interest, and the dosimetric uncertainties make estimates of the risk of cancer per unit dose difficult. Commonly, doses in these particular cohorts have been concentrated in one part of the informative range for estimating dose-response patterns, making it difficult to estimate effects at low or intermediate doses. Three studies, namely, those of the ankylosing spondylitis patients [D11, D21], the women treated with radiotherapy for cervical cancers [B12] and the survivors of the atomic bombings of Hiroshima and Nagasaki [S48, S49], have been the bases for estimating the frequency of occurrence of cancer, per unit dose, at multiple sites of malignancy. But, even here, however, derivation of a combined risk coefficient is difficult and has not been attempted.

529. While these three studies agree generally in identifying the sites at which the frequency of cancer is elevated following exposure to ionizing radiation, the study-specific estimates of the excess relative risk for specific malignancies per unit dose, based on the information currently published vary, particularly in so far as the cervical cancer series is concerned. There are numerous reasons why this should be so, but especially pertinent are the conditions of exposure, the nature of the dose data presently available, differences in the age or sex distribution of the exposed individuals in the study samples, the dissimilar periods of follow-up, and the background rates used to compute the expected number of cases. Table 55 summarizes the main characteristics of these three studies and illustrates the differences between them in the respects just enumerated. These points were reviewed in chapters III and IV.

530. Exposure of the patients with cervical disorders or ankylosing spondylitis occurred because of illness; this was not the case among the Japanese survivors. The reasons why these patients may not be representative of the general population were discussed in chapter IV. In the two patient series, exposure was to either x rays or gamma rays, whereas the atomic bomb survivors received a mixed dose of gamma rays and neutrons, albeit primarily the former. The Japanese sample alone includes a full representation of sexes and ages; the other two studies are restricted either wholly or largely to one sex, and they do not include a sufficient number of individuals below the age of 25 at the time of exposure, when the excess relative risk appears to be larger, to provide an estimate applicable to a general population. Individual estimates of dose are not available for all (or even the majority) of the patients with cervical disorders or ankylosing spondyl-

itis. Risk coefficients have been derived from the mean dose among a 7% random sample of the spondylitis patients, and dose-response estimates, based on the individual doses received by the cervical cancer patients in that series, encompass only a subset of the patients. However, mean doses are often poor descriptors of the dose distribution, notably among cancer patients, because of the highly skewed nature of the individual doses and their wide range. As to the periods of follow-up, the maximum length of follow-up of the first sample members is similar in the three studies, but since enrolment proceeded over a longer period of time in the two patient series, the mean years of surveillance for them is substantially shorter than for the atomic bomb survivors. In terms of sample size, the atomic bomb survivors and the cervical cancer series are approximately equivalent, but the number of the person-years at risk in the study on the atomic bomb survivors is much larger. In the cervical cancer and spondylitis series, unlike the atomic bomb survivors, the variation in doses among exposed organs is very different, because the treatment was concentrated on one part of the body. This makes whole-body equivalent dose estimation difficult. There have also been marked changes in the nature of x-ray equipment and in therapeutic methods. Finally, there are differences in the nature of the referent groups in the three studies; these were thoroughly discussed earlier in this Annex (see chapters I and III). A summary of the excess relative risks per gray obtained in these three studies is provided in Table 56.

531. A special feature of the atomic bomb survivors is that they received exposure from low-LET radiation and from neutrons simultaneously. It is important therefore to consider how the projections to follow would be affected by the assignment of either a fixed or a dose-variable RBE for neutrons, relative to gamma rays. Figure XIX provides, in graphical form, the change in the estimated number of excess deaths

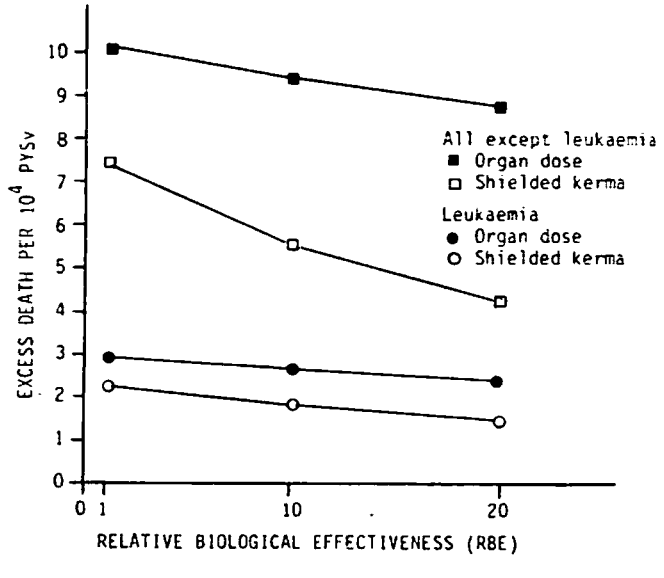

Figure XIX. Changes in risk coefficients for varying values of the RBE of neutrons, relative to gamma rays, based on Japanese DS86 data.
[S48]

with fixed values of RBE varying from 1 to 20. It will be noted that the risk coefficients for both leukaemia and all cancers other than leukaemia become smaller as the assigned RBE increases. Over the range of 1 to 20, the risk estimates based on shielded kerma diminish by about 54-72%; however, the estimates based on organ dose equivalent diminish by only 15-20%, reflecting the higher transmission values associated with gamma- and neutron-radiation under the DS86 system of dosimetry. Elsewhere, Shimizu et al. [S48] have shown that the use of a variable RBE, one changing as a multiple of the inverse of the square root of the neutron dose, would have an approximately equivalent effect on the projections. Thus, for example, the estimate of the excess deaths from leukaemia, using an RBE equal to $1/\sqrt{D_N}$, where D_N is the neutron dose in gray, is 2.93; whereas that based on an RBE equal to $20/\sqrt{D_N}$ is 2.47. Similar changes occur for excess deaths attributable to cancers other than leukaemia; the risk coefficient changes from 10.08 to 8.86 excess deaths per 10^4 PYGy, or slightly less than 20%.

2. Site-specific risk coefficients

532. In addition to the three studies that involved (albeit under different conditions of exposure) the simultaneous irradiation of many tissues in the body and from which risk estimates could be extracted and their relationships analysed, there are a large number of other studies in which single tissues were exposed to radiation for a variety of purposes and from which risk estimates to individual tissues have been derived, as reviewed in detail in chapter III. The risk coefficients resulting from these studies are summarized in this section.

533. Since the UNSCEAR 1977 Report [U2], in which radiation carcinogenesis in humans was last reviewed, several organizations have estimated summary risk coefficients (absolute or relative) for a variety of major organ sites. These include studies by BEIR III [C4] in 1980 and by the United States Nuclear Regulatory Commission in 1985 [G11], and the United States National Institutes of Health, also in 1985, in the radioepidemiological tables [U3]. Their reports attempted to combine the literature available at the time. The BEIR III Report synthesized individual studies along with the most recent Japanese data then available; Gilbert [G11] and the radioepidemiological tables relied heavily on BEIR III, modifying their estimates according to a few other reports but mainly basing them on the then-latest data from the patients treated for ankylosing spondylitis and the atomic bomb survivors. Each of these studies, to which the reader is referred, discussed the reasoning behind its respective site-specific coefficients. For comparison purposes only (because the information on which they are based is now out of date), the summary risk coefficients for each study are given in Tables 4 and 8 [C4, G11].

534. Because these studies appeared in the period 1980-1985, they could not include the results of the latest data and dose revisions in Japan [R20, S49] and

in the spondylitis series [D21, L16], nor could they include the recently published risk coefficients from the cervical cancer series [B36, B38]. Site-specific risk information from the most recent studies is given here in Tables 26, 46, 51, 52, 53 and 54. These tables provide the current estimates of risk from the three largest studies based on age at exposure, time since exposure and sex. The scientific details of these studies were discussed earlier, along with the limitations and specific characteristics of each study population.

535. Chapter III of this Annex reviewed many other studies in which site-specific risk coefficients were estimated. While they vary greatly in their particulars, e.g., sample size and the like, it is worth summarizing the risk coefficients from these studies. For each site, reference will be made to the tables and figures, discussed in chapter III, that provided the best data on risks. Also, the principal bibliographic references for each site will be given, as will be the general range of risk estimates from other studies not already included. Note that the estimates in the following summary that are derived from the Japanese or spondylitics are based on the old dosimetry and do not include the most recent data reports (these values are given in Table 56).

536. *Leukaemia.* While many studies of different types of exposure have found an excess of leukaemia, the best risk coefficients come from the spondylitis patients [D21, L16, S31], the Japanese data [S48, S49], and, very recently, from the marrow-weighted study of cervical cancer patients [B36]. These individuals received exposures to external low-LET radiation. The risk coefficients are included in Table 56. Some other dose-response data, from gynaecologic patients, were summarized in Table 31. The BEIR III Committee estimated the absolute risk coefficient for brief exposure in childhood to be about 0.01-2.2 excess cases per 10^4 PYGy [C4; see Tables V-17 and V-18], and Ron and Modan [R1] estimated absolute risk to be 0.60 male and 0.87 female cases for the same unit of exposure, although this difference was not statistically significant.

537. *Multiple myeloma.* Risk coefficients for multiple myeloma are summarized in Table 32. The most important reference for these data is [C10]. A recent estimate from Japan gives 0.48 incident cases per 10^4 PYGy bone marrow dose [H5, I2]; however, the latter estimate is based on the T65 dosimetry.

538. *Bone.* Table 33 provides a summary of risk coefficients for bone cancer based on an analysis by BEIR III [C4]. Figure VIII presents dose-response data. Other estimates for gamma-emitting radionuclides are 2.4 excess cases per 10^4 PYμCi for long-half-life isotopes (absolute); 1.8 (children) and 1 (adults) for short-half-life isotopes; and for alpha-emitting radionuclides (absolute) 200 per 10^4 PYGy [T11]. There are many cases of bone cancer in exposed children, but dose-response estimates are unreliable relative to a general population since most of the children, exposed during treatment of retinoblastoma, are genetically susceptible to osteosarcoma even in the absence of irradiation (see section III.B).

539. *Breast.* There are no data on breast cancer in males. Tables 36, 37 and 38 summarize the details of risk coefficients from a variety of studies. For adult exposures, the range of absolute risk coefficients is 3-10 per 10^4 PYGy, and that of relative risk coefficients is 2-5. For juvenile exposures the data are less reliable, but absolute risk coefficients are 3-8 per 10^4 PYGy [B6, T6] and the excess relative risk at 1 Gy, based on death certificates, is about 0.69 in the most recent T65D data from Japan [P15].

540. *Thyroid.* Thyroid cancer risks (incidence) are summarized in Tables 20, 21, 22 and 39 and in Figure I. Other estimates are 1-4 per 10^4 PYGy [C4, Z3] for adults and 1.5-9.5 for children [S13, S38], as judged by a variety of studies, including those of exposure to fallout. A major recent report discusses thyroid cancer induction in detail [N5]. There is about a 3:1 sex ratio of cases, with females predominating, and it has been estimated that only about 10% of all cases become fatal; many benign tumours also arise. The latency period for fatal cancers appears, however, to be very long (even up to 40 years or more), so that the current data may still be incomplete.

541. *Skin.* Satisfactory summary risk estimates for skin cancer incidence do not exist. The BEIR III estimates (see [C4], Table A-32) of between 0.44 and 1.02 cases per 10^4 PYGy, based on scalp and thymus irradiation, are not consistent with the chest fluoroscopy data. In uranium miners, Sevc [S51] has estimated one excess case of basal cell carcinoma of the skin per 10^4 PYSv. Information is not yet available from Japan.

542. *Lung.* Other than the Japanese and spondylitis patients, the best data on lung cancer are derived from individuals who inhale alpha-emitting radionuclides. Most data come from males, except in Japan, and the occurrence of this cancer is seriously affected by smoking interactions; there is as yet no consensus on whether the effects of smoking are more additive in nature or more multiplicative. Thomas and McNeill's [T11, T20] summary of these risks is provided in Table 10. These absolute risk coefficients are in units of million person-years per working level month (WLM); as discussed earlier, an approximate factor for converting from cases per 10^6 PY WLM to cases per 10^4 PYGy is 1.67 (with 1 WLM corresponding to 6 mGy absorbed dose in the bronchial tree). The absolute estimates range between 5 and 50 cases per 10^4 PYGy. As noted in chapter III, even when they are based on the same data, the estimates do not always agree, and they must be treated as uncertain. Most estimates of relative risk from brief external exposures to doses of less than 10 Gy are 1.2-2.0. Full treatments of risks to the lung will be found in [C20, I11].

543. *Digestive system.* The estimates presented from Japan and the spondylitis patients (see Table 56) constitute the best information available on most digestive system cancers. As previously discussed, the data from the spondylitis patients are not reliable in regard to colo-rectal cancer because of their high spontaneous rates of colo-rectal disease, and the results from studies of pelvic irradiation to treat gynaecological disorders are inconsistent and appear

to be affected by cell sterilization and other biological or environmental effects. Most liver cancer data come from internal emitters, particularly the Thorotrast patients. The Japanese and the spondylitis patients show uncertain results, and neither the studies by themselves nor their joint analysis has found an excess sufficient to derive useful risk coefficients (the BEIR III best estimate is given in Table 4). The liver is a site of frequent metastasis, and risk estimates may confound primary and secondary hepatic cancers.

544. *Salivary glands.* Detailed risk coefficients for salivary gland cancers, based on results of many studies, are given in Table 42. These were derived by Land [L11] in a summary analysis of this site. Land estimated the best overall absolute risk coefficient for this site to be 0.26 ± 0.06 cases per 10^4 PYGy. A recent estimate from Japan, based on T65 dosimetry, is 0.056 ± 0.036 per 10^4 PYGy [O4, T15].

545. As has been noted, the inter-study spread of values observed for single tissues is large, sometimes very large, presumably owing to the differences mentioned in paragraph 527. There is no fully satisfactory way to make suitable allowance for these differences in generating a combined estimate. Thus, there are only two options: either to combine the data without regard to the important differences enumerated above, a step that does not appear defensible; or to select the best possible set of estimates from among the various studies. Therefore, the Committee compares in the section to follow the data from the atomic bomb survivors, the ankylosing spondylitis series and the series of patients irradiated for cervical cancer.

B. SUMMARY OF RISK PROJECTION METHODS AND RISK ESTIMATES

546. The projections by the Committee will consider the induction of leukaemia and other cancers separately, drawing from the atomic bomb survivors and the ankylosing spondylitis and cervical cancer patients. Estimates are computed at 1 Gy of high dose rate exposure based on a linear dose-response model in the case of solid cancer. Data on leukaemia in the spondylitis and cervical cancer series take account of cell killing. The Committee has adjusted for this fact in the estimate it used to project the lifetime risk among the spondylitis patients, but could not do so in the case of the cervical cancer series. Separate estimates are computed from an additive projection model and a multiplicative projection model, using the life-table methods and minimum latency periods described below. These methods are similar in concept to those used by the BEIR III Committee [C4] and by the Nuclear Regulatory Commission of the United States [G11], although the details differ, largely to accommodate new data. It has generally been presumed that the additive and multiplicative models encompass the range of reasonable projections; however, Muirhead and Darby [M36, M37] have questioned whether this is true. As was seen in chapter II, they contend that it is difficult, given current data, statistical methods and biological theory, to choose between the additive and multiplicative models, or to determine some intermediate ones.

547. In addition to excess cases per 1,000 persons exposed to 1 Gy, estimates will be provided of lost life expectancy in person-years per 1,000 persons.

1. The basic projection model

548. The lifetime risk coefficients estimated in this chapter have been computed using an interactive, parametric demographic projection model developed by the Centre d'Etude sur l'Evaluation de la Protection dans le Domaine Nucléaire (CEPN, France) in 1985. It employs classical double-decrement life-table techniques and is not dependent on the data nor assumptions used in the present calculations. The model is sufficiently general to permit a wide range of choice of demographic, epidemiological and biological data or assumptions. Several kinds of computations can be made, including: (a) the effect of a single exposure on a cohort of a given age and sex, and (b) the effect of a single exposure to a given population of mixed ages and sexes.

2. Analytical expression of the model

549. The analytical formulation for calculating lifetime risk is the following: at age a and for a dose D, the absolute excess mortality rate V(a,D) is considered. If an exposure at age a_0 is assumed, the corresponding lifetime risk $U(a_0,D)$ is

$$U(a_0,D) = \int_{a_0}^{100} V(a,D)[N(a,D)/N(a_0)]da$$

where $N(a,D)/N(a_0)$ is the probability of survival to age a for an individual alive at age a_0, taking into account the risk of mortality both from radiation-induced cancer, and from all other causes.

550. To compute this lifetime risk, the studies on irradiated populations provide the following risk coefficients: (a) the absolute excess mortality rate, I(D), and (b) the excess relative risk per Gy, that is K(D).

551. The following two expressions for V(a,D) are associated with the additive (absolute) and multiplicative (relative) projection models, respectively:

$$V(a,D) = I(D)$$

$$V(a,D) = K(D)C(a)$$

where C(a) is the baseline cancer mortality rate in the population for the sites under consideration.

552. Thus, the lifetime risk estimates can be expressed as follows:

Additive risk projection model

$$U_a(a_0,D) = I(D) \int_{a_0+L}^{a_0+L+P} [N(a,D)/N(a_0)]da$$

Multiplicative risk projection model

$$U_m(a_0,D) = K(D) \int_{a_0+L}^{a_0+L+P} C(a)[N(a,D)/N(a_0)]da$$

where L is the minimum latency time and P the plateau period (i.e., the period of time following exposure during which manifestation occurs, and over which the risk is presumed to be constant). It should be noted that this method of estimating lifetime risks is essentially the same as that employed in the BEIR III Report [C4] and in the NUREG Report [G1]. An alternative approach would be to calculate separately the total number of cancers occurring in a lifetime in an exposed and a non-exposed population, and to take as the excess number of cancers the difference between these totals. The latter method would result in a smaller number of excess cases, for it excludes from the excess that fraction of cases which would have developed cancer for non-radiation related reasons at a later date.

3. Calculation of the loss of life expectancy

553. Using the same notation as in paragraph 549, the life expectancy at age a_0 (i.e., the average survival time for individuals alive at age a_0) is given by

$$\int_{a_0}^{100} [N(a,D)]/[N(a_0)]da$$

If this is computed both assuming no radiation exposure and assuming exposure at dose D, the difference between the two quantities is the loss of life expectancy due to exposure.

4. Demographic and background epidemiological data required

554. The characteristics that must be known for the population under study are the following: age and sex structure, overall mortality rate and cancer mortality rate by site. Since the model uses a year as the time-scale of interest, annual values are obtained by linear interpolation from the published information (which is generally presented in age intervals of 5 or 10 years). The survival rates are computed from current mortality rates and the projections assume that these will not change in the future.

5. The computational process

555. The principle of the model is to compute, by a discrete time analog of the basic demographic equation, for every year, i, the numbers of alternative outcomes possible for survivors through the previous year, $i - 1$, (that is, the numbers of fatal cancers at each site under observation, of deaths related to all other causes and of survivors to the next year). The first value is calculated and serves to increment the cumulated number of cancer cases already computed for the previous years and then to modify the baseline life-table. This calculation is limited to the assumed period of expression of excess cancer risk after the exposure. This period depends on the site or tissue under consideration: the minimum latency period has been taken to be 2 years for leukaemia and 10 years for all solid cancers. The plateau durations are assumed to be 40 years and lifetime for all cancers except leukaemia and 40 years for leukaemia. The number of survivors at age $i + 1$, $N(i + 1)$, is equal to the number of survivors at age i, $N(i)$, minus the number of those who die from baseline mortality, minus the number of those who die from radiation exposure.

6. Reference population

556. The reference populations considered here as the bases for the lifetime projections are the current general Japanese population for the atomic bomb survivors, the current adult male population of the United Kingdom for the spondylitis patients and the current adult female population in the United Kingdom for the cervical cancer series. The first two populations were selected since the studies were carried out in these two countries. The adult female population of the United Kingdom has been assumed to be representative of the other populations among which the cervical cancer study was conducted. The validity of extrapolation to other populations will be considered later.

7. Risk coefficients

557. The excess risk coefficients for the atomic bomb survivors are those based on the DS86 subcohort, as given in Table 54. These coefficients were derived by the authors [S48, S49] on a linear relative risk model, using organ absorbed doses from the explosions, and are restricted to mortality. The RBE of neutrons was assumed to be 1 in their estimation procedure. The coefficients represent mean values for both cities, both sexes (except for the breast and ovary), and all ages at the time of the bombings combined. The sites of cancer that have been selected for risk projection are those for which a statistically significantly increased mortality with increasing dose has been shown; namely, the bladder, breast, colon, leukaemia, multiple myeloma, oesophagus, ovary and stomach. Thyroid, lung and bone will be discussed later. For the spondylitis and cervical cancer series, the risk coefficients are given in Table 56. It is important to note, first, that in all three instances the risk coefficients that are used have been obtained from published reports and do not take into account the underreporting of cancer deaths on death certificates. BEIR III [C4], in its projections, increased these coefficients by 23% to take account of underreporting. A comparable action here would increase the Committee's projections of excess lifetime mortality by 20-25%. Second, and specifically with respect to the risk coefficients derived from the atomic bomb survivors, there is a levelling off, or a plateauing of the risk at shielded kerma of approximately 4 Gy and higher, and thus a linear relative risk model fitted to the full array of observed doses may underestimate the risk at doses below 4 Gy (approximately 3 Gy in organ absorbed dose). When the risk is estimated based on shielded kerma of less than 4 Gy, the excess deaths per 10^4 PYGy are approximately 5% higher for leukaemia,

and 15% higher for all cancers except leukaemia [S48].

558. Two kinds of coefficients are used as input, as in Table 54; these vary with the type of model chosen for the lifetime projection: the excess relative risk per Gy is used for the multiplicative projection model (constant relative risk) and the excess mortality per 10^4 PYGy is used for the additive projection model (constant absolute risk). In all cases, both the multiplicative and the additive projection models have been used, for comparative purposes (even though, as discussed earlier, for some sites one model appears to be more realistic than the other). Use of the two models in this context is not meant to imply any description of causative biological processes; the models are simply used to derive lifetime risk projections.

8. Indexes of harm

559. The indexes of harm have been restricted to different expressions of the effects of excess mortality associated with radiation-induced cancers in a lifetime after exposure. Two indexes are presented. The first is the lifetime excess number of fatal cancers, and the second is the loss of life expectancy in a population of 1,000 persons exposed at various ages to a single dose of 1 Gy of low-LET radiation at a high dose rate to each tissue.

560. Although the values of the indexes are calculated by the Committee at 1 Gy, values may be computed at other doses, provided the shape of the basic dose-risk relationship (linear, linear-quadratic, etc.) is known.

9. Treatment of uncertainty

561. The risk coefficients given in Tables 54 and 56 are accompanied by their 90% confidence intervals, when available. The upper and lower 90% statistical confidence intervals of these coefficients have been used to calculate the uncertainty inherent in the indexes of harm. It must be emphasized that this does not encompass the total uncertainty associated with the projections but only the statistical one attributable to the risk coefficients used as inputs. Uncertainties on dose and on demographic variables are not considered explicitly.

10. Fractionated and low-dose-rate exposure

562. The risk coefficients derived from the Life Span Study and given in Table 54 relate to instantaneous exposures to moderate to high doses and in principle represent only such exposure conditions. As shown in Table 55, irradiation of the cervical cancer patients was protracted over a few days or weeks, and that of the spondylitis patients was fractionated over a few weeks. For low-dose rates, an appropriate correction factor should be used if the indexes of harm are to reflect the experience coming from such epidemiological and experimental conditions.

C. RESULTS OF PROJECTIONS

563. The projections that follow should be prefaced by some statements about the approximations inherent in the model adopted. First, the Committee's lifetime projections are based on a simple modelling procedure, and deliberately so. More complex models could have been used and more effort to adjust for the known shortcomings in the data could have been made, but with each such *ad hoc* adjustment the results would have become progressively more particular and less and less applicable to the broad community of countries to which the Committee's deliberations are directed. As an illustration, the risk coefficients the Committee has employed are based on deaths reported to be due to the presence of a malignancy. Death certificates, however, are known to underreport the deaths that actually are attributable to cancer. An adjustment could have been made to account for this underreporting, but the Committee has not done so, for the degree of underreporting will undoubtedly vary from country to country.

564. The Committee has used other simplifications. Among these are age-constant risk coefficients (absolute or relative) that do not change with time following exposure (after the minimal latency period) or with age at exposure, as well as stable age-specific rates of mortality ascribable to cancer and other causes. The Committee has also ignored possible differences in mean survival time after the diagnosis of a malignancy as a consequence of different medical standards in different countries and their evolution with time. Again, while the model used could accommodate other assumptions, the data are still too sparse or contradictory to provide alternatives confidently. For example, the observations on the patients with ankylosing spondylitis suggest that the relative risk coefficient for all cancers other than leukaemia declines with time after exposure [D21], but this has not been seen, at least as yet, among the atomic bomb survivors of Hiroshima and Nagasaki [S49], except for those exposed as children. If the relative risk does in fact decline, then the Committee's projections will overestimate the lifetime risk. Similarly, if mean survival time of cancer cases increased, the loss of life expectancy would decline, although the total number of cancers would not change much (some diminution would be expected, however, as a result of the increased mortality from competing causes).

565. The coefficients assumed for the computations have been derived by linear regression analysis in the case of the atomic bomb survivors, the ankylosing spondylitis patients, and the cervical cancer series with cell-killing correction for leukaemia in the latter case. The model used by the Committee to project lifetime risks of cancer mortality or life span shortening at 1 Gy of low-LET irradiation administered at a high dose rate does not require selection of any given function of the dose-response relationship and therefore the Committee has not imposed any pre-selected function on the original data in order to derive its two projections.

566. For reasons of convenience, the Committee will consider separately the projections for the adult

population (males and females over 25 years of age), where comparisons among the three studies cited in paragraph 528 are possible; and the young population (below 25 years of age), for which the only reliable risk estimates are those that come from the atomic bomb survivors.

1. The adult population

567. The Committee's projections were carried out according to the assumptions given in Table 57. To allow a comparison of the results of the three studies cited in paragraph 528, the basic assumptions need to be adapted somewhat. First, the atomic bomb survivors study comprises individuals of both sexes and all ages, which the other two studies do not (see Table 55). Consequently, for a meaningful comparison, it has been necessary to examine only the adult population in the Japanese series. This means that the basic risk coefficients shown in Table 56 had to be modified to take into account the subtraction of the young cohorts. It is these modified figures for the absolute excess death per 10^4 PYGy, shown in Table 58, that have been used in the computations that will follow. Of necessity, since the requisite risk coefficients have not been published for the adult population only (nor for the working population, defined as aged 25-64), the Committee has been obliged to use excess risk coefficients based on averaging the sex-specific risks within the age groups 20-29, 30-39 and above 40, and weighting these averages by the proportion of the population within each of the age groups. To estimate lifetime risks in the working population, the Committee has used the risk coefficients derived from all ages and both sexes. Figures of the ankylosing spondylitis and cervical cancer series, which were not corrected, are also shown in Table 58. Second, the cervical cancer series refers only to females, so a female population (that of the United Kingdom) has been taken as the reference; similarly, the spondylitis cohorts are mostly males and therefore the male population of the United Kingdom has been adopted as a referent. Thus, the results of the extrapolations from the Japanese and cervical cancer series should be compared only in the female population, and the results from Japan and the spondylitis series should be compared only in the male population. Third, for cancers other than leukaemia, a comparison is only possible among two of the series, because no appropriate risk coefficient can be derived from the cervical cancer series. The spread of doses between the heavily irradiated pelvic organs (where cell sterilization could be important) and the organs in the upper part of the body (which received very low doses) is so large that risk coefficients based on averaged absorbed doses would have no meaning. These reservations also apply, though to a lesser extent, to the spondylitis series; however, since the authors [L16] provided an average whole-body dose (in addition to excess relative risk [D21]), the computations were made using these summary figures.

(a) Excess lifetime mortality: leukaemia

568. Table 59 shows the results of the Committee's projections, based on the model and assumptions described, in respect to excess lifetime mortality for leukaemia using the risk coefficients for each of the three major series (Table 58). Under the multiplicative risk projection model, the risk estimates range from 2.8 to 8.1 for females and 9.0 to 14 for males, and under the additive risk projection model from 1.4 to 7.0 for females and 4.4 to 13 for males (excess cases per 1,000 persons at 1 Gy of high dose rate low-LET exposure). Even considering the problems with the risk coefficients in these series, discussed earlier, these values are all well within an order of magnitude of each other.

(b) Excess lifetime mortality: all cancers other than leukaemia

569. Only two of the three general series the Committee has cited give usable estimates of the excess risk of cancers other than leukaemia, namely, the atomic bomb survivors and the patients with ankylosing spondylitis. For the assumption of a lifetime plateau (lower half of Table 59), the estimates of these two series are within a factor of about 2 to 4, the figures being lower for the series on the ankylosing spondylitis patients as compared to the Japanese atomic bomb survivors. It is tempting to speculate that this difference in risk relates to the modality of irradiation being instantaneous for the atomic bomb survivors and fractionated over a few weeks for the ankylosing spondylitis patients. Although this phenomenon is suggested by the data and by no means demonstrated, it is in the same direction that would be in agreement with a large body of radiobiological literature, reviewed most recently in the UNSCEAR 1986 Report and in reference [N1]. This shows that dilution in time of the dose yields generally lower effects than for the same dose delivered at high dose rate and/or without fractionation.

570. When the mortality from leukaemia is combined with that from all other cancers, assuming a plateau of 40 years for leukaemia and a lifetime plateau for all other malignancies (after the minimum latency), between 46 and 56 additional cancers would be expected in a population of 1,000 adults (500 males and 500 females), based on the Japanese risk coefficients, under the additive and multiplicative projection models, respectively.

(c) Loss of life expectancy: leukaemia

571. Table 60 shows the results of the projections in regard to loss of life expectancy attributable to the additional cases of leukaemia. The results are quite similar to those for excess lifetime mortality; namely, all three series provide estimates in generally good agreement. The spondylitis patients and the Japanese-based estimates under the multiplicative model are in fact very similar. Even the greatest discrepancies, between the cervical cancer and the other series, are within a factor of about 5.

(d) Loss of life expectancy: all cancers other than leukaemia

572. Taking the figures in the lower half of Table 60 as the most conservative ones, the projections derived from atomic bomb survivors are two to about four

times as great as those based on the ankylosing spondylitis patients. Life lost calculated by the additive risk projection model is higher than that calculated by the multiplicative risk projection model, but not by much.

573. As Table 60 shows, the expected number of person-years of life lost from all cancers would be about 840 for the additive model and about 620 for the multiplicative model for irradiation of 1,000 adults (500 males and 500 females), based on the Japanese risk coefficients, under the conditions described in Table 57.

574. It warrants reiteration that the absolute values given so far for irradiation of an adult (over 25 years) population apply to 1,000 persons of both sexes when a constant risk coefficient is used.

2. The population of children as a part of the population

575. The epidemiological evidence accumulated to date strongly suggests that the initial relative risk of subsequent malignancy following exposure to ionizing radiation is appreciably higher when exposure occurs early in life (within the first two decades after birth). From what is so far known about the biological aspects of cancer induction, this finding is not unexpected. However, apart from the data on the youngest age at the time of the bombings cohorts in the Japanese Life Span Study, there are few data from which specific risk coefficients can be derived, and even among the Japanese survivors the coefficients available are based on small case numbers and have relatively large sampling errors. These cohorts, furthermore, are those with the largest expected numbers of years still to live, and it is far from clear whether the high excess relative risks presently seen will persist. It is true that the Japanese data suggest a declining risk, both for leukaemia and all cancers except leukaemia, notably among survivors exposed before the age of 10 (the trends seen in Tables 51 and 52 for the 0-9 age group are statistically significant). These cohorts have only recently entered those years of life when the background rates for virtually all solid tumours, as well as for the chronic forms of leukaemia, increase markedly. Thus, it will be several decades before their cancer experience as middle- and older-aged individuals is clear. This poses a dilemma for the projection of lifetime risks. On the one hand, it would be unwise to assume that the risks will decline, for if they did not, the indices of harm could be grossly underestimated. On the other hand, to assume that these excess relative risks will persist throughout life, if in fact they do not, will project a harm that is much too high for these cohorts. It is largely for these reasons that the Committee has elected to examine the childhood population separately.

576. There are two separate aspects of the irradiation of young cohorts: first, their apparently greater susceptibility to the carcinogenic effect of radiation (this aspect can be studied by a discussion of the declining risk coefficient as a function of age) and second, their longer life expectancies relative to adults and the correspondingly longer time during which the consequences of exposure may be expressed.

577. The first aspect has already been considered in Tables 51 and 52. These Tables present excess relative risks and excess deaths for the atomic bomb survivors (which is the only series for which such estimates, although preliminary, are so far available) as a function of age at exposure and age at time of death, separately for leukaemia and all other cancers. The limited experience does not warrant analysing this phenomenon site-specifically.

578. The second aspect, as it applies to the population of Japan, is illustrated in Table 61. Since the risk coefficient has been presumed to remain constant over all ages, the impact of the demographic component introduced by the younger cohorts may be perceived from this Table.

579. The main difficulties in assessing the impact of exposure on the young arise when one attempts to evaluate the interaction between these two aspects for the purpose of calculating an overall measure of risk for the whole population, considering each age class separately. In fact, each age class will be characterized by its own coefficient of risk and its own demographic future. Since it appears from Tables 51 and 52 that the excess relative risk does not systematically change for ages above 20, and certainly not for ages above 30 (at least for solid cancer), the Committee has not attempted to calculate the whole extent of these changes. It should be pointed out that the observed values at the younger ages, based as they are on a relatively small number of cases, have large and unequal sampling errors. Previous attempts to take age-related coefficients into account have relied solely upon a statistical smoothing of the observed values. The Committee believes, however, that the observed changes in susceptibility as a function of age are not related to time alone, but also to the biological stages in development that are unique to certain ages, such as puberty and its associated hormonal changes.

580. Under these circumstances the Committee decided to make two separate sets of projections, using the multiplicative and the additive projection models: (a) the lifetime excess mortality and loss of life expectancy as it applies to a population for which the same risk coefficient is taken for all ages and (b) the lifetime excess mortality and loss of life expectancy as it applies to a population at ages 0-9 and 10-19, with coefficients specific for these age groups. These latter coefficients are as follows:

	Age at irradiation	
	0-9	*10-19*
Leukaemia		
Excess relative risk	19.1	4.5
Excess deaths	3.42	1.52
Other malignancies		
Excess relative risk	1.56	0.96
Excess death	2.77	6.16

These values have been taken from [S49] (Appendix Tables 5a and 5b), averaging over the two sexes.

581. For the multiplicative model and for excess lifetime mortality, the difference in the final effect introduced by using a measure of risk related to the specific age at exposure rather than a constant risk, that is a risk averaged over all ages and exposure, can be substantial. The computations show that one obtains three to four times more deaths due to leukaemia and all other cancers for the ages 0-9 at the time of exposure by using the age-specific risk. For the ages 10-19 at the time of exposure this difference in risk tends to reduce to 1-2. In both cases this difference will be expected to decline further as the average age in the cohorts increases and the coefficient of risk adopted approaches the coefficient for the whole population. This phenomenon is repeated in the projections for loss of life expectancy.

582. For the additive, rather than the multiplicative, model, the difference between the excess mortalities calculated using the constant coefficient and the age-related coefficient is very small for leukaemia, and it even tends to reverse for the age cohort 10-19. This accords with the fact that the risk coefficient for this particular age group happens to be lower than the average value adopted for computations on the whole population. As is true for the multiplicative model, these effects are almost repeated in the projections for loss of life expectancy.

583. To provide estimates of risk to be applied to the whole population, computations have been made using the various age-at-exposure classes, attributing to each of them the coefficient that applies to that particular class, projecting the risk of that class over the appropriate period of time (40 years or lifetime) and summing up the overall effects over all age-at-exposure classes. This was done for both the multiplicative and the additive projection models and for excess lifetime mortality and loss of life expectancy, separately. The final results of these summations are given in Table 62.

584. Considering the number of fatalities from leukaemia and other malignancies together (upper part of Table 62), it is seen that between about 42 and about 107 deaths would be predicted by the additive and the multiplicative model, respectively. The lower half of the Table shows that between about 950 and 1,370 person-years can be expected to be lost if the whole population is exposed to 1 Gy under the conditions specified.

585. There is a different way of arriving at similar projections; namely, to use a single risk coefficient which does not take age at exposure into account explicitly. It should be noted that this method of projection has its own shortcomings, for a risk coefficient so estimated is essentially a weighted average of the age-specific relative risks with weights proportional to the numbers of cancer deaths in the specific age groups. Thus most of the weight will be given to the older age groups whose actual relative risks are smaller. Nevertheless, to provide a comparison, this has been done in Table 63 in respect to the population of Japan. The values in parentheses provide the corresponding numbers when the upper and lower 90% bounds of the risk estimate are used (see Table 54). It shows that, under the conditions specified above, one would expect to observe a total of about 71 extra fatal cases under the assumption of a multiplicative projection, compared with about 45 cases for an additive projection model.

586. The first method (Table 62) may overestimate the lifetime excess mortality under the multiplicative model as the excess relative risk in the younger age-at-exposure groups has been falling with increasing time since exposure (see Table 51). On the other hand, this method may well underestimate the lifetime excess mortality using the additive model, as the excess risks have been increasing with time since exposure in the younger age-at-exposure groups (see Table 52). Conversely, under the second method (Table 63) the multiplicative model may underestimate the lifetime harm for the younger age-at-exposure groups, but the harm for these groups under the additive model may be overestimated. With the multiplicative risk projection model there is about a 30% decrease in estimated lifetime mortality from all malignancies using the second method compared with the first, while with the additive model the second method leads to about a 50% increase.

3. Extrapolation to other populations

587. Risk coefficients are always estimated, and lifetime risks projected, in the context of a particular population. Each exposed population from which risk coefficients are estimated has its own background mortality rates from all causes of death, and its own age- and sex-specific cancer rates. Indeed, not all individuals within any given population are at the same risk. Considering this, it is fair to ask what use can be made of risk coefficients obtained from one population for predicting lifetime risks in any other population. The projections given in this chapter have been derived by applying the closest possible expected rates of death (from cancer and from all other causes); namely, those from the same country as the exposed. Even so, baseline risks in the Japanese exposed to the atomic bombs, a wartime and post-war environment, were not the same as those of current Japanese, nor even precisely of those of Hiroshima and Nagasaki today. It is known that there are changes occurring in Japan in regard to baseline mortality rates and that there are also regional differences in Japan, as in every other country. Similarly, the all-United Kingdom mortality patterns are certainly not exactly those of the ankylosing spondylitis or the cervical cancer patients.

588. Because baseline mortality is always changing in every population, and this includes major changes in a variety of cancer risk factors, it is difficult to know how accurate lifetime risk projections might be. One way to place outer limits on this error would be to compare the lifetime risk estimates based on the same risk coefficients and different baseline mortality patterns. For this Annex three populations have been chosen to compare projected risks. To do this, the absolute and excess relative risk coefficients derived

from the experiences of the atomic bomb survivors have been applied to (a) the Japanese population using the Japanese 1980 national mortality patterns, representing the nearest available representative rates for those coefficients; (b) the United Kingdom, representing a rather typical older industrialized nation; and (c) Puerto Rico, representing (as best as worldwide data will permit) a population with high infant and infectious disease mortality, and low cancer rates.

589. The populations are compared in Table 64, and the results of this comparison for leukaemia, other cancers and all malignancies are given in Tables 65 and 66. The latter Tables show that across these three populations there is virtually no difference in risks projected by the additive model. Even for the multiplicative model, the maximum difference, using Japan as the basis of comparison, is a factor of 71/58 = 1.2. This clearly shows that the lifetime risk projections are very insensitive to differences in overall, and cancer-specific mortality differences within the range of contemporary large national populations, and for leukaemia or all cancers pooled. Thus, the risk projections derived here would seem to have rather broad generality and applicability. Much larger proportional differences may apply to site-specific cancer with large international variation in risk, such as female breast, stomach, large bowel and lung.

590. It should be pointed out, however, that this conclusion applies to only one of the uncertainties in the extrapolation of risk projections to other populations. It is not yet known how much the risk coefficients themselves might vary between different ethnic groups or populations with differing exposures to other carcinogens which could act synergistically; the range of today's knowledge is limited essentially to data from Japan and from a variety of industrialized populations. Within this context, and given the statistical problems in estimation, the range of risk coefficients is rather small, well below a full order of magnitude.

4. Comparisons with previous studies

591. The Committee made its last previous estimates of lifetime risk in the UNSCEAR 1977 Report [U2]. This Report gave values for all cancers of 1.0 $10^{-2}\,Gy^{-1}$ (range 0.75-1.75, Annex G, paragraph 318) for low dose low-LET radiation and 2.5 $10^{-2}\,Gy^{-1}$ (Annex G, paragraph 317) for high dose low-LET radiation. Leukaemia was about one fifth of the total. The projection was carried out by assessing the leukaemia risk and projecting a ratio of 5-7 for all cancers to leukaemia ultimately, thus obtaining the total cancer risk.

592. Additional epidemiological and other information has accumulated since 1977. This includes extensions and changes in the data for the Japanese atomic bomb survivors, in the ankylosing spondylitis series in the United Kingdom and in various other specific tumour sites such as lung (radon), thyroid and breast. A new study of patients surviving treatment for carcinoma of the cervix has provided additional information on second cancers at selected sites. Most of these studies make some contribution to quantitative risk estimates.

593. The atomic bomb survivors are especially important and provide the largest single data set over a range of doses. In this population the data have now accumulated over three additional time periods since the 1977 Report was written, viz. 1975-1978, 1979-1982 and 1983-1985. These are important time periods for the expression of solid tumours, 30-40 years after exposure to the bombs. Not only has the total amount of data on excess cancers increased by approximately threefold, but the extension of the data in time and the increasing information for all age cohorts, especially the young, provide further tests of models and thus aid in methods of projection and in knowledge of age dependence. Furthermore, the dosimetry of the survivors has been evaluated (and measured in the survivor range by thermoluminescent methods) and tends to increase risk by factors, when expressed in terms of shielded kerma, between 1 and 2 depending on the cancer site. Some improvements have also been made in statistical methods.

594. The atomic bomb survivors have been used in this report as the main source of risk estimates, while the Committee notes that other sources of data such as the ankylosing spondylitis patients are in general terms consistent with these estimates, especially when the mode of delivery of the exposure is taken into account. The Committee has not itself made primary estimates of risk in the Japanese atomic bomb survivors, but has relied on risk estimates developed in recent publications for the appropriate period of observation. These risk estimates have then been projected to a lifetime separately by the additive and multiplicative models and for both an age-structured population and for risks averaged over one intermediate age range. Lifetime risks have been estimated separately for an adult population alone and an entire population of all ages.

595. In this Annex the risk estimates for a population of all ages for mortality from all cancers at 1 Gy of high dose rate low-LET radiation range from 4 to 11 $10^{-2}\,Gy^{-1}$ (Table 62), whereas for an adult population alone the range is from 5 to 6 $10^{-2}\,Gy^{-1}$ (Table 59) (the ranges reflecting the additive and multiplicative models of projection, respectively). Leukaemia accounts for one quarter to one tenth of the total. The Committee has also provided estimates of the years of life lost as determined by the two projection methods. It may also be noted that while the age dependence has become more evident than in the UNSCEAR 1977 Report, sex differences have become smaller.

596. General appraisals of risk estimates have been made by various other groups since the UNSCEAR 1977 Report. The BEIR III Committee of the National Academy of Sciences in the United States produced a comprehensive report in 1980 [C4] which provided a range of from 0.1 to 5 $10^{-2}\,Gy^{-1}$ for all cancers using additive and multiplicative models for projection and quadratic, linear-quadratic and linear models for dose response. The preferred values of risk at the time the

report was issued were based on the linear-quadratic model and on additive projection and were quite similar to the values in the UNSCEAR 1977 Report (see [C4], Table V-25).

597. Later, for a report of the Nuclear Regulatory Commission in the United States, Gilbert [G11] developed risk estimates based on a linear-quadratic dose-response model (together with upper and lower bounds, roughly a factor of 3 above and below) and both additive and multiplicative projection models and years of life lost (Tables 8 and 9). The lifetime risk estimates ranged from about 0.3 to 6 10^{-2} Gy^{-1} with a central value of about 2 10^{-2} Gy^{-1} for low doses of low-LET radiation.

598. A group constituted by the National Institutes of Health in the United States assembled risk information for the purpose of developing tables of probability of causation (i.e., risk estimates for specific cancer sites at nominal ages as a function of time after exposure [U3]). The input used risk information updated from the BEIR III report similar to that of Gilbert. Lifetime risk estimates can be derived from the basic input using an additive projection model and again values of about 2 10^{-2} Gy^{-1} for low doses would be found.

599. These two recent groups had access to data from the Japanese atomic bomb survivors up to 1978 but were too early to obtain the full benefit of recent risk estimates from Japan utilizing the additional time periods and revised dosimetry now available to this Committee and included in this analysis.

D. RISKS AT LOW DOSES AND LOW DOSE RATES

600. At doses and dose rates defined by the Committee as low (less than 0.2 Gy and less than 0.05 mGy/min for low-LET radiation) radiation-related carcinogenic effects in an exposed population will almost always be masked by the larger carcinogenic effects of other factors. Moreover, in an exposed study population there will always be some level of dose below which no statistically significant excess of cancer occurs compared with the control population. In the dose range below this point, the excess cancer risk cannot be observed and cannot therefore be directly determined. In this dose range the Committee has to use a model to interpolate between the certainly zero excess risk at zero dose and the observed excess risk at doses of the order of 1 Gy. This may require the use of a correction factor if the projections based on high doses and high dose rates are to be applicable to exposures to low doses and low dose rates.

601. In the risk estimates derived above, no correction was made for the possible reduction of effects under conditions of low dose or low dose rate. The experimental literature contains a wealth of data showing that there are such effects. This has been recently reviewed by UNSCEAR [U1] and earlier by the National Council on Radiation Protection [N1]. The former report concludes that for low-LET radiation most dose-response curves for tumours induced in animals are concave upward and may be fitted by linear-quadratic or quadratic models, although in some cases linearity may apply. Moreover, dose rate studies with low-LET radiation almost invariably show a decreased incidence of tumours with decreasing dose rate in animal populations.

602. The human data on this subject are sparse, but are reviewed in the UNSCEAR 1986 Report [U1] which concludes that extrapolation linearly down to zero dose would overestimate the risk by a factor up to 5 in typical situations. The study by Howe [H6] and the very recent study by Holm [H28] are not considered in the UNSCEAR 1986 Report but are discussed earlier in the present Annex.

603. Since 1986 new data on human populations relevant to the effects of low doses have emerged from the revision of the experience in atomic bomb survivors [S49]. Table 67 shows the excess relative risk per 1 Gy of organ absorbed dose for doses above and below 0.5 Gy, using the entire 0-6 Gy dose range, and for progressively lower dose ranges below 1 Gy [S49]. Considering first leukaemia, a significant difference in the excess relative risk exists among survivors exposed to 0.5 Gy or more, as opposed to those exposed to lower doses (5.53 versus 2.44, respectively). This suggests persistence of a curvilinear dose-effect relationship for haematopoietic malignancies. In so far as all cancers except leukaemia are concerned, the excess relative risk associated with the higher doses does not differ significantly from that at the lower doses (0.41 versus 0.37, respectively). At doses below 0.20 Gy, the Japanese data have not revealed a significant excess of malignant tumours, and the nature of the dose-response relationship at these doses is uncertain. The expected numbers of additional cancer deaths at these lower doses are still small, relative to the background rate, even under the linear dose-response model, and the scatter of the data points is such that they can be fitted almost equally well by a quadratic, linear-quadratic or linear dose-response relationship [S48].

604. Epidemiologic studies of continuous internal irradiation of the thyroid gland by ^{131}I represent one source of information on the effect of low dose rates in human populations. A preliminary study of 10,000 patients who received doses to the thyroid gland in the range of 0.5 to 1.5 Gy found no excess of thyroid cancer after a mean follow-up of 17 years although 16 excess cases would have been expected based on external low-LET irradiation of the thyroid [H27]. An analysis of this study by the National Council on Radiation Protection of the United States concluded that ^{131}I should be considered no more than one-third as effective as external irradiation at high dose rates probably due to factors related to dose rate and dose distribution. An expanded study of 35,000 patients receiving diagnostic examinations has been published [H28]. These studies are discussed in paragraph 398. Both conclude that doses received from internal ^{131}I irradiation are less carcinogenic than similar doses from external acute irradiation. A factor of at least 3 has been proposed [N5], and possibly even 4 [H28]. Although the reduction of dose rate is held by some to

be a major contributor to the evident reduction in effectiveness, others contend that non-uniformity of dose distribution in the gland from ^{131}I may occur and contribute too [N5].

605. Epidemiologic studies of highly fractionated exposures to external low-LET radiation represent a second source of information on a low dose or low dose rate factor. As discussed in paragraph 367, there appears to be a non-linear dose response in the Canadian study of breast cancer following multiple fluoroscopic examinations [H6]. This appears to be related to the much smaller dose per examination received by the breasts of women who were irradiated posteriorly rather than anteriorly. A fractionation effect was not demonstrated in the similar but much smaller Massachusetts study [B3]. However, in this study it was not possible to distinguish a low dose cohort irradiated posteriorly. There appears to be a low dose or low dose rate factor of at least 3 in the Canadian study [H6].

606. Previous attempts to estimate lifetime risk for humans, such as BEIR III [C4], the Nuclear Regulatory Commission [G11], and the National Institutes of Health [U3] have handled the problem posed by low doses and low dose rates differently. BEIR III used a linear-quadratic dose-response function as one of their tested models, but the BEIR III Committee felt unable to recommend a specific general reduction for low dose rates. The other reports [G11, U3] both relied on the NCRP summary of the experimental literature [N1]. This was done by using a quadratic term in the dose-response function only for modelling exposure at low-dose rates. In the central estimates by Gilbert [G11], for which the results are given in Table 8, the linear coefficient was reduced by a factor of 3.3, and in the lower bound estimates by a factor of 10, for all organs except breast and thyroid.

607. From examination of both experimental and human data the Committee concludes that the carcinogenic effects of low-LET radiation are generally smaller at low doses and at low dose rates compared with those at high doses and dose rates. The reduction factors will vary with dose and dose rate and with organ system but will generally fall within the range 2 to 10.

E. LIFETIME RISK ESTIMATES FOR SPECIAL TISSUES

1. Lung

608. The computations provided in this Annex, based on DS86 risk coefficients, include low-LET, high-dose-rate exposure to the lung, based on the atomic bomb survivor experience. However, it is important to also estimate risk coefficients and lifetime risks for the alpha-irradiation experienced in connection with radon daughter exposure in the home and work-place. Thomas et al. [T20], ICRP [111], and BEIR IV [C20] have reviewed the literature on radon (and related) exposures and have estimated lifetime risk. The Committee has reviewed these findings, along with other recent reports, in chapters III and IV.

609. Life-table methods essentially identical to those used in this Annex have been used by ICRP [111] to derive lifetime risk estimates for continuous exposure to radon progeny, the high-LET exposure. Since the Committee believes that those estimates are reasonable in the light of the available data, it simply presents them in Table 68.

610. Thomas et al. [T20] have provided risk estimates for two types of exposure: (a) occupational exposure at 4 WLM up to a maximum of 200 WLM and (b) lifetime exposure of 0.02 WL 17 hours per day, 7 days per week as an upper tolerable limit for exposure that might be experienced in homes. The risk figures were adjusted for active breathing (occupational exposure) and quiescent breathing (home exposure), but they were not adjusted for age to account for varying susceptibility. The authors suggest, however, that since recent dosimetric studies indicate that breathing rate corrections may be inappropriate, their lifetime natural risk estimates might be doubled. Thomas et al. [T20] used eight risk models; namely, all combinations of additive and multiplicative projections; constant and age-varying risk coefficients; exposure times over which exposure is effective in incrementing risk. The projections were based on the Canadian life-table and lung cancer risks. Table 10 provides their risk coefficients and the lifetime excess risks.

611. The ICRP used similar assumptions and essentially the same demographic method of projection [111]. Their publication reviews the entire literature, biological and physical, on radon daughter exposure, including the available studies on mining, in-home exposures and the relationship of risk to smoking, age, sex, and latency period. Their results are given in Table 68. For details and the methods of deriving the risk coefficients see the ICRP study [111]. The ICRP concludes that the multiplicative risk projection model gives a better "best" fit for the data and provides a more realistic way of extrapolating from the higher mining doses to lower in-home doses than the additive model. It cites several published estimates, ranging from 0.10% to 1.0% excess cases at a constant annual exposure of 0.19 WLM.

612. Risk estimates for adult male uranium miners have been reviewed in [C20, 111, 112, U2]. More recent publications and papers prepared for publication have been noted by the Secretariat. Some of these data suggest that the minimum latency period to initial appearance of excess lung cancers after first exposure to high concentrations of radon progeny is five years, rather than the 10 years previously assumed [K28, M40, S51]. The interaction between cigarette smoking and exposure to radon progeny was closer to additive than to multiplicative for the Czech miners [S51]. These data have not yet been analysed in depth by the Committee. The preliminary analysis available does not suggest any reason for a major change in the previous risk estimates [112, U2] of 1.5-4.5 10^{-4} fatal lung cancers per WLM. More detailed consideration of epidemiological data relating to lifetime risk estimates for cancer induction by inhalation of radon and radon progeny is anticipated in future UNSCEAR Reports.

2. Bone

613. The Committee is unable to provide reasonable lifetime risk estimates for exposure to low- or high-LET irradiation for bone cancer. The data from Japan do not provide statistically meaningful risk coefficients, and there are problems with using the available literature on adults (e.g., the radium dial painters and patients injected with radium isotopes) to assess lifetime risk with demographic projection methods. The literature is summarized in chapter III.

614. While exposed children are probably sensitive to bone cancer induction, the genetically atypical nature of the available cohorts, relative to whole-body exposure, precludes a useful estimation of risk from their data, other than as discussed in chapter III.

3. Thyroid

615. The best estimates of thyroid cancer are available from [N5], and were given in Table 39. This was based on the most recent data yet available. There are no published data from Japan from which to make projections beyond those already given in this Table. Recent data from Holm [H28] have been noted earlier.

F. RISK ASSESSMENT BY CANCER TYPE

616. In section VII.C, the Committee calculates the projected risk of induction of malignancies for two broad classes of malignancy: leukaemia and other solid cancers. The reason for considering only these two classes is that in the patient series reported there are not enough observations to allow separate computations for all cancer sites and all ages in order to obtain an overall estimate. The only series in which there appears to be enough information for at least an exploratory site-specific analysis is that on the atomic bomb survivors from Hiroshima and Nagasaki. Therefore, simply to show what type of data the model used could generate—given the appropriate background information—the Committee has refined its analysis of the Japanese series to compute the risk of radiation-induced cancers by anatomical site, on the basis of multiplicative and additive risk projection models and for the two indices of radiation harm described earlier. These computations were performed using the risk coefficients and the assumptions specified in Tables 56 and 57; the results are given in Tables 69 and 70.

617. It should be borne in mind that the final results of the computations are no better than the original data from which they were derived. Although the atomic bomb survivor study is the only study that allows these projections, the number of cancers observed for each site and each age class is often small. Consequently, the projected values carry large uncertainties. It should also be pointed out that these computations apply strictly to the Japanese population and could not be transferred easily to other populations having different demographic and epidemiological characteristics.

618. The Tables are computed on the assumption of an age-constant risk coefficient, since there was not enough information for most sites (particularly in young cohorts) to allow meaningful analysis of the radiosusceptibility of young cohorts exposed below 20 years of age separately for each site. Taking this factor into account would, of course, increase the risk attributable to younger ages and, since these ages dominate the overall risk projection, they would increase the expected risk substantially. The Committee considers that taking an average risk coefficient overestimates to some extent the risk of the age classes that are old at the time of exposure and underestimates the risk of the younger cohorts. The data available at present do not allow quantification of this statement on a site-by-site basis.

619. Table 69 presents the expected additional cancer cases at nine specific sites, including the marrow, and for all other sites collectively, designated as the remainder, under the two risk projection models. It should be noted that the number of excess cancers at sites not specifically identified, i.e., the remainder, has been computed, first, through subtraction of the excess cancers at the identified sites from the total projected excess number at all sites and, second, through the actual computation of the risk coefficient associated with this collection of sites, and projecting the excess number from the estimated risk coefficient. The difference between these two methods of estimating the expected number is small. Table 70 summarizes the loss of life expectancy per person after exposure to 1 Gy, under the same assumptions as in Table 69.

G. SUMMARY AND CONCLUSIONS

620. The Committee had available to it certain additional data that made it desirable to reconsider the assessment of the risk of radiation-induced cancer. These additional data were the result of: (a) a re-evaluation of the doses of the Japanese atomic bomb survivors; (b) an extension of the observation periods for several cohorts during which radiation-induced cancers continued to occur; (c) the availability of data from several new cohorts; and (d) the introduction into the analysis of both the additive and the multiplicative risk projection models for lifetime cancers and loss of life expectancy, taking into account competing causes of mortality.

621. In its projections, summarized in Table 71, some of the risk coefficients used by the Committee were derived by the authors of the reports from which they were taken using a linear dose-response relationship. However, there is no direct epidemiological evidence that substantiates this at low doses and/or low dose rates, and there is in addition some epidemiological evidence of non-linearity. Based primarily on the experience of the atomic bomb survivors who received uniform whole-body irradiation at high doses and dose rates and low-LET, the Committee derived excess absolute and relative risk coefficients. Using these risk coefficients, the Committee estimated lifetime risks of mortality in the range of 4 to 11 $10^{-2}\,Gy^{-1}$. The Committee considered that these risk estimates apply to a dose range of 0.5-6 Gy and noted that they are

strongly influenced by the finding that children are considerably more sensitive to radiation effects than adults.

622. The above estimates are qualified by the facts that (a) the estimates have been derived using Japanese data and the extent to which they apply to other populations is not clear; (b) although the multiplicative model leads to higher estimates of projected mortality than the additive model, the projected estimates of the expected years of life lost are similar under the two models. This is because under the multiplicative model a large proportion of the projected deaths occur in very old people when the years of life lost are few; and (c) there are two other major cohorts, those of patients irradiated for ankylosing spondylitis and cervical cancer, which give rise to somewhat lower estimates of lifetime risk.

623. The Committee agreed that there was a need for a correction factor to modify the risks given above for low doses and low dose rates. The Committee considered that such a factor certainly varies widely with individual tumour type and with dose rate. However, the appropriate value to be applied to total risk for low dose and low dose rate should lie between 2 and 10. The Committee intends to study this matter in detail in the future.

624. The Committee has not presented risk estimates for high-LET radiation in general in this Annex except for the exposure to radon of uranium miners. For low doses of external high-LET radiation it would be necessary to multiply the risks for low-LET radiation by an appropriate quality factor. No dose or dose rate reduction factor is considered necessary for high-LET radiation at low doses.

Table 1

Population groups exposed to ionizing radiation used in risk evaluation studies

Populations	Study population size
Atomic bombings	
Residents of Hiroshima and Nagasaki	91,000
Nuclear weapons testing	
Military test observers	10,000
Populations near test sites	500,000
Marshall Islanders	250
Medical therapeutic exposures	
Ankylosing spondylitis patients	14,000
Cervical cancer patients	180,000
Patients receiving chest irradiation	10,000
Patients receiving Thorotrast injections	2,000
Patients receiving head and thymus irradiation	20,000
Hodgkin's disease patients	10,000
Patients irradiated for immunosuppression	
Hemangioma patients	
Childhood cancer patients b/	10,000
Fetus receiving pre-natal examination	1,000
Occupational exposure	
Nuclear shipyard workers	24,000
Reactor and processing plant personnel	30,000
Underground miners	22,000
Radium dial painters	4,000
Radiologists	10,000
Natural background	
Persons with elevated exposure due to geography	
Persons living in houses with high radon levels	
Nuclear accidents	
Individuals in public, workers, emergency crews	

Table 2

Epidemiological studies used to assess radiation carcinogenesis in man

- Cohort studies
  - Prospective follow-up
    - Survivors of atomic bombings
    - Marshall Islanders
  - Retrospective/prospective follow-up
    - Individuals identified from medical records
      - Patients irradiated therapeutically
      - Radiological workers and radiologists
    - Individuals identified through employment records
      - Occupationally exposed individuals
      - Military test observers
    - Individuals identified through geographic data
      - Populations in high natural background areas
      - Individuals living in local fallout areas
- Case-control studies
  - Retrospective ascertainment
    - Pre-natal exposures
- Opportunistic or ad hoc studies
  - Nuclear accident victims

Table 3

Mortality to incidence ratios for transforming risk estimates
[C4]

Site of cancer	Expectation of cancer (per cent)				Mortality/ incidence ratio	
	Males		Females			
	Mortality	Incidence	Mortality	Incidence	Males	Females
Oesophagus	0.4	0.4	0.2	0.2	1.00	1.00
Stomach	0.9	1.2	0.7	0.9	0.75	0.78
Intestine	2.3	4.4	2.8	5.1	0.52	0.55
Pancreas	1.0	1.1	0.9	1.0	0.91	0.90
Lung	4.9	5.9	1.2	1.6	0.83	0.75
Urinary	1.0	2.7	0.6	1.3	0.37	0.46
Lymphoma	1.1	1.5	0.9	1.2	0.73	0.75
Breast	0	0	3.0	7.7	-	0.39
Thyroid	0.03	0.17	0.09	0.46	0.18	0.20
Liver	0.20	0.20	0.18	0.18	1.00	1.00

Table 4

Excess cancer incidence (excluding leukaemia and bone cancer) per 10^4 person years and Gy, 11-30 years after exposure, estimated in the BEIR 1980 Report
[C4]

Site	Age at exposure (years)					Age-weighted average a/
	0-9	10-19	20-34	35-40	>50	
Males						
Thyroid	2.20	2.20	2.20	2.20	2.20	2.20
Lung	0.00	0.54	2.45	5.10	6.79	3.64
Oesophagus	0.07	0.07	0.13	0.21	0.56	0.26
Stomach	0.40	0.40	0.77	1.27	3.35	1.53
Intestine	0.26	0.26	0.52	0.84	2.23	1.02
Liver	0.70	0.70	0.70	0.70	0.70	0.70
Pancreas	0.24	0.24	0.45	0.75	1.97	0.90
Urinary	0.04	0.23	0.50	0.92	1.62	0.81
Lymphoma	0.27	0.27	0.27	0.27	0.27	0.27
Other	0.62	0.38	1.12	1.40	2.90	1.52
All sites	4.80	5.29	9.11	13.66	22.59	12.85
Females						
Thyroid	5.80	5.80	5.80	5.80	5.80	5.80
Breast	0.00	7.30	6.60	6.60	6.60	5.82
Lung	0.00	0.54	2.45	5.10	6.79	3.94
Oesophagus	0.07	0.07	0.13	0.21	0.56	0.28
Stomach	0.40	0.40	0.77	1.27	3.35	1.68
Intestine	0.26	0.26	0.52	0.84	2.23	1.12
Liver	0.70	0.70	0.70	0.70	0.70	0.70
Pancreas	0.24	0.24	0.45	0.75	1.97	0.99
Urinary	0.04	0.23	0.50	0.92	1.62	0.88
Lymphoma	0.27	0.27	0.27	0.27	0.27	0.27
Other	0.62	0.38	1.12	1.40	2.90	1.64
All sites	8.40	16.19	19.31	23.86	32.79	23.10

a/ Average of the age-specific coefficients, weighted according to the age distribution of the population of the United States.

Table 5

Excess mortality from all cancers per 10^4 persons exposed to low-LET radiation estimated in the BEIR 1980 Report [C4]

Dose-response model applied	Excess deaths: Absolute risk projection model	Excess deaths: Relative risk projection model
Single exposure to 0.1 Gy a/		
Linear-quadratic	7.66	22.55
Linear	16.71	50.14
Quadratic	0.95	2.76
Continuous exposure to 0.01 Gy per year b/		
Linear-quadratic	47.51	119.70
Linear	112.60	286.90

a/ Normal expectation of deaths from cancer in follow-up period: 1638.
b/ Normal expectation of deaths from cancer in follow-up period: 1673.

Table 6

Lifetime risk of cancer mortality from low-LET radiation estimated in the BEIR 1980 Report [C4]

Dose-response model applied	Excess deaths per 10^4 per Gy: Absolute risk projection model	Excess deaths per 10^4 per Gy: Relative risk projection model
Single exposure to 0.1 Gy		
Linear-quadratic	77	226
Linear	167	501
Quadratic	10	28
Continuous exposure to 0.01 Gy per year		
Linear-quadratic	67	169
Linear	158	403

Table 7

Ratios of excess deaths from radiation-induced cancers other than leukaemia and bone cancer to excess deaths from radiation-induced leukaemia and bone cancer
[C4]

Duration of exposure and dose rate	Dose-response model	Absolute risk projection model	Relative risk projection model
Lifetime			
(0.01 Gy/year)	Linear-quadratic	2.4	7.5
	Linear	2.6	8.1
Between ages 20-65			
(0.01 Gy/year)	Linear-quadratic	3.0	5.7
	Linear	3.3	6.2

Table 8

Models and assumptions for cancer risk estimates used in the United States Nuclear Regulatory Commission study
[G11]

Effect	Type of model	Latency (Years)	Plateau (Years)	Mortality risk coefficient: Absolute per 10^4 per Gy	Mortality risk coefficient: Relative per cent per Gy
Leukaemia	Absolute, linear-quadratic	2	25	2.2	n/a
Bone cancer	Absolute, linear-quadratic	2	25	0.1	n/a
Breast cancer	Relative, linear, non-age-specific	10	-	3.5, 2.3 a/	103, 42
Lung cancer	Relative, linear-quadratic	10	-	2.0	37
GI cancer b/	Relative, linear-quadratic	10	-	2.7	39
Thyroid cancer	Absolute, linear, age- and sex-specific age < 18	5	-	0.25, 0.125 a/	
Other cancers c/	Relative, linear-quadratic	10	-	1.5	20
	Absolute, linear	0	10	28	n/a

a/ First coefficient is for exposure under, second for exposure over age 20.
b/ Including cancers of the oesophagus, stomach, colon, rectum, pancreas, and other unspecified gastointestinal cancers.
c/ Including all cancers except leukaemia.

Table 9

Lifetime risks of cancer mortality from low-LET radiation at low dose rates (<0.05 Gy per day) estimated in the United States Nuclear Regulatory Commission study [G11]

	Number of deaths (10^4 per Gy)			Years of life lost (10^4 per Gy)		
	Lower bound	Central estimate	Upper bound	Lower bound	Central estimate	Upper bound
Leukaemia	5	14	48	168	505	1682
Bone	0.2	1	2	7	22	75
Breast	4	60	87	97	955	1452
Lung	5	20	138	100	288	1971
Gastrointestinal	9	57	189	222	661	2202
Thyroid	7	7	7	20	203	203
Other	5	29	96	124	378	1260
Leukaemia a/	1.2	1.2	3	80	80	200
Other a/	1.2	1.2	3	80	80	200

a/ Due to in utero exposure.

Table 10

Estimated risks of lung cancer in uranium and non-uranium miners [T11]

Location of mine	Type of mine	Mean dose (WLM)	Lung cancer deaths		Excess cases per 10^6 PY WLM a/	Relative risk per 10^2 WLM a/	Ref.
			Observed	Expected			
Canada							
Ontario	Uranium	36	62	25.54	9.59 (2.07)	4.97 (0.86)	[H17]
Ontario	Uranium	40-90	119	65.78	2-5	1.5-2.3	[M19]
Ontario	Gold		279	230.85	3-15	1.25-1.75	[M19]
Newfoundland	Fluorspar	204	65	3.76	17.82 (2.35)	3.30 (0.34)	[D14]
Czechoslovakia	Uranium	313	198	28.24	16.82 (1.40)	2.92 (0.16)	[S19]
Sweden							
Kiruna	Iron	110	13	4.47	2.72 (1.15)	2.74 (0.73)	[J3]
Zinkgruven	Lead,zinc	270	20	2.32	30.40 (7.69)	3.82 (0.71)	[A13]
Malmberget	Iron	170	14	2.95	5.53 (0.63)	3.21 (0.75)	[R14]
National survey		163	36	6.07	3.43 (0.69)	4.03 (0.61)	[S32]
United Kingdom	Iron	260	36	20.58		1.29 (0.11)	[B25]
United States	Metal	620	47	16.1	1.99 (0.44)	1.33 (0.07)	[W15]
Colorado plateau	Uranium	1180	159	25.24	3.52 (0.33)	1.45 (0.04)	[A12]

a/ Values in parentheses are standard errors.

Table 11

Goodness of fit of different lifetime projection models for all cancers other than leukaemia in the population of England and Wales [M37]

(Single exposure to 0.1 Gy with gamma and neutron components in the same proportion as at Hiroshima; The models that fit the data well are underlined.)

Covariate of risk	Minimum deviance a/	γ b/	Deviance a/ at γ = 0 (Multiplicative risk model)	Deviance a/ at γ = 1 (Additive risk model)
None (constant risk)	42.02	0.43	57.54	58.87
Sex	38.87	0.40	49.78	55.15
Age	34.34	0.01	34.34	53.92
Time since exposure	<u>26.06</u>	0.57	50.31	31.64
Sex, age	27.01	-0.19	<u>28.48</u>	50.73
Sex, time since exposure	<u>23.03</u>	0.55	42.23	28.90
Age, time since exposure	<u>24.11</u>	1.35	32.31	<u>24.34</u>
Sex, age, time since exposure	<u>22.49</u>	0.91	<u>24.94</u>	<u>22.51</u>

a/ For ease of presentation, a value of 400 has been subtracted from these deviances. In comparing the difference in deviance resulting from the inclusion of an extra variable in the model, a reduction in deviance of 3.84 is significant at the 5% level and a reduction of 6.63 is significant at the 1% level.

b/ γ denotes the value of the parameter that minimizes the deviance.

Table 12

Application of a generalized risk model to data on mortality from all cancers other than leukaemia in the Hiroshima atomic bomb survivors [M37]

(A 10-year minimal latent period is assumed.)

Covariates of risk	Risk model	Number of deaths per 10^4	Years of life lost per 10^4
Sex, age	Multiplicative	225	2940
	Additive a/	28	535
Time since exposure	γ = 0.57 b/	1460	14800
Sex, age, time since exposure	Multiplicative	983	10300
	Addivive	816	7350
Sex, age, log (time since exposure)	Multiplicative	104	1760
	Additive	87	1310

a/ This model does not fit the Hiroshima data well.

b/ γ denotes the value of the parameter that minimizes the deviance.

Table 13

Stage in carcinogenesis possibly affected by radiation in various organs as deduced from theoretical multi-stage models

Stage affected and tumour site	Population studied	Exposure type	Ref.
Early stage possibly affected			
All cancers except leukaemia	Atomic bomb survivors	Single	[D1]
All cancers except leukaemia	Ankylosing spondylitis patients	Brief chronic irradiation	[S2]
All heavily irradiated sites	Metropathic hemorrhagica patients	Brief chronic irradiation	[S3]
Breast	Atomic bomb survivors	Single	[D1]
Breast	Atomic bomb survivors	Single	[M5]
Breast	Chest fluoroscopy patients	Brief chronic irradiation	[B3]
Breast	Post-partum mastitis patients	Brief chronic irradiation	[S1]
Thyroid	Infant patients	Brief chronic irradiation	[H1]
Lung	Underground miners	Long chronic exposure to radon	[W12]
Late stage possibly affected			
Leukaemia	Ankylosing spondylitis patients	Brief chronic irradiation	[C2]
Leukaemia	Radiologists	Long chronic exposure to x rays	[S4]
All stages possibly affected			
Osteosarcoma	Radium dial painters	Long chronic exposure to radium-226	[M3]
Uncertain stages affected			
Leukaemia	Atomic bomb survivors	Single	[B4]

Table 14

Relative risk of leukaemia in pre-natally exposed children

Study location	Years of incidence	Relative risk	Ref.
UNSCEAR (weighted mean)	1940-1957	1.58 a/	[U1]
United States	1947-1960	1.52 a/	[M8]
United States			
White	1947-1967	2.88	[D3]
Black	1947-1967	0.00 b/	[D3]
United Kingdom	1943-1965	1.48 a/	[S5]
Finland	1959-1968	1.90	[S7]
Hiroshima-Nagasaki	1945-1979	2.15	[I1]
English twins	1943-1965	2.20 a/	[M7]
United States twins	1930-1969	1.60 c/	[H11]

a/ Significant at 0.05 level or better. UNSCEAR value is sample-size weighted value of studies done before 1964, including nine sets of data with a total of 1,626 cases contrasted with 2,706 controls. Relative risk from Japan was calculated from the dose-specific risk rate data in [I1] and is not significant (see text). Most cases and excess risk reported here occurred before the age of 10.

b/ No cases observed.

c/ In this series, relative risk of solid tumours was 3.2, not significant; other series have found values less than the RR for leukaemia.

Table 15

Second primary cancer in children treated for a primary cancer
[M28]

Type of second cancer	Number of second cancers			
	Total	Radio-therapy group	Chemo-therapy group	Other non-radio-therapy
Bone	67	52	8	7
Soft-tissue sarcomas	59	43	6	10
Haematopoietic	59	36	12	11
Skin	30	19	5	6
Brain	28	13	11	4
Thyroid	26	24	1	1
Breast	13	9	0	4
All others	24	12	6	6

Table 16

Development of second primary cancers in children
[T4]

Primary cancer	Number of children	Percentage with second cancer
Wilms' tumour	1248	2.2 a/
Hodgkin's disease	1036	2.5
Retinoblastoma	319	6.3
Neuroblastoma	790	2.4
Ewing's sarcoma	213	5.2 b/
Rhabdomyosarcoma	385	2.9
Brain (excluding medulloblastoma)	764	1.4
Medulloblastoma	285	2.4
Soft-tissue sarcoma	550	1.3
Non-Hodgkin's lymphoma	423	1.2
Acute lymphocytic leukaemia	1530	0.3
Osteosarcoma	271	1.5

a/ In other studies, occurrence of second cancers in Wilms' tumour patients is about 2.5% [L3, S10].

b/ Involved about 5 years of follow-up; in another study [S11] of 10 years of follow-up it has been estimated to be 35%.

Table 17

Cancer risk in children irradiated to treat tumours having a substantial heritable fraction

Disease	Site irradiated	Dose range (Gy)	Increased second cancer risk (%) 10 years	Increased second cancer risk (%) 30 years	Second cancer type	Ref.
Retinoblastoma	Head	>100 a/ 35-50 b/	2-3	9-14	80% osteosarcomas 20% other sarcomas	[A3,A8, D15,F2, S26,V1]
Ewing's sarcoma	Bones	54-65 b/	35		80% osteosarcomas	[S11]
Wilms' tumour	Abdomen	25-30 a/	1-2	15	Various carcinomas	[L3]

a/ Orthovoltage therapy.
b/ Megavoltage therapy.

Table 18

Occurrence of second non-ocular tumours in patients with retinoblastomas [A3]

Primary tumours and type of treatment	Number of cases	Number of second tumours	Number in treatment field	Number outside treatment field
Bilateral	693	89	58	31
Unilateral	18	5 a/	2	3
Radiation	688	89	62	27 b/
No radiation	23	5	1	4

a/ Includes three patients without family history of retinoblastoma.
b/ Includes one case with cobalt plaque who developed tumour in the humerus.

Table 19

Cumulative incidence of second primary cancers in patients with genetic retinoblastoma [D15]

Site of second cancer	Number of patients	Type of second cancer	Number of second cancers	Cumulative incidence (per cent) at 12 years	Cumulative incidence (per cent) at 18 years
All sites	384	All cancers	26	4.3	8.4
		Osteosarcomas	17	3.6	6.0
In radiation field	314	All cancers	14	3.4	6.6
		Osteosarcomas	8	2.4	3.7
Outside radiation field	384	All cancers	12	1.6	3.0
		Osteosarcomas	9	1.6	3.0

Table 20

Risk of thyroid and other cancer in children receiving head and neck irradiation

Site or cause of irradiation	Number in study	Dose (Gy)	Tumour type	Increased risk at 30 years	Excess risk per 10^4 PYGy	Reference
Tinea capitis	10842	0.09	Thyroid	0.15		[R1, S27]
		3.8	Leukaemia	0.13		
	2226	3-6	Basal cell	<1.0		
Thymus	2651	1-10	Thyroid	1.0	3.5	[S13, H1
			Leukaemia	<0.5	1.0	
Tonsils	2578	7.8	Thyroid	8.0	3.6	[L9]
Marshall Islanders	250	7-20	Thyroid	7.3	1.8	[C6,C10,L9]
Atomic bomb survivors		0-6	Thyroid		7.1	[U2]
Local fallout (USA)	2945	0.3-2.4	Thyroid	?		[C4]

Table 21

Dose-response relationships for thyroid cancer in children irradiated for enlarged thymus [S38]

	Control	Dose range (Gy)				
		0.01-0.49	0.50-1.99	2.00-3.99	4.00-5.99	>6.00
Person-years	118157	33449	6020	11456	6382	1727
Mean dose	0	0.17	1.2	2.5	4.5	7.5
Cancers	1	4	1	6	11	5
Rate (/10^5 PY)	0.7	12.3	18.8	51.0	128	154
Expected cases a/	1.43	0.31	0.07	0.13	0.08	0.03
Relative risk	0.7	12.9	13.6	45	130	196
Excess/10^4 PYGy	-	6.9	1.5	2.0	2.8	2.0

a/ Expected based on age and sex-specific thyroid cancer rates for New York state 1969-1971.

Table 22

Dose fractionation and risk in thymus-irradiated children [S38]

	Mean dose (Gy)	Person-years	Cancers	Expected cancers	Excess per 10^4 PYGy
Dose per fraction					
0.01-0.49 Gy	0.18	33268	4	1.0	6.1
0.50-1.99 Gy	2.2	8622	6	6.8	2.2
>2 Gy	3.1	14340	12	14.2	2.3
Number of fractions					
1	0.74	29414	7	5.1	2.9
2	1.5	22417	6	9.7	1.5
3	2.5	5445	9	7.3	3.8

Table 23

Risk of breast cancer in atomic bomb survivors under 10 years of age at the time of the bombings
[T7]

	Dose range (Gy) (T65DR)				
	0	0.01-0.09	0.10-0.49	0.50-0.99	>1.00
	Mean dose (Gy)				
	0	0.026	0.17	0.55	1.9
Observed	6	5	5	5	3
Expected	13.2	5.45	3.56	0.85	0.93
Relative risk	0.45	0.92	1.40	5.88	3.23

Table 24

Risks of leukaemia and other cancers in patients treated for Hodgkin's disease

Treatment	Relative risk a/					Risk rates b/			
	All cancers			Leukaemia		All cancers		Leukaemia	
	[B11]	[B11]	[G4]	[G4]	[B9]	[B11]	[C7]	[B11]	[C7]
X rays only	1.6	15.0	-	-	1.0 c/	0.0034	0.015	0.0010	0.000
Chemotherapy only	4.0	9.3	2.60	147	1.0 c/	0.0075	0.029	0.0040	0.062
Combined	14.5	20.0	4.27	118	261.0	0.0093	0.030	0.0070	0.064

a/ Relative risks are observed/expected.
b/ Risk rate data are cases/person-year except for the last, which is a cumulative (actuarial) risk after 7 years post-exposure.
c/ No second cancers were observed in these groups, so baseline value (relative risk) may be set to 1.0.

Table 25

Relative risk of second cancer following radiotherapy for benign gynaecologic disorders and for cervical cancer [W6, B12]

(Cancer patients followed for at least 10 years; other patients followed for a variable length of time, but generally more than 10 years. Risks significant at the 0.05 or better level are underlined.)

Organ dose level and site of second cancer	Relative risk		
	Radiotherapy treatment		No radiotherapy treatment
	Cancer patients	Non-cancer patients	
High doses a/			
Colon	1.1	1.1	1.3
Rectum	1.8	1.2	1.4
Uterine corpus	1.0	2.8	0.1
Ovary	0.9		0.5
Bladder	3.5	2.1	1.2
Other genital	3.2		2.8
Intermediate doses b/			
Stomach	1.0	0.8	0.6
Pancreas	1.2	0.5	0.9
Kidney	0.8	2.1	2.1
Oesophagus	1.1		0.0
Small bowel	2.4	1.3	5.0
Gallbladder	0.8	0.9	1.5
Low doses (remote sites) c/			
Lung	2.3	0.9	2.2
Breast	0.7	0.9	0.9
Thyroid	1.4		0.6
Oral cavity	1.7	1.9	1.7
Salivary gland	1.7		0.0
Brain	0.6		2.0
General systemic cancer of unknown original site			
Leukaemia	0.9	2.3	0.6
Multiple myeloma	1.4		0.0
Lymphoma	1.3	2.3	0.3
Hodgkin's disease	1.0		1.1

a/ For cancer patients around 10 Gy; for non-cancer patients 1-10 Gy.
b/ For cancer patients 1-10 Gy; for non-cancer patients comparatively smaller.
c/ For cancer patients around 0.1 Gy; for non-cancer patients comparatively smaller.

Table 26

Relative risk and excess cases of cancer following irradiation to treat cervical cancer
[838]

(90% confidence intervals in parentheses.)

Second cancers	Relative risk at 1 Gy a/	Excess cases per 10^4 PYGy
Colon	1.00 (0.00-1.02)	0.01 (-0.03- 0.18)
Cecum	1.02 (0.99-1.09)	-
Rectum	1.02 (1.00-1.04)	0.06 (0.00- 0.16)
All female genital	1.01 (1.00-1.02)	0.05 (-0.01- 0.17)
Ovary	1.01 (0.98-1.14)	0.05 (-0.03- 0.60)
Vagina	1.03 (1.00-1.08)	-
Other genital	0.98 (0.95-1.07)	-0.01 (-0.02- 0.03)
Bladder	1.07 (1.02-1.17)	0.12 (0.01- 0.30)
Connective tissue	0.95 (0.89-1.13)	-0.01 (-0.03- 0.03)
Stomach	1.69 (1.01-3.25)	3.16 (0.05-10.40)
Pancreas	1.00 (0.72-1.62)	0.00 (-0.65- 1.43)
Kidney	1.71 (1.03-3.24)	1.10 (0.06- 3.50)
Breast	1.03 (0.13-2.29)	0.54 (-14.6-21.7)
Thyroid	13.30 (0.00-77.0)	6.87 (-2.04-39.2)
Leukaemia		
CLL	1.00 (0.90-1.43)	0.00 (0.00- 0.17)
AL and CML	1.14 (1.00-1.45)	0.10 (0.00- 0.31)

a/ See original publication for details of the calculations.

Table 27

Relative risk of second cancers in patients treated for ovarian cancer
[811]

(Risks significant at the 0.05 or better level are underlined.)

Site of second cancer	Relative risk		
	Non-irradiated patients		Irradiated patients
	N = 6713 a/	N = 5455 b/	N = 6596 a/
All sites	1.1	1.5	2.1
Bladder	0.4	2.8	c/
Breast	1.0	1.1	2.2
Colon	1.3	1.9	2.8
Connective tissue	0.9	3.3	c/
Endometrium	2.9	4.5	5.9
Leukaemia	0.3	1.3	9.3
Lung	2.0	0.7	0.8
Lymphoma	1.6	2.7	3.6
Myeloma	2.8	0.0	c/
Rectum	1.1	0.3	1.1
Stomach	0.8	0.5	c/

a/ Study group from United States National Cancer Institute End Results Program.
b/ Study of a survey of 70 United States medical centers using chemotherapy.
c/ Less than 2 patients observed.

Table 28

Relative risk of second cancers in patients treated for breast cancer
[H20]

Site of second cancer	Relative risk	
	Irradiated patients	Non-irradiated patients
Kidney	1.9	0.7
Oesophagus	1.7	0.7
Non-Hodgkin's lymphoma	1.7	0.7
Chronic lymphocytic leukaemia	1.2	0.4
Acute non-lymphocytic leukaemia	2.5	1.2
Lung, 10-19 years after irradiation	2.3	1.5
Lung, 20-29 years after irradiation	4.8	1.8

Table 29

Relative risk of leukaemia following treatment of other primary cancers in adults
[C8]

(Sites not listed produced no cases of subsequent leukaemia and therefore the relative risk is not estimable (or is 0.0).
Risks significant at the 0.05 or better level are underlined.)

Site of primary cancer	Surgery only		Radiation only		Chemotherapy only	
	Number	Risk	Number	Risk	Number	Risk
Oral/buccal	5634	1.1	6663	1.3	768	0.0
Oesophagus	675	0.0	2464	0.0	503	0.0
Stomach	5279	0.0	617	5.0	2882	2.5
Colon	31109	0.9	798	0.0	5490	1.4
Rectum	13493	1.1	2286	1.5	2197	1.0
Larynx	1817	0.7	3636	1.2	122	0.0
Lung/bronchus	11527	0.7	22113	0.5	13215	0.9
Connective	1420	1.0	527	3.3	528	0.0
Melanoma	8078	1.3	142	0.0	455	0.0
Breast	39416	1.0	10854	1.7	6040	3.8
Endometrium	8855	0.4	10276	2.1	523	0.0
Ovary	2864	0.8	1283	10.0	4407	9.0
Prostate	22322	0.9	7035	1.4	539	0.0
Testis	883	0.0	1147	3.3	674	17.9
Bladder	14824	1.1	2878	1.2	855	1.4
Kidney/renal	5118	2.5	1056	2.0	870	0.0
Thyroid	4154	2.1	925	0.0	103	45.3
Multiple myeloma	39	25.0	332	5.0	3603	4.0
All other	8710	1.5	8896	1.1	20863	1.7
All sites	192754	1.0	93651	1.4	70674	2.3

Table 30

Relative risk of leukaemia in ankylosing spondylitis patients and in atomic bomb survivors
[C11]

Population	Relative risk	Excess cases per 10^4 PY
Ankylosing spondylitis patients	4.79 (3.4- 6.6)	1.96 (1.24-2.88)
Atomic bomb survivors	9.38 (7.0-12.6) a/	3.95 (3.04-4.86)

a/ Individuals exposed to >1 Gy compared to those exposed to <0.1 Gy (T65DR).

Table 31

Relative risk of leukaemia (incidence) in women treated for gynaecological disorders
[W6]

Dose (Gy)	Condition treated	Cases	Relative risk
Mean marrow dose a/			
0.4-1.3	Benign and malignant disorders	9	3.5
1.0-3.0	Benign and malignant disorders	3	1.2
3.0-15	Benign and malignant disorders	9	1.1
Total		21	1.5
Mean pelvic marrow dose			
1.6-3.2	Benign disorders	10 b/	2.8
1.6-5.0	Benign disorders	9	3.5
2.2-5.2	Metropathia haemorrhagica	6 b/	4.6
3.0-9.0	Benign disorders	3	1.2
9.0-45	Uterine malignancies	9	1.1

a/ Includes cases treated for benign as well as malignant disorders.
b/ Based on leukaemia deaths.

Table 32

Relative risk of multiple myeloma following various radiation exposures
[C10]

(Risks significant at the 0.01 or better level are underlined, the others are significant at the 0.05 level. 90% confidence intervals in parentheses.)

Group and exposure type	Number of cases	Relative risk
Uterine cancer	3	0.28 (0.1-0.7)
All cohorts receiving appreciable alpha-radiation	14	4.32 (2.6-6.8)
All other cohorts receiving gamma- or x-ray therapy or diagnostic exposure	13	2.05 (1.2-3.3)
All cohorts receiving only x rays	11	2.02 (1.1-3.3)
All cohorts except uterine cancer	50	2.25 (1.7-2.8)
All cohorts	53	1.61 (1.3-2.0)

Table 33

Risk coefficients for radiation-induced bone sarcomas estimated in the BEIR 1980 Report
[C4]

Type of radiation and model	Cumulative risk coefficient per 10^4 PGy	Risk rate coefficient per 10^4 PYGy
Alpha particles (high-LET)		
Linear	27	1
Dose-squared	3.7 a/	980 b/
Beta, gamma, and x rays (low-LET)		
Linear	1.4	0.05
Dose-squared	9200 a/	24000 b/

a/ Per 10^4 PGy^2.
b/ Per 10^4 $PYGy^2$.
Provisional coefficients for endosteal doses up to a few Gy. The risk-rate coefficient is determined by dividing the cumulative risk by 27 years, the total risk period, for alpha particle exposure.

Table 34

Relative risk of skin cancer in patients who received scalp irradiation to treat tinea capitis
[C4]

Age or time (years)	Number of skin cancers	Relative risk
Age at exposure		
1-19	0	0
20-24	0	0
25-29	4	2.9
30-34	8	4.5
35-39	12	5.2
40-44	5	3.1
>45	0	0
Time since exposure		
1- 9	0	0
10-14	0	0
15-19	1	0.9
20-24	7	3.8
25-29	14	4.7
30-34	9	4.8

Table 35

Relative risk of breast cancer by dose and age at exposure, Hiroshima and Nagasaki incidence data, 1950-1980
[T14]

Age at the time of bombing	Kerma (Gy) a/							
	0.0	0.01-0.09	0.10-0.49	0.50-0.99	1.00-1.99	2.00-2.99	3.00-3.99	>4.00
	Average tissue dose (Gy) a/							
	0	0.026	0.168	0.55	1.10	1.89	2.65	4.03
0- 9	1.0	2.01	3.10	13.03	8.98	0	25.4	0
10-19	1.0	0.91	1.73	2.27	3.45	5.80	4.86	12.7
20-29	1.0	0.90	1.42	1.58	2.57	2.80	4.02	5.95
30-39	1.0	1.09	0.68	0.93	2.96	4.30	2.21	7.61
40-49	1.0	0.81	1.35	0.25	0.84	2.43	2.06	0
>50	1.0	0.97	1.08	0.52	3.53	2.77	0	0
Total b/	1.0	1.0	1.3	1.4	2.7	4.5		

a/ Based on T65DR.
b/ Totals obtained from [T6].

Table 36

Relative risk of breast cancer in four different studies
(Modified from [H6], based on data from [B3, H6, L6, S1, T14])

Age at exposure	Atomic bomb survivors (T65DR)	New York mastitis	Massachusetts fluoroscopy	Canadian fluoroscopy
10-19	5.6	a/	4.8	4.6
20-29	2.8	2.2	1.5	4.0
30-39	4.0	1.6	1.4	1.7
40-49	0.6	5.2	2.0	0.8
>50	a/	a/	0.0	

Rates compare disease in women exposed to more than 1 Gy with cases expected in unexposed women.
a/ Insufficient data.

Table 37

Breast cancer incidence in women
[L6]

Study series	Age at exposure	Number of breast cancers	Relative risk at age					
			20-29	30-39	40-49	50-59	60-69	>70
Rochester mastitis patients	20-29	18		3.0	1.9	2.5		
	30-39	13			1.7	3.1	2.8	
Massachusetts fluoroscopy patients	10-19	15	9.0	4.1	3.1	3.1		
	20-39	24		1.1	1.7	1.2	2.1	
LSS patients, 1950-1974 (T65DR doses)	10-19	40 a/	8.8	4.9	3.1			
	20-29	31		2.0	1.9	3.4		
	30-39	19			0.3	2.4	1.2	
	40-49	12			2.2	0.8	1.0	0.7
	>50	11				0	1.8	1.6

a/ Exposure of 0.1 Gy or more.

Table 38

Breast cancer incidence in irradiated relative to unexposed women
[H6], based on [B3, H6, L6, S1])

Breast tissue dose (Gy)	Atomic bomb survivors a/	New York mastitis	Massachusetts fluoroscopy	Canadian fluoroscopy
0	1.0	1.0	1.0	1.0
0.01-0.99	1.2	0.7	1.2	1.1
1.00-1.99	3.3	1.9	2.0	2.0
2.00-2.99	2.3	1.8	2.3	3.5
3.00-3.99	3.0	3.0	1.5	3.0
>4.00	8.1	2.3	5.2	14.6

Data given are relative rates of breast cancer excluding the first five years of post-operation observation in all except the Canadian series, to exclude non-radiogenic cases.
a/ Based on T65DR.

Table 39

Estimate of excess thyroid cancer cases (incidence) per 10^4 individuals exposed to 0.06-15 Gy
[N5]

Source of irradiation	Exposed under age 18		Exposed over age 18	
	Male	Female	Male	Female
Annual excess				
Internal a/	0.28	0.56	0.56	1.12
External b/	0.84	1.68	1.68	3.36
Lifetime excess				
Internal a/	2.74	6.80	4.83	10.50
External b/	8.22	20.40	14.50	31.50

a/ Internal doses include exposure to iodine isotopes 125, 131.
b/ External doses include exposure to x or gamma radiation or to iodine isotopes 132, 133, 135.
Mortality can be assumed to be 1/10 the incidence values given in the Table. Assumes excess risk = 2.5 cases per 10^4 PYGy, based on data derived from North American children, both sexes pooled.

Table 40

Relative risk of cancer in heavily irradiated sites for combined data of ankylosing spondylitis patients and the Japanese Life Span Study
[D11]

(Risks significant at the 0.05 or better level are underlined. 90% confidence intervals in parentheses.)

Site of second cancer	Relative risk	Excess cases per 10^4 PY
Pharynx	1.76 (0.73-4.22)	0.02 (-0.10-0.14)
Oesophagus	1.82 (1.29-2.57)	0.45 (0.11-0.80)
Stomach	1.24 (1.09-1.41)	1.14 (0.31-1.96)
Pancreas	1.24 (0.87-1.76)	0.15 (-0.18-0.47)
Larynx	1.35 (0.70-2.59)	0.07 (-0.11-0.25)
Lung	1.54 (1.36-1.76)	2.30 (1.49-3.12)
Ovaries	2.39 (1.54-3.72)	0.93 (0.28-1.57)
Skin	0.61 (0.19-1.98)	-0.03 (-0.16-0.10)
Bones (excluding jaw and nose)	2.40 (1.08-5.34)	0.09 (-0.05-0.23)
Multiple myeloma	2.16 (1.11-4.20)	0.16 (-0.02-0.34)
Other lymphomas	1.58 (1.07-2.33)	0.20 (0.10-0.49)
CNS tumours (spinal cord and nerves)	9.31 (4.72-18.4)	0.23 (0.07-0.40)
Others	1.62 (1.28-2.03)	0.93 (0.41-1.46)
All heavily irradiated sites	1.46 (1.35-1.57)	6.56 (4.98-8.15)
Selected sites	1.41 (1.30-1.53)	4.99 (3.55-6.42)

Table 41

Sex differences in cancer mortality risks in atomic bomb survivors (T65DR doses)
[P15]

Site	Relative risk at 1 Gy exposure		P	Excess risk per 10^4 PYGy		P	Background mortality sex ratio (F/M)
	Male	Female	a/	Male	Female	a/	
Leukaemia	3.84	4.08	0.96	1.95	1.20	0.03	0.54
All cancers except leukaemia	1.11	1.25	0.007	3.29	4.42	0.3	0.57
Bladder, kidney	1.41	1.77	0.5	0.27	0.23	0.8	0.47
Colon	1.18	1.60	0.8	0.19	0.37	0.5	0.55
Oesophagus	1.09	2.23	0.03	0.14	0.22	0.8	0.12
Liver b/	1.31	1.46	0.8	0.11	0.06	0.6	0.33
Lung c/	1.19	1.67	0.03	0.78	0.92	0.95	0.32
Multiple myeloma	1.64	1.43	0.8	0.07	0.06	0.7	1.27
Stomach	1.07	1.19	0.15	0.90	1.07	0.8	0.46

a/ Significance is two-sided test of sex difference.
b/ Includes intra-hepatic bile ducts.
c/ Includes trachea and bronchial tree.

Table 42

Risk of salivary gland cancer incidence in medically irradiated populations
[L11]

Mean age at exposure	Number irradiated	Mean follow-up (years)	Mean dose (Gy)	Number of cancers	Excess cases per 10^4 PYGy a/	Ref.
0-15	1644	18.2	2.0	2	0.43±0.32	[S24]
0-1	2872	23.3	1.4	2 b/	0.40±0.28	[H1]
5.1	554	21.5	5.0	3	0.65±0.38	[M11]
5	466	27.7	2.4	1 b/	0.21±0.37	[J4]
5.5	1922	29.9	7.9	8	0.21±0.07	[S15]
				19 b/	0.48±0.12	
7.9	2215	20.5	0.4	1	0.67±0.75	[S16]
				3 b/	1.90±1.30	
>15	10902	16.8	0.4	4	0.72±0.40	[M13]
				3 b/	0.47±0.37	
57	1005	19.1	5.3	2	0.26±0.28	[H21]

a/ After a five-year latency.
b/ Benign cases.

Table 43

Effect of type of treatment on cumulative incidence of second primary cancers in patients with genetic retinoblastoma [D15]

Type of treatment	Site of second cancer	Number of patients	Number of second cancers	Cumulative incidence (per cent) at 12 years	Cumulative incidence (per cent) at 18 years
Radiotherapy	All sites	140	4	4.2	4.2
	In radiation field	140	3	2.9	2.9
	Outside radiation field	188	1	1.0	1.0
Radiotherapy with chemotherapy	All sites	62	8	6.6	14.2
	In radiation field	62	4	4.2	9.9
	Outside radiation field	65	5	4.6	7.5
Chemotherapy	All sites	3	1	100	100
No radiotherapy, no chemotherapy	All sites	48	0	0	0

Table 44

Comparison of predicted and observed excess second cancers in women irradiated to treat cervical cancer [B12]

Second primary cancers	Risk coefficient (cases per 10^4 PYGy) [C4]	Organ dose (Gy)	Predicted excess cancers	Observed excess cancers a/
Stomach	1.68	2.0 (0.5-3.5)	60	+3
Colon	1.12	5.0	100	+15
Liver	0.70	1.5 (0.5-2.5)	20	0
Pancreas	0.99	1.5 (0.3-3.0)	25	+9
Lung	3.94	0.3 (0.1-0.6)	25	+77
Breast	5.82	0.3 (0.1-0.6)	37	-101
Kidney	0.88	2.0 (0.6-3.5)	30	-6
Bladder	0.88	30	475	+82
Thyroid	5.80	0.1 (0.0-0.3)	15	+4
Lymphoma	0.27	~10 (3.0-13)	50	+15
Acute and NL leukaemia	2.70	7.5 (3.0-13) b/	1000	+13
		2.5 (0.8-3.3) c/	350	+13
		0.3 (0.1-0.4) d/	45	+13

a/ In women, except those with leukaemia, living more than 10 years; for women with leukaemia, values are for 1-20 years after irradiation.
b/ Averaged over entire bone marrow.
c/ Excluding pelvis contribution.
d/ Excluding pelvis, lumbar spine, and upper femur contributions.

Table 45

Relative risk of cancer mortality in ankylosing spondylitis patients
[021]

Site	Time since first treatment (years)							Total
	0-2.5	2.5-4.9	5-9.9	10-14.9	15-19.9	20-24.9	>25	
All neoplasms	1.77	1.53	1.48	1.61	1.51	1.15	1.08	1.33
Leukaemia	5.45	12.51	4.67	2.41	2.19	1.46	1.94	3.17
Colon cancer	2.40	0.54	2.41	1.38	1.87	0.46	1.02	1.30
All other a/	1.57	1.28	1.30	1.60	1.47	1.19	1.07	1.28

a/ All neoplasms other than leukaemia and cancer of the colon.

Table 46

Relative risk of cancer mortality at specific sites in ankylosing spondylitis patients
[021]

(Risks significant at the 0.05 or better level are underlined.
Relative risk computed as observed/expected ratio.)

Site	Time since first treatment (years)			Total >5 a/
	<5	5-25	>25	
Mouth	0.00	1.68	1.41	1.58
Pharynx	0.00	1.77	1.14	1.56
Oesophagus	0.84	2.05	2.41	2.20
Stomach	1.01	1.20	0.62	1.01
Rectum	0.94	1.14	0.96	1.07
Liver	2.71	0.58	2.01	1.10
Pancreas	3.24	1.13	0.86	1.02
Larynx	2.84	1.37	1.85	1.54
Lung	1.22	1.37	0.97	1.21
Breast	1.58	1.88	1.02	1.62
Uterus	0.00	1.15	0.65	1.02
Ovary	1.17	1.07	0.62	0.93
Prostate	3.04	1.24	1.07	1.16
Kidney	1.11	1.61	1.36	1.52
Bladder	1.96	0.91	1.62	1.20
Skin	0.00	1.23	1.52	1.33
Spinal cord	96.6	6.77	0.00	4.72
Other CNS	0.67	1.60	1.49	1.57
Bone	1.88	2.95	2.96	2.95
Hodgkin's disease	2.42	1.66	0.00	1.32
Other lymphoma	2.03	2.89	1.13	2.24
Multiple myeloma	0.00	1.52	1.97	1.72
Other	1.90	1.35	1.10	1.25
Total	1.44	1.38	1.07	1.26

a/ At least five years have elapsed since treatment.

Table 47

Relative risk of cancer mortality in age groups of ankylosing spondylitis patients
[D21]

Age at first treatment	Time since first treatments (years)					
	All but colon and leukaemia			Leukaemia		
	5-25	>25	Total >5 a/	5-25	>25	Total >1 b/
<25	1.97	1.06	1.38	2.98	0.00	1.10
25-34	1.50	0.98	1.22	4.98	2.21	3.06
35-44	1.37	1.10	1.26	6.66	1.44	3.02
45-54	1.35	1.25	1.33	3.90	3.34	3.57
>55	1.16	0.78	1.15	4.85	0.00	3.28
Total	1.38	1.07	1.26	5.01	1.87	3.03

a/ At least five years elapsed since treatment.
b/ At least one year elapsed since treatment.

Table 48

Organ dose estimates (Gy) for ankylosing spondylitis patients calculated using the Monte Carlo technique
[L16]

Organ	Mean of estimated doses	Median of estimated doses	Standard deviation	Range	10-90% range
Adrenals	7.3	6.6	5.8	0-38.1	0.08-14.4
Bladder	1.5	1.1	1.7	0-17.2	0.05- 3.3
Brain	0.14	0.11	0.20	0- 4.8	0- 0.3
Gastrointestinal tract					
Stomach	2.5	2.4	1.7	0-12.8	0.18- 4.5
Upper large intestine	3.0	3.1	1.8	0-11.6	0.43- 5.4
Lower large intestine	2.6	2.1	2.7	0-25.7	0.15- 5.4
Small intestine	4.3	4.4	2.6	0-16.1	0.59- 7.3
Oesophagus	4.2	4.3	3.2	0-27.2	0.05- 8.1
Genitalia					
Testes	0.23	0.08	0.60	0- 6.6	0- 0.5
Ovaries	3.8	3.3	3.7	0-21.0	0.02- 8.2
Other than above	0.24	0.09	0.74	0-13.0	0- 0.4
Heart	2.5	2.6	1.9	0-17.3	0.04- 4.8
Kidneys	4.6	4.3	3.6	0-30.8	0.24- 9.0
Liver	1.6	1.6	1.3	0-11.5	0.11- 3.0
Nasal region	0.47	0.44	0.44	0- 3.1	0- 1.1
Pancreas	3.5	3.5	2.4	0-17.0	0.15- 6.3
Pulmonary region					
Lungs	1.8	1.6	1.6	0-13.3	0.05- 3.5
Main bronchi	6.8	6.5	5.4	0-59.9	0.19-13.9
Trachea	3.6	3.6	3.0	0-14.3	0- 7.6
Skeleton					
Pelvis	9.4	10.0	6.1	0-35.3	0.48-16.1
Ribs	4.4	4.2	3.4	0-28.7	0.15- 8.3
Spine	14.4	14.5	9.7	0-65.9	1.2 -27.0
Other than above a/	0.48	0.36	0.61	0- 6.3	0.06- 0.9
Skin					
Trunk skin	1.8	1.7	1.1	0- 9.7	0.47- 3.0
Skin excluding trunk a/	0.19	0.17	0.20	0- 2.3	0.01- 0.4
Spleen	1.6	1.2	1.9	0-21.6	0.12- 2.7
Thyroid	0.99	0.97	0.89	0-10.5	0- 2.1
Uterus	3.5	3.1	3.5	0-18.1	0.04- 7.0
Totals					
Trunk	2.9	2.9	1.7	0-12.5	0.78- 5.0
Legs	0.08	0.03	0.22	0- 4.0	0- 0.1
Head	1.5	1.5	1.3	0- 7.6	0- 3.2
Total body	1.9	1.9	1.1	0- 8.1	0.51- 3.3

a/ Indirect calculation.

Table 49

Red bone marrow dose estimates (Gy) for ankylosing spondylitis patients calculated using the Monte Carlo technique
[L16]

Marrow site	Proportion of total red bone marrow mass (%)	Mean of estimated doses	Median of estimated doses	Standard deviation	Range	10-90% range
Arms	1.9	0.43	0.17	1.4	0-15.4	0-0.41
Legs	3.8	0.09	0.03	0.25	0- 4.2	0-0.15
Cranium	11.9	0.32	0.23	0.36	0- 4.0	0-0.78
Mandible	1.2	0.26	0.20	0.27	0- 1.8	0-0.66
Clavicles	1.6	0.41	0.31	0.64	0- 8.5	0-0.80
Scapulae	4.8	0.65	0.38	1.4	0-20.6	0.01-0.93
Ribs	10.2	1.9	1.9	1.4	0-11.3	0.05-3.6
Pelvis	36.2	4.3	4.6	2.8	0-16.7	0.16-7.7
Upper spine	3.4	4.7	5.2	4.0	0-20.8	0-9.9
Mid spine	14.1	6.9	7.1	5.0	0-37.6	0.06-13.1
Lower spine	10.9	7.8	8.1	5.0	0-31.3	0.35-13.8
Total marrow	100.0	3.8	3.7	2.2	0-13.1	0.89-6.7

Table 50

Comparison of the mean shielded kerma and organ absorbed dose (mGy) in atomic bomb survivors exposed to 0.01 Gy and over under the T65DR and DS86 dosimetries
[S48]

Organ dose	Dose system	Both cities				Hiroshima				Nagasaki			
		Number	Total	Gamma	Neutron	Number	Total	Gamma	Neutron	Number	Total	Gamma	Neutron
Shielded kerma	DS86	41719	295	287	8	31044	304	295	10	10675	267	265	3
	T65DR	41316	414	350	64	26146	442	344	99	15170	366	362	4
Bone marrow	DS86	40701	242	239	3	30569	247	243	3	10132	228	227	1
	T65DR	41316	219	201	18	26146	227	199	28	15170	204	203	1
Large intestine	DS86	39859	223	222	1	30083	226	225	2	9776	215	215	1
	T65DR	31958	198	186	12	21873	189	173	16	10085	217	216	1
Lung	DS86	40382	240	238	2	30469	244	242	3	9913	228	227	1
	T65DR	41316	194	180	14	26146	200	179	22	15170	182	181	1
Stomach	DS86	39961	228	226	2	30136	232	229	2	9825	218	217	1
	T65DR	41316	181	169	12	26146	187	169	18	15170	171	171	1
Female breast	DS86	25252	240	236	4	18803	248	243	5	6449	217	216	1
	T65DR	25211	309	276	33	15937	321	270	51	9274	288	286	2
Bladder	DS86	40060	231	229	1	30240	235	233	2	9820	220	219	1
	T65DR	41316	174	162	12	26146	180	162	18	15170	164	164	1
Ovary	DS86	24581	211	210	1	18439	215	214	1	6142	198	198	0
	T65DR	19563	190	181	9	13214	182	168	13	6349	208	207	1

Table 51

Relative risk at 1 Gy by age at the time of the bombings (ATB) and age at death based on DS86 shielded kerma
[S49]

(Risk before the assumed minimum latency period of 10 years indicated in parentheses.)

Age ATB	Age at time of death						
	0-20	20-29	30-39	40-49	50-59	60-69	>70
Leukaemia							
0-10	44.16	3.41	8.64	0.95			
10-19	54.74	-	2.45	1.02	0.82		
20-29		5.33	3.54	43.09	1.02	0.82	
30-39			0	24.05	10.58	1.47	3.89
40-49				0.83	3.82	0.82	3.10
>50					15.63	5.18	6.90
Total	46.47	9.81	4.75	5.68	3.98	1.70	4.40
All cancers except leukaemia							
0-10	(70.07)	5.89	1.96	1.86			
10-19	(40.90)	(0.82)	1.66	1.59	1.68		
20-29			(1.38)	2.09	1.74	1.37	
30-39			(0.84)	(1.12)	1.11	1.23	1.48
40-49				(1.25)	(1.12)	1.13	1.33
>50					(2.58)	(0.95)	1.15
Total	75.32	2.22	1.60	1.58	1.39	1.13	1.29
Stomach cancer							
0-10	(0)	7.22	1.30	1.54			
10-19	(0)	(0.82)	1.26	1.21	2.88		
20-29			(0.82)	2.66	1.93	1.77	
30-39			(76.88)	(1.00)	0.97	1.18	1.48
40-49				(1.60)	(1.17)	1.05	1.24
>50					(3.30)	(0.92)	1.12
Total	0	1.30	1.26	1.70	1.40	1.06	1.22
Lung cancer							
0-10	(0)	0.84	0.82	0.83			
10-19	(0)	(0)	0.81	5.56	1.50		
20-29			(0)	0.83	1.75	1.03	
30-39			(0)	(0.81)	1.49	1.50	1.26
40-49				(0)	(1.58)	1.34	1.40
>50					(0.85)	(2.29)	1.44
Total	0	0.84	0.82	2.32	1.57	1.44	1.39
Breast cancer							
0-10	(0)	0	0.92	3.04			
10-19	(0)	(0)	10.48	2.16	4.21		
20-29			(2.10)	0.81	2.05	5.78	
30-39			(0.83)	(0.80)	2.86	2.28	1.03
40-49				(0)	(0.82)	1.13	0.82
>50					(8.16)	(0.82)	1.37
Total	0	0	3.72	1.63	2.57	1.61	1.01

Table 52

Excess deaths per 10^4 PYGy by age at the time of the bombings (ATB) and age at death based on DS86 shielded kerma
[S49]

(Risk before the assumed minimum latency period of 10 years indicated in parentheses.)

Age ATB	Age at death						
	0-20	20-29	30-39	40-49	50-59	60-69	>70
Leukaemia							
0-10	6.71	0.93	1.27	-0.01			
10-19	3.95	-	0.56	0.02	-0.06		
20-29		3.93	1.52	4.84	0.01	-0.28	
30-39			0	3.18	2.26	1.09	3.89
40-49				-0.35	3.07	-0.24	3.50
>50					4.31	3.84	5.12
Total	6.48	2.17	1.16	1.88	1.54	1.09	4.24
All cancers except leukaemia							
0-10	(0.43)	1.32	2.85	5.16			
10-19	(3.96)	(-0.12)	2.00	5.84	13.91		
20-29			(1.39)	9.40	15.71	14.33	
30-39			(-1.32)	(1.33)	3.16	11.00	41.01
40-49				(2.48)	(3.37)	7.31	37.30
>50					(35.29)	(-2.88)	17.21
Total	0.79	0.54	1.98	5.35	9.62	6.85	30.53
Stomach cancer							
0-10	(0)	0.43	0.43	1.24			
10-19	(0)	(-0.06)	0.23	0.58	6.61		
20-29			(-0.29)	5.40	5.46	8.21	
30-39			(4.77)	(0.01)	-0.35	2.82	11.93
40-49				(2.62)	(2.24)	1.15	8.52
>50					(15.79)	(-2.34)	5.56
Total	0	0.06	0.31	2.10	3.41	1.19	8.20
Lung cancer							
0-10	(0)	-0.01	-0.02	0.10			
10-19	(0)	(-0)	0.03	1.75	1.15		
20-29			(0)	-0.06	1.71	0.19	
30-39			(0)	(0.10)	-1.10	3.82	3.11
40-49				(0)	(0.68)	2.19	7.26
>50					(-0.15)	(3.11)	4.74
Total	0	-0.00	-0.02	0.56	1.11	2.62	5.50
Breast cancer							
0-10	(0)	-0	-0.03	1.18			
10-19	(0)	(-0)	2.99	2.39	4.55		
20-29			(0.37)	-0.16	1.90	4.14	
30-39			(-0.16)	(-0.18)	-3.83	0.66	0.05
40-49				(0)	(-0.41)	0.20	-0.23
>50					(11.27)	(-0.12)	0.87
Total	0	0	1.09	0.76	2.88	0.61	0.02

Table 53

Comparison of DS86 and T65DR risk coefficients for mortality based on shielded kerma in the Japanese DS86 subcohort [S48]

(Both cities, both sexes and all ages ATB combined. 90% confidence intervals in parentheses.)

Site of cancer	Dose system a/	Excess relative risk per Gy	Ratio DS86/ T65DR	Excess deaths per 10^4 PYGy	Ratio DS86/ T65DR
All malignant neoplasms	DS86	0.39 (0.32-0.46)	1.39	10.0 (8.56-11.8)	1.43
	T65DR	0.28 (0.23-0.33)		7.00 (5.87-8.19)	
	T65DRf	0.25 (0.21-0.30)		6.21 (5.24-7.22)	
Leukaemia	DS86	3.92 (2.89-5.40)	1.37	2.29 (1.88-2.73)	1.43
	T65DR	2.87 (2.10-3.89)		1.60 (1.30-1.91)	
	T65DRf	2.62 (1.95-3.48)		1.40 (1.16-1.65)	
All except leukaemia	DS86	0.29 (0.23-0.36)	1.32	7.41 (5.23-9.08)	1.41
	T65DR	0.22 (0.17-0.27)		5.25 (4.17-6.37)	
	T65DRf	0.19 (0.15-0.23)		4.62 (3.70-5.58)	
Oesophagus	DS86	0.43 (0.09-0.91)	1.43	0.34 (0.08-0.67)	1.55
	T65DR	0.30 (0.06-0.65)		0.22 (0.05-0.45)	
	T65DRf	0.18 (-0.01-0.45)		0.14 (-0.01-0.32)	
Stomach	DS86	0.23 (0.13-0.34)	1.35	2.07 (1.19-3.05)	1.46
	T65DR	0.17 (0.09-0.25)		1.42 (0.83-2.07)	
	T65DRf	0.13 (0.07-0.20)		1.13 (0.63-1.67)	
Large intestine except rectum	DS86	0.56 (0.25-0.98)	1.37	0.56 (0.26-0.91)	1.47
	T65DR	0.41 (0.18-0.71)		0.38 (0.18-0.63)	
	T65DRf	0.41 (0.20-0.69)		0.37 (0.19-0.58)	
Trachea, bronchus and lung	DS86	0.46 (0.25-0.72)	1.39	1.25 (0.70-1.89)	1.44
	T65DR	0.33 (0.18-0.51)		0.87 (0.49-1.29)	
	T65DRf	0.30 (0.17-0.44)		0.83 (0.50-1.20)	
Female breast	DS86	1.00 (0.48-1.75)	1.41	1.02 (0.53-1.60)	1.46
	T65DR	0.71 (0.34-1.21)		0.70 (0.36-1.10)	
	T65DRf	0.83 (0.46-1.32)		0.80 (0.48-1.17)	
Ovary and other uterine adnexa	DS86	0.81 (0.16-1.89)	1.53	0.45 (0.10-0.90)	1.61
	T65DR	0.53 (0.07-1.24)		0.28 (0.04-0.58)	
	T65DRf	0.50 (0.09-1.10)		0.28 (0.06-0.55)	
Bladder, other unspecified urinary	DS86	1.02 (0.45-1.07)	1.44	0.55 (0.26-0.89)	1.53
	T65DR	0.71 (0.30-1.30)		0.36 (0.17-0.60)	
	T65DRf	0.61 (0.25-1.12)		0.29 (0.13-0.48)	
Multiple myeloma	DS86	1.86 (0.55-4.45)	1.43	0.21 (0.07-0.39)	1.31
	T65DR	1.30 (0.40-2.99)		0.16 (0.06-0.28)	
	T65DRf	0.70 (0.15-1.72)		0.10 (0.02-0.20)	

a/ T65DRf = T65DR-full, meaning that risk coefficients were calculated by using TR65DR dose on the full T65DR cohort.

Table 54

Comparison of DS86 and T65DR risk coefficients for mortality based on absorbed dose in the Japanese DS86 subcohort [548]

(Both cities, both sexes and all ages ATB combined. 90% confidence intervals in parentheses.)

Site of cancer	Dose system	Excess relative risk per Gy	Ratio DS86/ T65DR	Excess deaths per 10^4 PYGy	Ratio DS86/ T65DR
Leukaemia	DS86	5.21 (3.83-7.12)	0.90	2.94 (2.43-3.49)	0.95
	T65DR	5.76 (4.24-7.86)		3.11 (2.56-3.71)	
All except leukaemia	DS86	0.41 (0.32-0.51)	0.71	10.13 (7.96-12.44)	0.73
	T65DR	0.58 (0.46-0.72)		13.97 (11.11-17.04)	
Oesophagus	DS86	0.58 (0.13-1.24)	0.87	0.45 (0.10-0.88)	0.92
	T65DR	0.67 (0.12-1.47)		0.49 (0.09-1.00)	
Stomach	DS86	0.27 (0.14-0.43)	0.69	2.42 (1.26-3.72)	0.72
	T65DR	0.39 (0.23-0.58)		3.34 (1.95-4.83)	
Large intestine except rectum	DS86	0.85 (0.39-1.45)	0.82	0.81 (0.40-1.30)	0.83
	T65DR	1.04 (0.43-1.85)		0.98 (0.42-1.63)	
Lung	DS86	0.63 (0.35-0.97)	0.88	1.68 (0.97-2.49)	0.89
	T65DR	0.72 (0.41-1.11)		1.89 (1.10-2.79)	
Female breast	DS86	1.19 (0.56-2.09)	1.31	1.20 (0.61-1.91)	1.33
	T65DR	0.91 (0.43-1.57)		0.90 (0.46-1.42)	
Ovary	DS86	1.33 (0.37-2.86)	1.10	0.71 (0.22-1.32)	1.11
	T65DR	1.21 (0.11-3.06)		0.64 (0.06-1.43)	
Bladder	DS86	1.27 (0.53-2.37)	0.80	0.66 (0.31-1.12)	0.81
	T65DR	1.59 (0.63-3.03)		0.81 (0.34-1.38)	
Multiple myeloma	DS86	2.29 (0.67-5.31)	0.96	0.26 (0.09-0.47)	0.90
	T65DR	2.39 (0.75-5.56)		0.29 (0.11-0.53)	

Table 55

Comparison of the main characteristics of the atomic bomb, ankylosing spondylitis and cervical cancer series

	Atomic bomb survivors	Spondylitis series	Cervical cancer series
Nature of study	Prospective	Retrospective-prospective	Retrospective-prospective
Sample size	76000	14000	83000
Sex composition	F = 59%	F = 17%	F = 100%
Age at irradiation (years)	0->90	>15	<30->70
Average follow-up (years)	28.8	13.0	7.6
Type of control	Internal	National rates	National rates and internal
Type of dosimetry	Individual (DS86)	Individual for leukaemia, 1/15 random sample elsewhere	Mean dose of a sample
Type of irradiation	Instantaneous, whole-body	Fractionated, non-uniform, partial-body	Chronic, fractionated, partial-body
Dose distribution			
Mean dose (Gy)	0.24	1.9	Extremely uneven
Range of doses (Gy)	(0.01-6.0)	(0-8.06)	
Person-years at risk	2185000	184000	623800

Table 56

Summary of the estimated risk of cancer per 1 Gy of organ absorbed dose obtained from the atomic bomb, ankylosing spondylitis and cervical cancer series

EXCESS RELATIVE RISK

Organ or tissue	Atomic bomb survivors (Table 54) [S49]	Spondylitis series [S31] and [D21]	Cervical cancer series [B38]
Leukaemia	5.21 (3.83-7.12) a/	3.5 b/	0.88
All cancers except leukaemia	0.41 (0.32-0.51)	0.14 c/	d/
Bladder	1.27 (0.53-2.37)	0.19	0.07 (0.02-0.17)
Breast	1.19 (0.56-2.09)	-	0.03 (0.00-1.29)
Kidney	0.58 (-0.09-1.94) e/	0.12	0.71 (0.03-2.24)
Large intestine	0.85 (0.39-1.45)	-	0.00 (0.00-0.02)
Larynx	0.51 (-0.05-1.68) e/	0.15	
Lung	0.63 (0.35-0.97)	0.13	
Multiple myeloma	2.29 (0.67-5.31)	-	
Oesophagus	0.58 (0.13-1.24)	0.29	
Ovary	1.33 (0.37-2.86)	0.00	0.01 (0.00-0.14)
Rectum	0.00 e/	0.03	0.02 (0.00-0.04)
Stomach	0.27 (0.14-0.43)	0.004	0.69 (0.01-2.25)

ABSOLUTE RISK
(excess deaths per 10^4 PYGy)

Organ or tissue	Atomic bomb survivors (Table 54) [S49]	Spondylitis series [S31] and [D21]	Cervical cancer series [B38]
Leukaemia	2.94 (2.43- 3.49)	2.02	0.61
All cancers except leukaemia	10.13 (7.96-12.44)	4.67	d/

a/ Values in parentheses are 90% confidence intervals. They are those given by the authors.

b/ This figure was derived by the Committee from [S31] using data from individuals receiving a mean marrow dose of 3 Gy or less.

c/ All cancers except leukaemia and colon cancer.

d/ An estimate of the risk of all cancers except leukaemia cannot be made for this series. An estimate of the whole-body dose does not exist, and probably cannot be estimated given the nature of the exposures.

e/ Shielded kerma.

Table 57

Summary of assumptions used in the projections

Minimum latency	For leukaemia: 2 years For all other sites: 10 years
Plateau	For leukaemia: 40 years For all other sites: Lifetime
Extrapolation models	For leukaemia: Additive and multiplicative For all other sites: Additive and multiplicative
Detriment indicators	Lifetime excess mortality for cancer of each site Loss of life expectancy in person-years
Population exposed	1,000 persons
Exposure	1 Gy to each site at a high dose rate
Baseline mortality	Cancer mortality in Japan (1982) or in the United Kingdom [W21]
Whole population Working population	Age structure of the population in 1982 Population in 1982 between 25 and 64 years of age, both sexes

Table 58

Risk coefficients for adults from the atomic bomb, ankylosing spondylitis and cervical cancer series

(These coefficients were used for the calculations in Tables 59 and 60.)

Malignancy	Sex	Atomic bomb study a/		Spondylitis series b/		Cervical cancer series c/	
		Multiplicative e/	Additive f/	Multiplicative e/	Additive f/	Multiplicative e/	Additive f/
Leukaemia	M	3.7	5.0	3.5	2.0	-	-
	F	3.8	2.9	-	-	0.88	0.61
	Average	3.8	3.9				
Other malignancies	M	0.24	15	0.14	4.7	-	-
	F	0.46	17	-	-	-	-
	Average	0.35	16				

a/ DS86; average values of 25-29, 30-39, and >40 weighted by the proportions of the Japanese population within these age groups. From [S49], appendix tables 5a and 5B, Part II.
b/ From [D21] and [L16].
c/ From [B36], page 1307.
e/ Excess relative risk per Gy.
f/ Excess deaths per 10^4 PYGy.

Table 59

Projection of excess lifetime mortality
for an adult population of both sexes (1000 males or 1000 females)
exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

PLATEAU = 40 years

Malignancy	Sex	Atomic bomb study a/		Spondylitis series b/		Cervical cancer series b/	
		Multiplicative	Additive	Multiplicative	Additive	Multiplicative	Additive
Leukaemia (1) c/	M	9.0	13	14	4.4	-	-
	F	8.1	7.0	-	-	2.8	1.4
	Average	8.6	10				
Other malignancies (2) d/	M	37	29	21	7.6	-	-
	F	46	39	-	-	-	-
	Average	42	34				
Total (1) + (2) (average)		51	44	35	12	-	-

PLATEAU = lifetime

Malignancy	Sex	Atomic bomb study a/		Spondylitis series b/		Cervical cancer series b/	
		Multiplicative	Additive	Multiplicative	Additive	Multiplicative	Additive
Other malignancies (3) d/	M	41	30	23	7.8	-	-
	F	52	42	-	-	-	-
	Average	47	36				
Total (1) + (3)		56	46	37	12		

a/ Reference population: Japan, 1982.
b/ Reference population: United Kingdom, 1982.
c/ Assumed latency time: 2 years.
d/ Assumed latency time: 10 years.

Table 60

Projection of loss of life expectancy
for an adult population of both sexes (1000 males or 1000 females)
exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

PLATEAU = 40 years

Malignancy	Sex	Atomic bomb study a/		Spondylitis series b/		Cervical cancer series b/	
		Multiplicative	Additive	Multiplicative	Additive	Multiplicative	Additive
Leukaemia (1) c/	M	140	290	140	79	-	-
	F	120	170	-	-	31	28
	Average	130	230				
Other malignancies (2) d/	M	400	500	200	120	-	-
	F	530	700	-	-	-	-
	Average	470	600				
Total (1) + (2) (average)		600	830	340	200	-	-

PLATEAU = lifetime

Malignancy	Sex	Atomic bomb study a/		Spondylitis series b/		Cervical cancer series b/	
		Multiplicative	Additive	Multiplicative	Additive	Multiplicative	Additive
Other malignancies (3) d/	M	420	510	210	120	-	-
	F	570	710	-	-	-	-
	Average	490	610				
Total (1) + (3) (average)		620	840	350	200		

a/ Reference population: Japan, 1982.
b/ Reference population: United Kingdom, 1982.
c/ Assumed latency time: 2 years.
d/ Assumed latency time: 10 years.

Table 61

Projection of loss of life expectancy
for a population of both sexes (500 males and 500 females)
exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate
as a function of age using an age-constant risk coefficient

ADDITIVE MODEL

Organ or tissue	Age at exposure							
	0	10	20	30	40	50	60	70
Leukaemia	640	530	420	310	210	120	62	24
All cancers except leukaemia	2360	1750	1230	800	470	240	93	22

MULTIPLICATIVE MODEL

Organ or tissue	Age at exposure							
	0	10	20	30	40	50	60	70
Leukaemia	250	240	250	260	240	190	130	63
All cancers except leukaemia	920	930	920	880	790	620	370	130

Table 62

Projections of excess lifetime mortality and loss of life expectancy
for a population of both sexes (500 males and 500 females) and all ages
exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate
using age-specific risk coefficients

Risk coefficients at ages 0-9 and 10-19 are given in the text;
risk coefficients for adults are given in Table 58.
All risk coefficients are based on the Japanese atomic bomb survivors.

(Based on the population of Japan in 1982.)

EXCESS LIFETIME MORTALITY

	Multiplicative risk projection model	Additive risk projection model
Leukaemia a/	10	10
Other malignancies b/	97	32
All malignancies	107	42

LOSS OF LIFE EXPECTANCY

	Multiplicative risk projection model	Additive risk projection model
Leukaemia a/	260	300
Other malignancies b/	1110	650
All malignancies	1370	950

a/ Plateau: 40 years.
b/ Plateau: lifetime.

Table 63

Projections of excess lifetime mortality and loss of life expectancy for a population of both sexes (500 males and 500 females) and all ages exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate using an age-averaged risk coefficient

The risk coefficients are based on Japanese atomic bomb survivors. Risk coefficients given in Table 54 are averaged over all classes of age at exposure according to the size of each class.

(90% confidence intervals in parentheses.)

EXCESS LIFETIME MORTALITY

	Multiplicative risk projection model	Additive risk projection model
Leukaemia	9.7 (7.1-13)	9.3 (7.7-11)
Other malignancies	61 (48 -75)	36 (28 -44)
All malignancies	71	45

LOSS OF LIFE EXPECTANCY

	Multiplicative risk projection model	Additive risk projection model
Leukaemia	220 (160-270)	300 (250-360)
Other malignancies	730 (570-900)	910 (710-1110)
All malignancies	950	1210

Table 64

Demographic characteristics of countries used to compare risk projections [W21]

	Japan	United Kingdom	Puerto Rico
Life expectancy (years)	76.6	73.7	73.9
Infant mortality (per 1000 births)	8	12	17
Percentage of population under 15 years	23.5	20.3	32
Percentage of population over 64 years	9.3	15.3	8
Cancer mortality rate a/ (1985) (10^{-5})	108.4	150.0	93.9
Death rates a/ (1983-1985) (10^{-3})	4.2	5.8	5.6

a/ Age-standardized.

Table 65

Comparison of projections of lifetime excess mortality
in three reference countries
for 1000 persons of the general population
exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

Excess risk coefficients derived from atomic bomb survivors (Table 56)
and the assumptions given in Table 57.

	Japan		United Kingdom		Puerto Rico	
	Multi-plicative	Additive	Multi-plicative	Additive	Multi-plicative	Additive
Leukaemia	9.7	9.3	13	8.5	9.4	9.7
Other malignancies	61	36	63	31	49	40
Total	71	45	76	40	58	50

Table 66

Comparison of projections of loss of life expectancy
in three reference countries
for 1000 persons of the general population
exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

Excess risk coefficients derived from atomic bomb survivors (Table 56)
and the assumptions given in Table 57.

	Japan		United Kingdom		Puerto Rico	
	Multi-plicative	Additive	Multi-plicative	Additive	Multi-plicative	Additive
Leukaemia	220	300	200	260	200	360
Other malignancies	730	910	740	750	580	1090
Total	950	1210	940	1010	780	1450

Table 67

Excess relative risk per 1 Gy of organ absorbed dose in the low dose range
[S49]

Type of cancer	Dose range (Gy)			
	< 6.0	< 1.0	< 0.5	> 0.5 a/
Leukaemia	5.21 b/	3.96 b/	2.44 d/	5.53
All cancers except leukaemia	0.41 b/	0.46 b/	0.37 d/	0.41
Stomach	0.27 b/	0.41 c/	0.45 e/	0.26
Lung	0.63 b/	0.83 c/	1.06 d/	0.60
Female breast	1.19 b/	1.78 c/	0.82	1.21
Colon	0.85 b/	-0.10	-0.52	0.98

a/ Excess relative risk for doses > 0.5 Gy compared to doses < 0.5 Gy is significantly different only for leukaemia ($p < 0.05$) and for colon cancer ($p < 0.1$).
b/ $p < 0.001$.
c/ $p < 0.01$.
d/ $p < 0.05$.
e/ $p < 0.10$.

Table 68

Lung cancer risk from chronic exposure to radon daughters indoors and outdoors [111]

Source and location	Equilibrium equivalent concentration (Bq/m^3)	Annual exposure (10^5 Bq h/m^3)	Excess relative risk (%)	Excess frequency of lung cancers per million persons per year			
				Reference population			Non-smokers (average both sexes)
				Males	Females	Total	
Radon-222 daughters							
Indoors a/	15	0.90	9.0	54	11	32	7.2
Indoors b/	15	0.23	2.3	14	2.7	8.1	1.8
Outdoors	4	0.040	0.52	3.1	0.62	1.9	0.42
Subtotal		1.2	11.8	71	14	42	9.4
Radon-220 daughters							
Indoors a/	0.5	0.030	1.0	6.0	1.2	3.6	0.80
Indoors b/	0.5	0.0075	0.25	1.5	0.30	0.90	0.20
Outdoors	0.2	0.0020	0.66	0.40	0.079	0.24	0.053
Subtotal		0.040	1.3	7.9	1.6	4.7	1.05
Total			13	79	16	47	10.5

a/ At home.
b/ Elsewhere.

Table 69

Projection of excess lifetime mortality for specific cancers for 1000 persons exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

(Based on the population of Japan.
90% confidence intervals in parentheses.)

Malignancy	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	9.7 (7.1-13)	9.3 (7.7-11)
All cancers except leukaemia	61 (48 -75)	36 (28 -44)
Bladder	3.9 (1.6- 7.3)	2.3 (1.1- 4.0)
Breast a/	6.0 (2.8-10.5)	4.3 (2.2- 6.9)
Colon	7.9 (3.6-13.4)	2.9 (1.4- 4.6)
Lung	15.1 (8.4-23.0)	5.9 (3.4- 8.8)
Multiple myeloma	2.2 (0.6- 5.1)	0.9 (0.3- 1.7)
Ovary a/	3.1 (0.9- 6.8)	2.6 (0.8- 4.8)
Oesophagus	3.4 (0.8- 7.2)	1.6 (0.3- 3.1)
Stomach	12.6 (6.6-19.9)	8.6 (4.5-13.1)
Remainder	11.4 b/ 11.8 c/	10.3 b/ 6.6 c/
Total	70.7 d/ 71.2 e/	45.3 d/ 41.6 e/

a/ These values have to be divided by 2 to calculate the total and other organ risks.
b/ This value is derived by subtracting the sum of the risks at the sites specified from the risks for all cancers except leukaemia.
c/ This value is derived by fitting a linear relative risk model to the basic cancer data after the exclusion of those cases of cancer at the specific sites listed. (Coefficient 0.19 excess relative risk per Gy and 1.87 per 10^4 PYGy).
d/ Red bone marrow plus all other cancers.
e/ Red bone marrow plus other individual sites including remainder.

Table 70

Projection of loss of life expectancy for specific cancers per person exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

(Based on the population of Japan.
90% confidence intervals in parentheses.)

Malignancy	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	0.22 (0.16-0.27)	0.30 (0.25-0.36)
All cancers except leukaemia	0.73 (0.57-0.90)	0.91 (0.71-1.10)
Bladder	0.03 (0.01-0.06)	0.04 (0.02-0.07)
Breast a/	0.11 (0.05-1.90)	0.11 (0.05-0.17)
Colon	0.09 (0.04-0.15)	0.07 (0.04-0.12)
Lung	0.17 (0.09-0.25)	0.15 (0.09-0.22)
Multiple myeloma	0.03 (0.0 -0.06)	0.02 (0.01-0.04)
Ovary a/	0.06 (0.02-0.12)	0.07 (0.02-0.12)
Oesophagus	0.04 (0.01-0.08)	0.04 (0.01-0.08)
Stomach	0.15 (0.07-0.23)	0.22 (0.11-0.33)
Remainder	0.14 b/ 0.14 c/	0.28 b/ 0.17 c/
Total	0.95 d/ 0.94 e/	1.2 d/ 1.1 e/

a/ These values have to be divided by 2 to calculate the total and other organ risks.
b/ This value is derived by subtracting the sum of the risks at the sites specified from the risks for all cancers except leukaemia.
c/ This value is derived by fitting a linear relative risk model to the basic cancer data after the exclusion of those cases of cancer at the specific sites listed. (Coefficient 0.19 excess relative risk per Gy and 1.87 per 10^4 PYGy).
d/ Red bone marrow plus all other cancers.
e/ Red bone marrow plus other individual sites including remainder.

Table 71

Summary of projections of lifetime risks for 1000 persons (500 males and 500 females) exposed to 1 Gy of organ absorbed dose of low-LET radiation at high dose rate

(Based on the population of Japan.)

	Risk projection model	Excess fatal cancers	Years of life lost
Total population a/	Additive	40 c/ - 50 d/	950 c/ - 1200 d/
	Multiplicative	70 d/ -110 c/	950 d/ - 1400 c/
Working population (aged 25-64 years)	Additive	40 d/ - 60 c/	880 d/ - 1330 c/
	Multiplicative	70 c/ - 80 d/	820 c/ - 970 d/
Adult population b/ (over 25 years)	Additive	50 d/	840 d/
	Multiplicative	60 d/	620 d/

a/ Derived from Tables 62 and 63.
b/ Derived from Tables 59 and 60.
c/ Age-specific risk coefficient.
d/ Constant (age-averaged) risk coefficient.

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back to first page

ANNEX G

Early effects in man of high doses of radiation

CONTENTS

Introduction

1. A review of the early somatic effects of radiation in man was published in the UNSCEAR 1962 Report [U1]. This was supplemented in the UNSCEAR 1969 Report by two Annexes, one on radiation-induced chromosome aberrations, the other on the action of radiation on the nervous system [U2], and in the UNSCEAR 1972 Report [U3] by an Annex on the radiation response of the immunological system. The effects of high radiation doses in man were recently re-addressed in part in the UNSCEAR 1982 Report [U4], Annex J, which dealt with non-stochastic effects resulting from localized irradiation of single organs or tissues.

2. In this Annex the Committee reviews data on the early effects of high doses of radiation delivered to the whole human body. There is continuing interest in the effects of whole-body irradiation because of the persistent possibilities of exposure in accidents or from acts of warfare. Whole-body irradiation is also being used in the treatment of disseminated malignancies. However, reliable quantitative data in this field are very limited. They are drawn essentially from isolated accidental exposures, from information gathered on the Japanese population exposed to radiation from the atomic bombs exploded in the Second World War, and from experience with groups of patients receiving whole-body irradiation for cancer or prior to the transplantation of organs.

3. This Annex reviews data on the effects occurring in man within 2-3 months of whole-body doses of more than approximately 1 Gy of low linear energy transfer (LET) radiation or biologically equivalent doses of other radiation types. However, it also includes mention, in some cases, of doses down to 0.5 Gy, of protracted exposures resulting in the same levels of effect as acute doses, and of exposure to internal emitters where the doses were sufficient to have serious effects within 2-3 months. Gaps in the knowledge for man are filled partially by information derived from experimental work with mammals, particularly those with a body size approaching that of man; in general, however, large-animal data are intended to be used for interpretation of responses rather than for extrapolation. Exposures of the whole body resulting in doses to different regions that vary by less than 10%, apply mainly to treatments in radiotherapy. In accidents or in acts of warfare, whole-body doses usually are highly non-uniform (for example unilateral), with the variation in dose from low-LET radiation by a factor of 2 to 3, and from neutrons up to a factor of 10 or more (see, for example, Figure XIX). In these cases, the dose at the midline of the body may bear little relationship to the signs of injury.

4. Many accidents and some oncological treatments involve irradiation of large regions of the body, for example the trunk or the chest. In these cases the doses to specific target organs will determine the response of the individual. The response may differ from that of the same organ exposed to the same dose from irradiation of the whole body, if there are contributions to the expression of injury in the organ from other irradiated tissues, for example granulocytopenia exacerbating intestinal injury.

5. Much information was gathered from the Japanese exposed to the atomic bombs in the Second World War. However, at distances from the hypocentre where doses received were a few Gy, there were also heat and mechanical injuries. Furthermore, the radiation doses received by these individuals remain somewhat uncertain, and recent calculations suggest that the contribution to the dose from neutrons was much less than considered in previous (T65D) estimates of dose. Other groups of individuals exposed to high doses of nuclear fallout radiation were the Marshall Islanders and 23 Japanese fishermen exposed to the nuclear explosions on Bikini Atoll in 1954. These groups received comparatively uniform external gamma-irradiation, beta-irradiation of the skin and internal irradiation. Groups of individuals irradiated with high doses to the whole body in accidents included those at Oak Ridge, United States (the group is widely referred to as "Y-12"), at Vinca, Yugoslavia, in 1958, in China in 1963, in Algeria in 1978, in Morocco in 1985, at Chernobyl, USSR, in 1986, and in Brazil in 1987.

6. When this Annex was approaching completion, important information on the subject became available in connection with the nuclear accident that occurred at the power plant in Chernobyl, USSR, where about 100 people were exposed to external and internal irradiation amounting to 1 Gy or more. The delegation from the USSR has made available especially to UNSCEAR a report on the data gathered in the wake of the accident. The Committee wishes to

acknowledge with gratitude this important contribution. Since time was too short for a definitive study of the data collected and for their incorporation into the text of this Annex, the Committee decided to present them as an Appendix.

7. Clinical data relate to the use of radiation delivered to the whole body to suppress the immune system prior to organ transplantation, to control multiple or systemic metastases from solid tumours, and to treat leukaemia. Although the radiation doses are known accurately for these patients, their responses to these treatments may be confounded to an uncertain extent by debility and disease, by the prior or concomitant use, in many cases, of cytotoxic or immunosuppressive drugs and by different degrees of medical treatment after irradiation.

8. Most of the doses quoted in the literature reviewed in this Annex were given in rad, or in terms of exposure, roentgen (R). As in the UNSCEAR 1982 Report [U4], 100 R of exposure will be taken to be equivalent to 1 Gy absorbed dose in the case of small animals. For larger animals, the doses at depth for equivalent surface doses become progressively less, and this depends on the radiation quality. Doses in the literature are quoted either as surface doses or, more commonly, as midline tissue doses. Conversions will be made where necessary to allow these doses to be expressed in terms of dose in the target tissue under consideration.

9. This Annex is intended to be a scientific compendium on the early effects of radiation in man. It is not meant to be a manual on the care and treatment of irradiated persons, although the information it contains is relevant to evaluating the radiological health consequences of accidents or acts of warfare and the effects of radiotherapy.

I. PATHOGENESIS AND DOSE-RESPONSE RELATIONSHIPS

A. CELLULAR EFFECTS

10. The cellular effects that are important in the response of tissues to irradiation have been described and discussed previously by the Committee [U4]. The severest injuries from radiation in most early-responding tissues are caused by a loss of cells. This results either from death of cells in interphase, as in the case of lymphocytes or, more commonly, from killing of progenitor cells at mitosis, which leads to a lack of replacement of mature cells lost through natural senescence and death. Most mature cells are radioresistant because they divide only occasionally or not at all. In "flexible" type cell populations in tissues such as the liver, the low rate of division of the mature functional cells can be increased, e.g., by partial hepatectomy, and in this case the cells may appear radiosensitive. In the renewing "hierarchical" type tissues [P25] which are specifically discussed in this Annex, such as the bone marrow, gastrointestinal mucosa, epidermis and testis, the maturing and mature cells are resistant because they have, respectively, little or no mitotic potential. In contrast, their progenitor cells have the potential for many divisions and may die from mitotic death. The probability of mitotic death of a cell is a function of the dose and of the number of divisions a cell has undergone since irradiation. After doses up to 6 Gy, irradiated cells have a high probability of completing one division successfully, but a much lower probability of completing six divisions [H41]. Cells that successfully complete six divisions or more can form colonies of more than 50 cells and generally are capable of many more divisions if the cells remain undifferentiated. These colony-forming cells are vitally important for the repopulation of many early-responding tissues (see below).

11. The dose-response curve for the survival of these cells in some tissues (skin, intestine) shows a relatively low sensitivity to doses up to 1 or 2 Gy, followed by an increasing sensitivity at higher doses. The sensitivity to high doses can be approximated to an exponential curve, which is expected due to the stochastic nature of radiation action [I8, T24, U4]. This is characterized by the parameter D_0, which is the dose required to reduce survival by a factor 1/e on the exponential portion of the survival curve. Other associated parameters are the size of the "shoulder" region, which is characterized by the intercept of the exponential survival curve on the linear dose axis, D_q, or on the logarithmic survival axis, n, of a semi-logarithmic plot. These are related by $D_q = D_0 \ln n$. Survival parameters measured for various human clonogenic cells assayed in primary culture are given in Table 1. Cells that die by interphase death are often very radiosensitive, e.g., lymphocytes [W26], and this increases the overall range of sensitivities. Alternatively, the shape can be described by a continuously bending curve when log survival, S, is plotted against dose, D, where

$$S = \exp - (\alpha D + \beta D^2)$$

In this case α is the parameter describing the initial sensitivity, and the sensitivity increases at higher doses depending on the value of β and the dose. This formulation is generally considered to represent better the response of cells to fractionated exposures than formulations based on D_0 [T24].

12. The response of cells in vitro to single doses of radiation, in terms of their colony-forming ability, can be modified by a delay after radiation and before the cells are induced to proliferate. This time interval allows repair of potentially lethal injury to occur, such that more cells retain their colony-forming ability. This type of repair is likely to be important in the recovery of tissues after irradiation. The amount of repair in the tissues under consideration in this Annex will be smaller than in late-responding tissues, where the rates of cell division are lower and remain low for long periods of time after irradiation so that more repair can occur. In the normal tissues of rodents, where repair of potentially lethal damage has been investigated in vivo, the effect generally does not change the D_0 value but it increases all levels of survival on the exponential portion of the curve by factors of about 5 for mammary epithelium [G6], and

about 3 for thyroid epithelium [M24] and hepatocytes [J5]. Other data for hepatocytes show an increase in D_0 [F11]. In bone marrow the opposite effect is observed; namely, a decrease in survival by a factor of 2, which could be due to radiation-induced differentiation [H11], specific for this cell type. The increase in survival observed for most tissues and attributable to repair of potentially lethal damage shows a peak in survival level by about 4 hours which remains unchanged at 24 hours. Studies using assays in vitro have revealed a time-related increase in D_0 for mouse lung cells and kidney cells [U4]. With the latter, the effect observed at 8 hours disappeared by 24 hours. The effects of protracted doses are discussed in chapter III.

13. The earliest effects on irradiated cells are not mediated through mitotic death but are connected usually with membrane integrity. Examples of such early phenomena are the effect on cells comprising the autonomic nervous system that leads to the symptoms and signs of the prodromal syndrome, the interphase cell death characteristic of certain lymphocytes [Y5] and salivary gland cells [S32] and blood vessel injury associated with acute erythema [P28]. When cells are not killed after low doses, membrane injury is generally recoverable. After high doses, these acute effects are often prognostic for later more serious injuries which develop as a consequence of subsequent cell death in other cell populations.

B. TISSUE EFFECTS

14. The majority of the tissues that respond early after irradiation are hierarchical in structure [P25]. In these, mature cells are replenished from proliferative cells by division, differentiation and maturation. The proliferative cells committed to differentiation are produced by very few ancestral stem cells, which are capable of self-renewal and of differentiation (Figure I). Under normal steady-state conditions, the rate of loss of mature cells is equal to the rate of their production.

15. Clinical signs of injury will occur when the loss of mature cells has reached a critical level in any particular tissue. The loss may be induced directly in the mature cell population, as in the case of lymphopenia. Alternatively, it may occur gradually at a rate governed by the natural lifetime of the mature cells when their numbers are not replenished because their precursors are sterilized, as in intestinal mucosa [M16, P25]. In the intestine, the rate of loss may be exacerbated by other factors, such as bacterial infection, which can modify the normal rate of turnover of the cells [M5]. Also, there may be a variable lag period between the time the critical level is reached and the time of failure of the tissue or death: an example is death due to bacteraemia and electrolyte losses which follow cellular depletion in the intestinal mucosa.

16. Effects that are characterized by a threshold dose and by a severity that increases with increasing dose are called non-stochastic effects [I9, U4]. Threshold doses for relatively minor effects are generally smaller than those for severe tissue injury. The time for the maximum effect is also usually dependent on the dose, occurring earlier after higher doses. When doses are relatively low and not all stem cells are killed, tissue injury is followed by recovery mediated through repopulation and differentiation of the precursor cells. The stem cells reproduce themselves and they also differentiate into precursor cells which divide and amplify the number of repopulating cells. After several or many divisions, these "transit" cells mature into the functional cells in the tissue. The time course of repopulation of the mature cells depends therefore on the rate of differentiation of the stem cells, the number of amplifying cell divisions and the cell cycle times [B16, M16, P25].

17. After doses higher than about 10 Gy, where virtually all cells in hierarchical tissues are sterilized, the time required for ablation of the mature and functional cell population is independent of dose, and in many cases it approximates the normal transit time

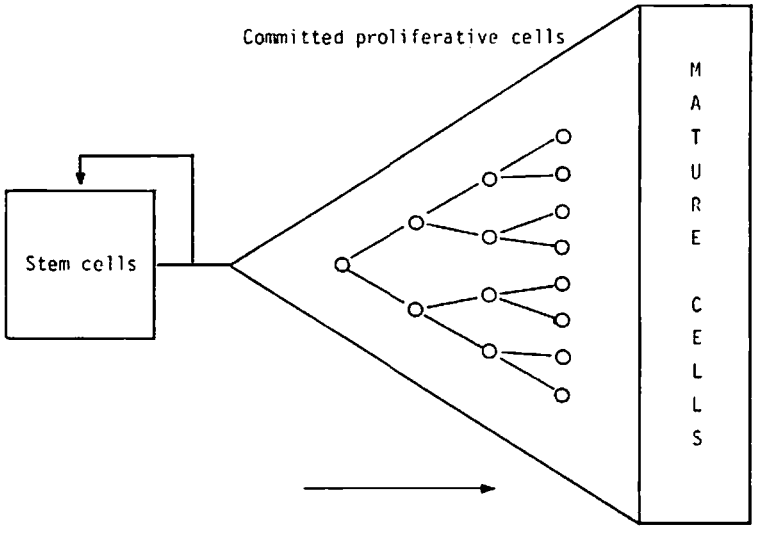


Figure I. Diagrammatic representation of cell population hierarchy where mature cells are produced from proliferative cells. The ancestors of the lineage are the stem cells which renew themselves (left arrow) and which also differentiate into various maturing cell lineages (right arrow).

from one of the lesser differentiated precursor cells to maturity [M16, P8]. For the few non-hierarchical tissues that respond relatively early after irradiation, such as the lung, the latency interval from irradiation to failure may indeed be dependent on dose after fairly high doses before a plateau in latency is reached [M16].

18. After intermediate doses, where most cells in hierarchical tissues are sterilized, the small number of surviving cells in a given tissue type will vary markedly from one animal to another, for the same dose; this results from the stochastic nature of radiation in killing cells, which follows a Poisson distribution. It may be expected that in some cases the number of surviving cells necessary for regeneration of the tissue will have fallen below a critical number, and it may also be expected that the incidence of such cases is dose-dependent [H12, T24]. This allows the construction of dose-incidence curves for particular levels of effect in tissues, e.g., tissue or organ failure, or death of animals, as shown in Figure II.

19. The incidence of a given level of injury is usually related in a sigmoid fashion to the dose. Many empirical distributions have been tested for their goodness-of-fit to a large number of dose-incidence curves for marrow failure in various species, and overall the logistic and probit models were the best

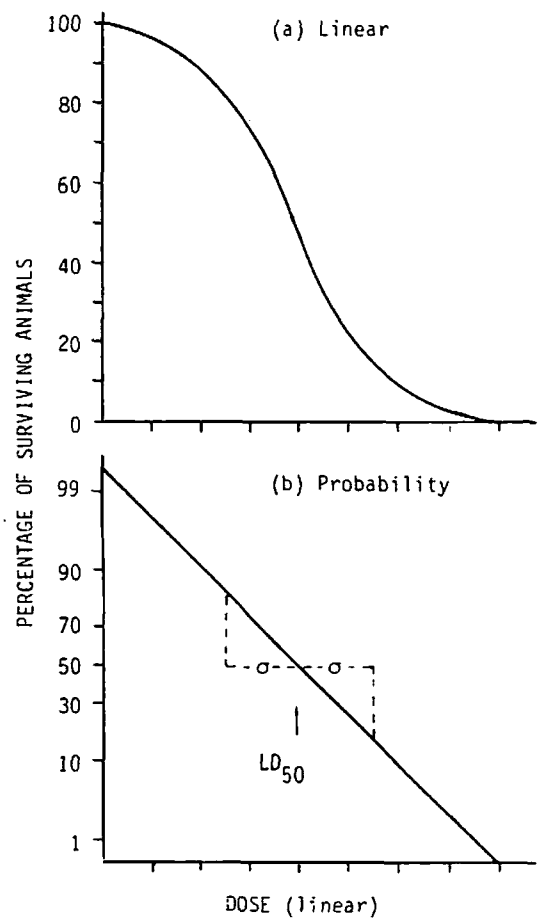


Figure II. Diagrammatic representation of a typical dose-survival curve for irradiated animals, using a linear ordinate or a probability ordinate. The LD_{50} is the dose for 50% incidence, and the slope is characterized by the standard deviation (σ) of the distribution.

representations of the data [M48]. The probit model is based on the normal (Gaussian) distribution [U4, I8]. The 50% incidence level may be estimated most accurately. The slope of the curve, characterized by the standard deviation of the distribution (commonly called the probit width), is a measure of the variation in response among individuals in the population at risk. The dose for 50% incidence of lethality (LD_{50}) or other effects (ED_{50}) and the probit width (σ) are the two parameters commonly used to describe the shape of the curve (Figure II; see also other examples in Figures XXI and XXII).

20. Three main sources of variation may contribute to the probit width [H12]. First, there is the Poisson distribution of lethal events among the critical cells at risk. The probit width generally is not less than the D_0 value for the target cells (which may be in sensitive or resistant phases at the time of irradiation), and in those systems that the Poisson distribution adequately describes event frequencies, the probit width is empirically about 1.2 D_0 [L3]. Second, there is the variation in sensitivity, $1/D_0$, between cells in different individuals. Third, there is the variation in dose delivered to different individuals. This last source of variation may relate to the distance from the source or, in some situations, variations in the shielding of parts of the body. In cases where the first source of variation predominates, a Poisson model can be used to construct a dose-mortality relationship, and this is not markedly different in shape from a Gaussian curve over the range of mortalities measured from about 5% to 95% [L3]. Conversely, a lower limit to the sensitivity of the target cells can be deduced from a mathematical transformation of the mortality probabilities versus dose [G3].

C. THE RADIATION SYNDROMES

21. The lethal effects of radiation in animals reflect failure of particular organs. These fail after different periods of time, related to the underlying cell kinetics (see section I.B). There is a latency period before the development of injury, and following the expression of injury there may be a recovery phase, depending on the dose. The temporal sequence of events is characterized by a combination of symptoms and signs (a syndrome). Radiation syndromes in man have been discussed in a number of publications [e.g., A16, B31, C36, C41, G26, L22, T23, U1, U4, U9, W13, Y7].

22. Different organs fail over different ranges of dose. The response of an organ is due primarily to the dose it receives, but this can be modified by effects in other irradiated organs: for example, granulocytopenia allows the development of bacterial invasion following epithelial loss in the irradiated gut. These additional features will change the incidence of mortality as a function of increasing dose by an amount that depends on the target tissue at risk and the particular confounding effects applicable.

23. In studies using groups of animals belonging to different mammalian species, the pattern of mortality versus acute dose can be delineated into a series of typical syndromes; namely, the bone marrow syndrome,

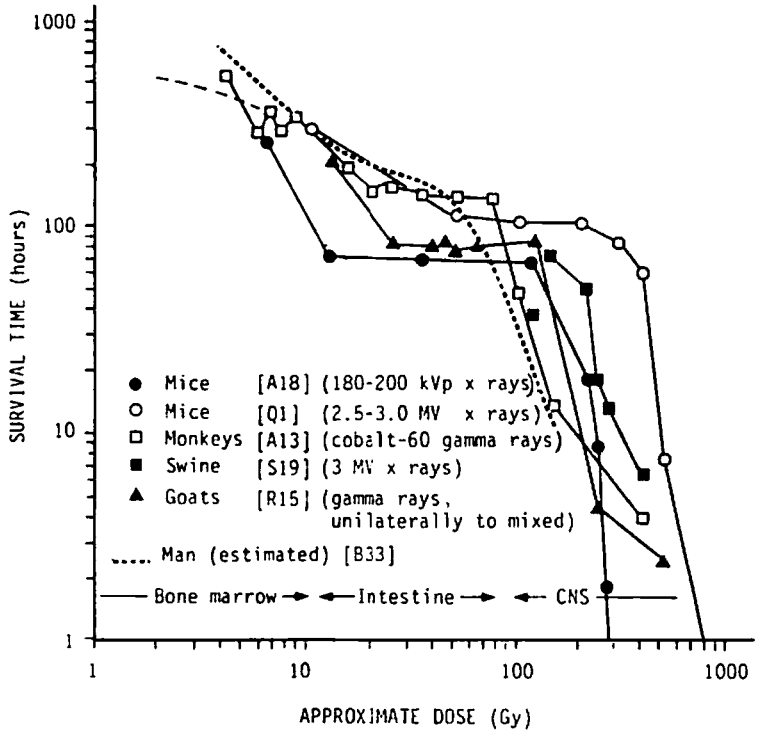


Figure III. Survival time of several mammalian species after various whole-body doses (doses are quoted as approximate maximum tissue doses).
[B16, T24]

the gastrointestinal syndrome and the neurological (or neurovascular) syndrome. Representative data for animals are shown in Figure III. Doses (Gy) are quoted as approximate maximum tissue doses. With mice and monkeys, doses in the target tissues, i.e., marrow, intestine and CNS, probably are within 10% of these doses. With swine and goats, doses in the marrow and intestine may be less than the quoted doses by slightly more than 10%; for swine, this figure may be about 20% for bone marrow and could be up to 30-40% for the intestine if the dose in the middle of the abdomen is the most relevant dose. The percentage for goats is uncertain, as the irradiation was unilateral using mixed gamma rays and neutrons. Man is expected to conform to a similar pattern of response versus dose (dotted curve, Figure III). Figure III shows that in the interval of dose from roughly 2 to 10 Gy, where the bone marrow syndrome occurs, survival time decreases with increasing dose; survival time remains relatively constant between roughly 10 to 50 Gy, where the intestinal syndrome prevails; at still higher doses, the neurological syndrome becomes predominant and over this interval survival time again becomes very dependent on dose. It should, however, be emphasized that the syndromes are idealized clinical pictures, which are difficult to distinguish in practice, particularly when the inhomogeneities in dose are very pronounced and when injury from other causes is present [B57, W28, W29].

1. The prodromal phase

24. The prodromal phase comprises the symptoms and signs appearing in the first 48 hours post-irradiation [C36, G2]. After supralethal doses of several tens of Gy, all individuals begin to show all symptoms characteristic of this phase within five to 15 minutes. The reaction is mediated through the response of the autonomic nervous system and is expressed as gastrointestinal and neuromuscular symptoms. The former symptoms are anorexia, nausea, vomiting, diarrhoea, intestinal cramps, salivation and dehydration. The neuromuscular symptoms are fatigue, apathy, listlessness, sweating, fever, headache and hypotension, followed by hypotensive shock. The reaction after high doses is maximal within 30 minutes, then diminishing until it merges closely with the neurological syndrome or, later, with the gastrointestinal syndrome. Leukaemic patients given 10 Gy to the whole body at 0.05 Gy per minute in many cases had a fever, occasionally associated with chills at the end of irradiation, but they were usually afebrile by 24 hours [D17]. After lower doses, the symptoms are delayed, fewer and less severe, comprising mainly anorexia, nausea, vomiting and fatigue. Vomiting is infrequent after doses below 1 Gy [B32, D9, L8]. The responses can be produced by separate irradiation of the head, thorax or abdomen, the last being the most sensitive region [G2]. Also, the region below the umbilicus is less responsive than the region above it, as shown by prodromal responses in cancer patients receiving half-body irradiation at 3-10 Gy [F18]. In monkeys, vomiting is suppressed during incapacitation after high doses [M29].

25. Mechanisms of radiation-induced nausea and vomiting have been discussed [H34, Y3]. The neural control mechanism for emesis is located in two distinct regions of the medulla oblongata: the area postrema containing the chemoreceptor trigger zone

(CTZ) and the vomiting centre [B34]. The latter is the final pathway for emesis, whether the signal originates from the gastrointestinal tract or the CTZ. Ablation of the CTZ eliminates prodromal vomiting in the dog, monkey and man. Small peptides are implicated as mediators of emesis [C23]. Inflammatory processes could be involved in post-irradiation vomiting, as suggested by the success of anti-inflammatory agents in controlling emesis in animals [H30] and in patients receiving large-field or whole-body irradiation for radiotherapy [B32, S17].

26. Attempts have been made to define dose-response relationships for the various signs and symptoms of the prodromal phase. This has been done for casualties of the atomic bombs [O5], nuclear accident victims and cancer patients receiving therapeutic whole-body irradiation [M18, L10]. The most comprehensive studies with cancer patients involved 504 individuals irradiated at various hospitals in the United States and Canada [L8]. The observations were corrected for the natural incidence of between 8% and 19% of non-radiologically induced symptoms. ED_{50} values (effective dose for a given response in 50% of the irradiated individuals) for various prodromal symptoms occurring within 48 hours are given in Table 2. Higher doses were required to elicit responses within 12 hours rather than within 48 hours, and after lethal doses the onset of vomiting in 100 patients was calculated to be greatest about two hours after irradiation [L4]. After very low doses, the peak incidence of nausea and/or vomiting, if these symptoms occurred, was calculated to be approximately 6 hours after exposure [G2]. An approximate relationship between the time of onset of prodromal symptoms and dose is shown in Figure IV. A comparison of ED_{10} values for patients not showing signs of illness before irradiation and ED_{10} values for all patients showed that the values for the former were only slightly greater than for the latter, suggesting that illness did not markedly predispose to greater responsiveness to prodromal symptoms. This was also indicated by the similarity in the dose-incidence relationship for vomiting, when the clinical data were compared with those for 45 healthy individuals who were separated into four average dose groups (label 2 in Figure V) [L4, U5]. The start of the prodromal reaction in people suffering from the bone marrow syndrome coincides satisfactorily with the data in Figure IV.

27. In relatively healthy Ewing's sarcoma patients treated with whole-body irradiation [M34, R6], prodromal symptoms were observed in all those receiving 3 Gy, but not in those receiving 0.5-2.2 Gy. With whole-body irradiation of leukaemic patients using 10 Gy to the midline delivered at 0.05 Gy per minute, nausea and vomiting began after 3-4 Gy had been given [T19, T20]. These patients were treated with high-dose cyclophosphamide during the week preceding irradiation, and they received sedation with barbiturates and chloropromazine before irradiation. Vomiting after 3 Gy had been accumulated was also seen in another series of leukaemic patients given whole-body irradiation [B32]. Vomiting did not occur

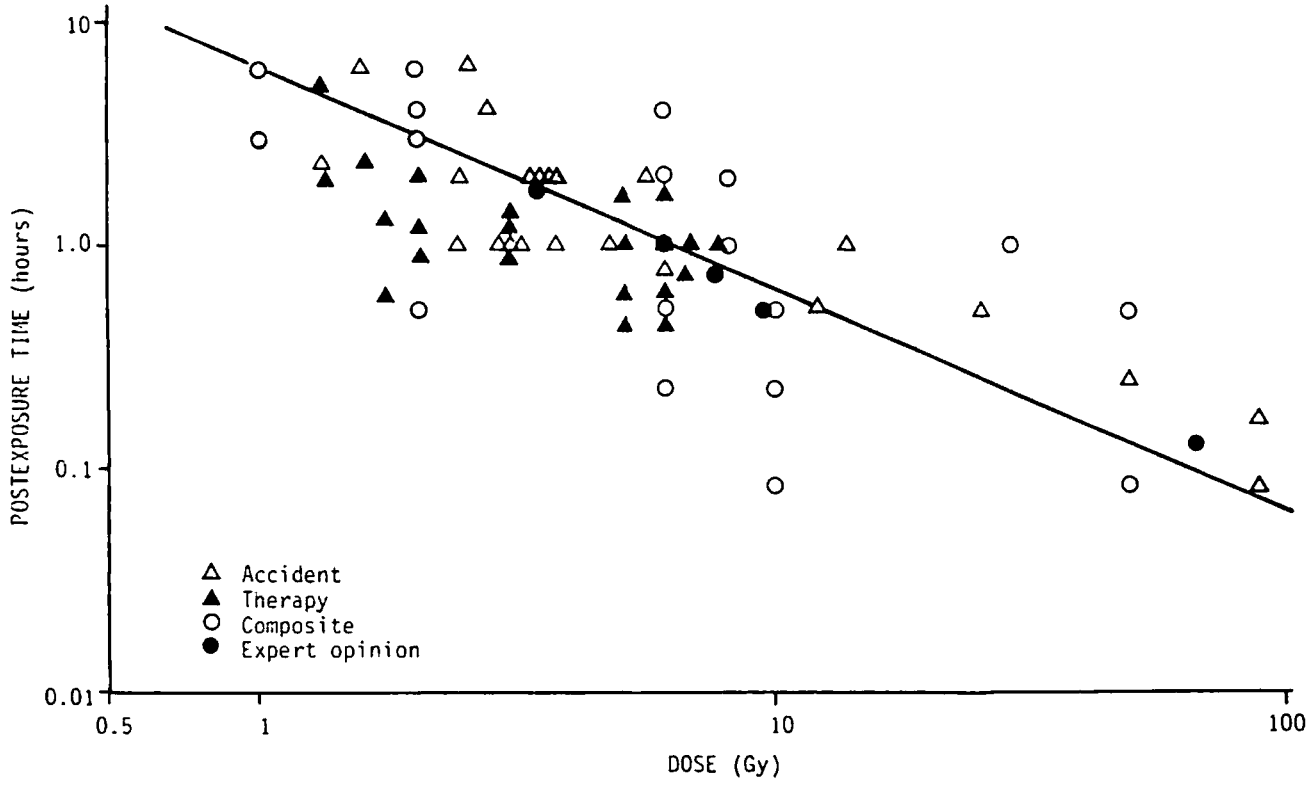


Figure IV. Relationship between time of onset of prodromal symptoms and dose in man. Dose rates ranged from very high (accident cases) down to 0.3 Gy per minute (radiotherapy patients). Approximate midline doses are quoted. (Modified from [B33].)

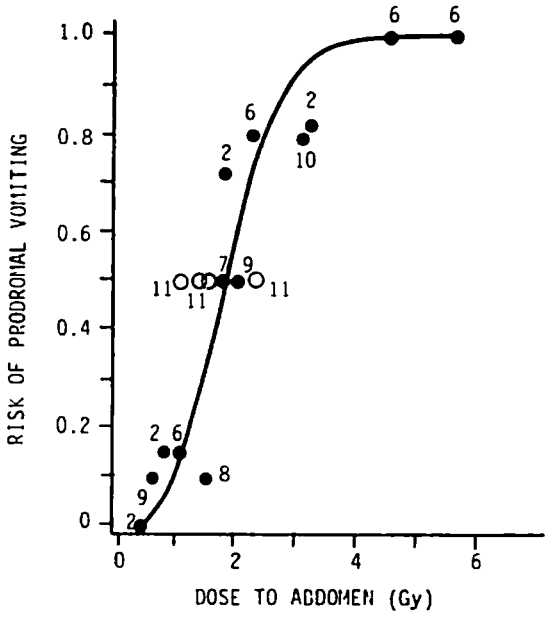


Figure V. Dose-effect relationship for prodromal vomiting within two days in man.
[U5]
2 — accident cases [L4]; 6 — accident cases [T5]; 7 — therapy patients [T17]; 8 — Rongelap natives exposed to fallout radiation [L4]; 9 — midway between the normal arithmetical and log-normal values in the analysis given by Langham [L4]; 10 — Toronto-therapy cases (11/14) with Gravol pretreatment; 11 — doses calculated for a risk of 0.5 of, respectively, anorexia, nausea, fatigue and vomiting (same as symbol 7) and diarrhoea (from left to right in figure) in 163 therapy cases [L9]. Doses are original estimates in the cases of accidents.

earlier than 30 minutes after doses from 2.7 to 7.0 Gy. The effects were independent of dose rate above 0.06 Gy per minute.

28. Quite marked variations in responses are apparent between various small series of leukaemic patients irradiated similarly; this could be due to differences in the severity of their illnesses and in medications supplied. For example, only two out of eight patients with haematological malignancies vomited during irradiation with 10 Gy given at 0.05 Gy per minute. One of the two vomited after 5 Gy had been delivered and the other after 7 Gy had been delivered [C35]. Four out of seven ill cancer patients given about 1 Gy at 0.06 Gy per minute vomited, between 1 and 4.5 hours after irradiation, as did three out of four at 1.5-2.5 hours after about 1.3 Gy [L34]. Twenty-two out of 30 patients with various advanced cancers given 1.3 Gy at 0.02-0.05 Gy per minute experienced nausea but did not vomit [M18].

2. The neurological (neurovascular) syndrome

29. Doses higher than about 100 Gy to most mammalian species result in death from cerebrovascular injury within two days. Survival times are shorter for higher doses, and after 1,000 Gy most species survive only a few hours or less [B16]. The effects of radiation on the central nervous system (CNS) were reviewed in the UNSCEAR 1969 Report [U2]. The CNS syndrome is characterized by severe symptoms and signs of the prodromal syndrome, followed by transient periods of depressed or enhanced motor activity leading to total incapacitation and death.

30. Histological studies on the brains of rhesus monkeys receiving 100 Gy showed perivascular infiltration, haemorrhages and oedema, reaching a peak at 8 hours after irradiation [V6]; pycnosis of neurons was maximal at 24 hours, suggesting that vascular changes might be the initiating lesion in the brain.

31. A study of the brains of 49 casualties who died at various times greater than 6 days after the Hiroshima and Nagasaki bombings revealed pathological changes characteristic of perturbations in vascular permeability [S7]. In 10 patients surviving accidental gamma- and neutron-irradiation (average whole-body dose, 5-6 Gy; average head dose, 8-10 Gy), cerebral lesions (disturbances in the brain circulation of the blood and cerebrospinal fluid) were found soon after irradiation [K8]. In monkeys, irradiation of the head alone produces the CNS syndrome [C5]. One man receiving inhomogeneous whole-body irradiation, with a dose to the front of the head of about 100 Gy of mixed gamma and neutron radiation, died after 35 hours. The main neuropathological finding in the brain (mean dose of about 25 Gy) was severe oedema. The heart (dose of about 120 Gy) showed interstitial myocarditis, which was considered the primary cause of death in this particular case [S6]. The findings among the victims at Chernobyl, in connection with the neurological syndrome, are described in the Appendix.

32. High doses can result in severe cardiovascular dysfunction [H46]. For example, in two persons involved in criticality accidents, the inability to maintain systemic arterial blood pressure was considered the primary cause of death [S6, F17]. Also, in a study of cancer patients given half-body irradiation, two deaths were attributed to myocardial infarction after an acute hypertension episode during the first few hours post-irradiation [S17].

33. Changes in sensory perceptions are also produced by high radiation doses. Reduction of tactile sensitivity and skin sensitivity has been reported in cases of accidental irradiation in the lethal range of doses [K8, S24].

3. The gastrointestinal syndrome

34. Animals receiving doses of between about 10 and 50 Gy die with signs of the gastrointestinal syndrome. The mean time to death after doses of about 50 Gy in various large species of animal varies between 3.5 and 9 days [B16]. The symptoms in man follow those of the prodromal phase, and include anorexia, increased lethargy, diarrhoea, infection, and loss of fluids and electrolytes. Other signs include weight loss, diminishing food and water intake, gastric retention and decreased intestinal absorption [B16, B56, G31]. The leucocyte count falls dramatically, and there may be haemorrhages and bacteraemia, which aggravate the injury and contribute to death after high doses and also after

lower doses where the gastrointestinal and bone marrow syndromes overlap.

35. The intestinal signs that follow the prodromal phase appear as a consequence of cell depletion of the intestinal lining, as described in detail in the UNSCEAR 1982 Report [U4]. The depletion is due to loss of reproductive capacity of the clonogenic cells in the crypts, so that the normal continuous flow of new cells on to the villi ceases. The hierarchy of cell populations in the intestinal mucosa is shown diagrammatically in Figure VI. The amount of cell sterilization is dependent on dose.

36. Histological specimens from individuals who died with signs of severe intestinal damage after irradiation from the atomic bombs in Japan revealed atypical epithelial cells, an oedematous and atrophic mucosa and petechiae, as well as ulcerative lesions after the seventh day [O5]. Similar histological findings were observed in monkeys dying 6-8 days after whole-body gamma-irradiation [W7]. In these monkeys the most prominent findings at necropsy were gastric and colonic ulcers, together with severe mucosal atrophy. The incidence of colonic ulceration was independent of dose over the range tested, 15-75 Gy, but the incidence of gastric ulceration increased with increasing dose. Gastric ulceration developed after the fourth day, predominantly in regions of the stomach richest in parietal cells.

37. The time course of events is almost independent of dose between 10 and 50 Gy but is very dependent on the species. The time course is correlated with the rate of loss of the intestinal cells covering the villi. For example, the development of the gastrointestinal syndrome is longer in germ-free than in conventionally housed mice, in which the villus transit time is shorter [M5, T26]. In man, the cell transit time on the villus is 3-4 days, as shown in Table 3, which summarizes kinetic data for the intestine. The time of death is also influenced by other concomitant factors, such as infection, haemorrhage and fluid loss. The dose range resulting in the gastrointestinal syndrome in man is unknown, but it is probably similar to that observed for large animals (see Figure III). Gastrointestinal signs were noted after whole-body irradiation of leukaemic patients prior to marrow transplantation, when the dose delivered at about 0.05 Gy per minute was increased to 12 Gy [D17].

38. The time to death can be deduced from the time course of the frequency of deaths following the atomic bombs in Japan. For a total of 757 documented deaths in Hiroshima and Nagasaki [O4], the time course of deaths showed two clear peaks in frequency, one between days 6 and 9 and the other between days 20 and 30 (Table 4). The first peak is attributed to the intestinal syndrome and the second to the bone marrow syndrome. One group of people dying at times around the first peak comprised 21 documented individuals who were in the Bankers Club in Hiroshima at the time of the explosion [O5]. Eight of them suffered radiation injury only and died at various times between 6 and 17 days after irradiation. On the fifth day after exposure, the leucocyte counts were below 500 per μl in five of the seven cases in the Bankers Club who died in the first week. The degree of anaemia was very variable. The sample in Table 4 is a very small proportion of the people that died after the bombing, and therefore selection procedures may have influenced the apparent distribution of deaths.

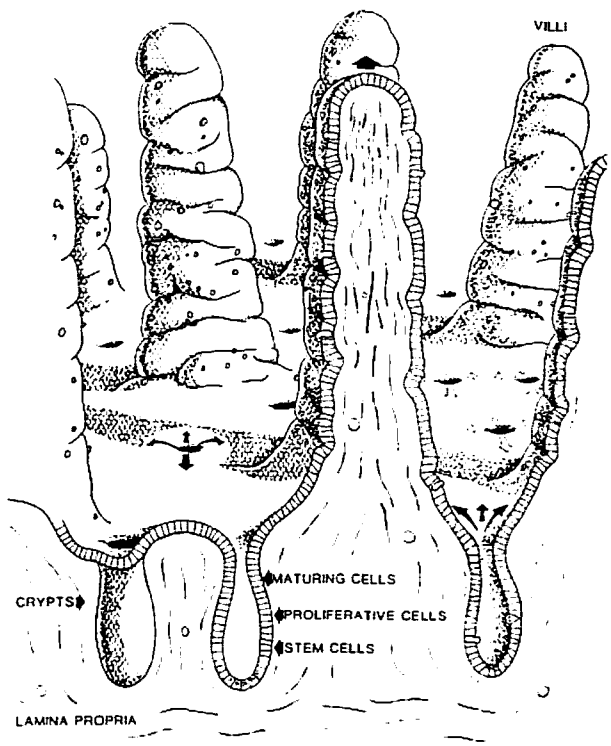


Figure VI. Diagrammatic representation of cell production in intestinal crypts, with new cells migrating on to the functional units, the villi.
(Adapted from [P29].)

Also, there may have been a contribution from mechanical injuries. A more extensive analysis of mortality versus distance from the hypocentre and time after the bombing in Hiroshima was undertaken [I11]. This revealed a peak in mortality rate slightly before 10 days for individuals exposed at distances between 500 m and 999 m from the hypocentre, and a peak at about 20 days for individuals between 1000 m and 1499 m, after allowing for an estimated contribution to death from mechanical injuries. This is probably the best evidence available concerning time to death of people from the gastrointestinal and bone marrow syndromes.

39. Deaths at these times from accidental exposures have been rare, e.g., one person in the 1946 Los Alamos criticality accident died at day 9. The granulocyte count was below 500 per μl on day 6, and it remained low until death on day 9 [H9] (see also the Appendix for other cases of accidental exposure).

40. The gastrointestinal syndrome in all species occurs concomitantly with various degrees of fluid, protein and electrolyte loss, mucosal atrophy and ulceration, infection and haemorrhage [B16, B56, G31]. In man, severe enteritis occurs from about day 4 after doses above 10 Gy and from about day 7 after 6-10 Gy (see Appendix). In animals, the incidence of intestinal death can be reduced by transfusions with balanced salt solutions and antibiotics; for example, the $LD_{50/5}$ for rats can be increased by a factor of 1.4 by the use of antibiotics [T1]. Fluid loss in the gastrointestinal syndrome can be counteracted by infusions of electrolyte solutions [F3]. In most species, it has been stated in general that early mortality (3-6 days after exposure) after doses of 2-4 times the $LD_{50/30}$ or $LD_{50/60}$ can be reduced to zero if supportive care is employed [F3]. Such procedures, which involve fluid replacement, parenteral nutrition, antibiotic and blood-component transfusions, are effective in humans suffering from the gastrointestinal syndrome. However, no accurate assessment of their efficacy in man is available even following the experience in Chernobyl (see Appendix).

4. Haematological and immunological effects, and the bone marrow syndrome

41. Animals die from marrow failure within 30 days after doses between about 2 Gy and 10 Gy, depending on the species. The $LD_{50/30}$ is related to body weight, as shown in Figure VII. Death from bone marrow failure is associated variously among species with granulocytopenia, thrombocytopenia and lymphocytopenia [B16]. In most species, anaemia is less severe than neutropenia or thrombopenia and does not correlate well with time of death [B16]. This is due partly to the radioresistance and the long life span of red blood cells (109-127 days in man). The lack of a severe response indicates that haemorrhage is not a major problem after doses in the LD_{50} range, but it would become increasingly important with higher doses. Similarly, thrombocytopenia, occurring because of the sensitivity of megakaryocytes and the relatively short life time of platelets in the blood (8-9 days in man [L5, C17, B16]), would not be regarded as a major contributor to mortality in the LD_{50} range but would become increasingly important after high doses.

42. Regeneration of these mature populations of cells occurs from the surviving precursor cells after

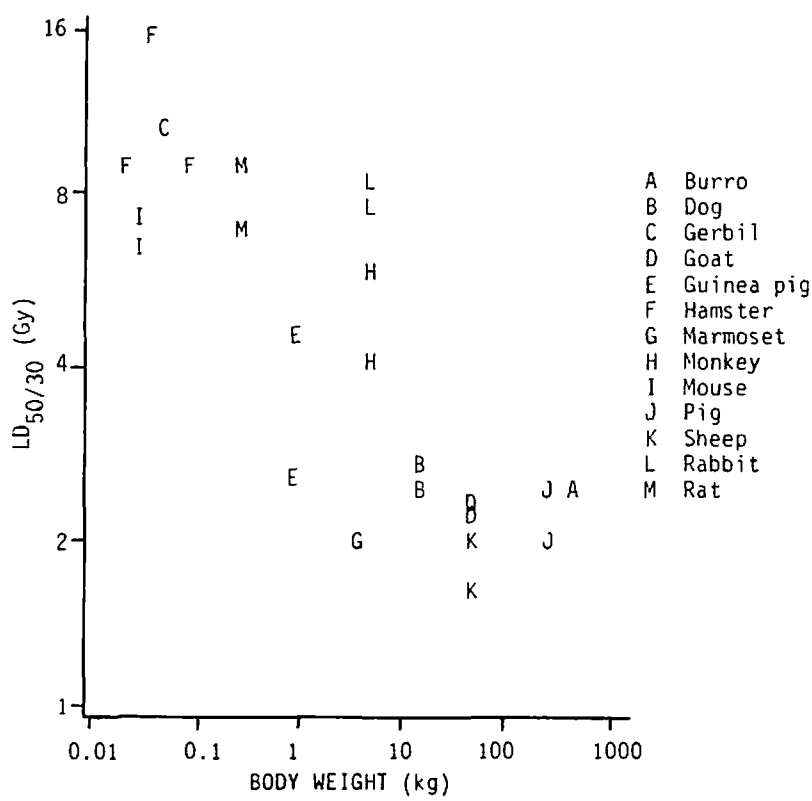


Figure VII. Relationship between $LD_{50/30}$ and body weight for various mammals. (Modified from [U4, U5].)

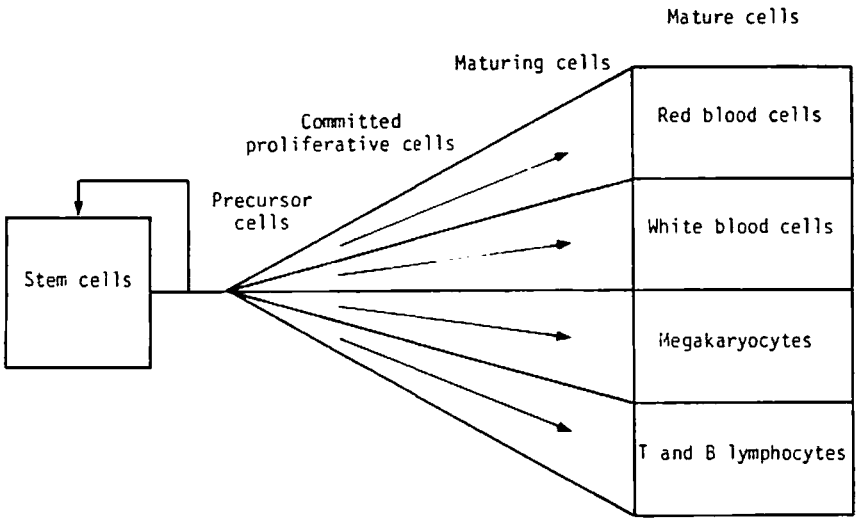


Figure VIII. Simplified diagrammatic representation of the haemopoietic hierarchy, where mature cells are produced from proliferative precursor cells. Left arrow indicates renewal of stem cells, right arrows indicate differentiation and maturation down particular lineages.

irradiation; the hierarchy of haemopoietic cells is shown diagrammatically in Figure VIII. The longer the animal survives, the greater will be the contribution to survival of cell progeny from primitive surviving precursor cells in the marrow. Hence, in the short term, rescue of the animal will be assisted by survival of the more mature precursors, e.g., the granulocyte/macrophage colony-forming cells (GM-CFC); and, in the longer term, rescue will be dependent on the survival of multipotential stem cells. GM-CFC are assayed in vitro, and differences in radiosensitivity have been reported among species (reviewed in [H11]). GM-CFC in dogs are more sensitive than in mice or in man. However, in view of the marked differences in apparent sensitivity of human GM-CFC measured using different culture conditions [B28], it is not clear whether the differences reported among species are artefactual or absolute.

43. The sensitivity of haemopoietic stem cells has been measured using the spleen colony technique in the mouse [T8] and in the rat [C8], but not in other animals. The possibility exists to measure the radiosensitivity of these cells in other species from the formation of foli of undifferentiated cells in irradiated bone marrow [H48, S47]. The precursor cell type that can be grown in vitro from different species and which is so far known to be nearest to the stem cell in the hierarchy is a cell that is capable of forming colonies in vitro comprising many haemopoietic cell types (Table 1). The concentration of these cells in bone marrow is very low, as expected, so it is difficult to measure their intrinsic radiosensitivity. Their sensitivity has been measured in mouse and in man, but not in other species.

44. In human bone marrow, the total number of nucleated cells is reduced at day 1 by 10-20% after 1-2 Gy, by 25-30% after 3-4 Gy, by 50-60% after 5-7 Gy, and by a maximum of 80-85% after 8-10 Gy. Resistant cells remain, such as macrophages, stromal cells, vascular endothelium and some mature granulocytes and eosinophils [M25]. At day 1 after doses of a few Gy, resulting in the bone marrow syndrome, a relative trebling of macrophages and stromal elements has been reported [S21]. Bone marrow cellularity reaches a minimum value by days 3-4 after 5 Gy or above and by days 5-7 after 2-4 Gy. Regeneration can be detected in the marrow at days 4-6 by the presence of colonies of undifferentiated cells. The phase of pronounced aplasia is characterized in the marrow by oedema, a lack of adipose cells and a cellular composition of mainly lymphocytes, monocytes and plasma cells. When regeneration occurs, the number of undifferentiated cells increases to a maximum at days 14-20. It has been reported that after doses of up to 10 Gy cell regeneration in the marrow begins earlier than after lower doses [B38, V12].

45. Various attempts have been made to construct dose- and time-response curves for the changes in concentration of platelets, lymphocytes and neutrophils in the peripheral blood of healthy humans receiving whole-body exposures [A14, B31, C37, P13, W2]. A schematic picture of the smooth average time courses for the various blood cell types after different ranges of dose (Figure IX) was deduced from accidental human exposures [H9, C15, G9, H6, B29, J4, T5, B17, S6, C11]. The values in these idealized pictures are expressed as percentages of average levels in the normal population. Control ranges (±2 SD) measured in five separate studies have been summarized [T29]. The extremes are 4-11 10^9 WBC/l for males and 4-9 for females; 4-6 10^{12} RBC/l for males and 3.7-5 for females; 34-54% haematocrit for males and 33-48% for females; 130-176 g haemoglobin/l for males and 113-162 for females.

46. The patients irradiated prior to kidney transplantation showed an earlier and more rapid decline in numbers of lymphocytes and granulocytes than the accident victims at Oak Ridge (Y-12) and Vinca irradiated with comparable doses. Also, in the patients the nadir levels (minimum values) were lower, but the regeneration of granulocytes began earlier and rose to higher levels. These differences would be compatible

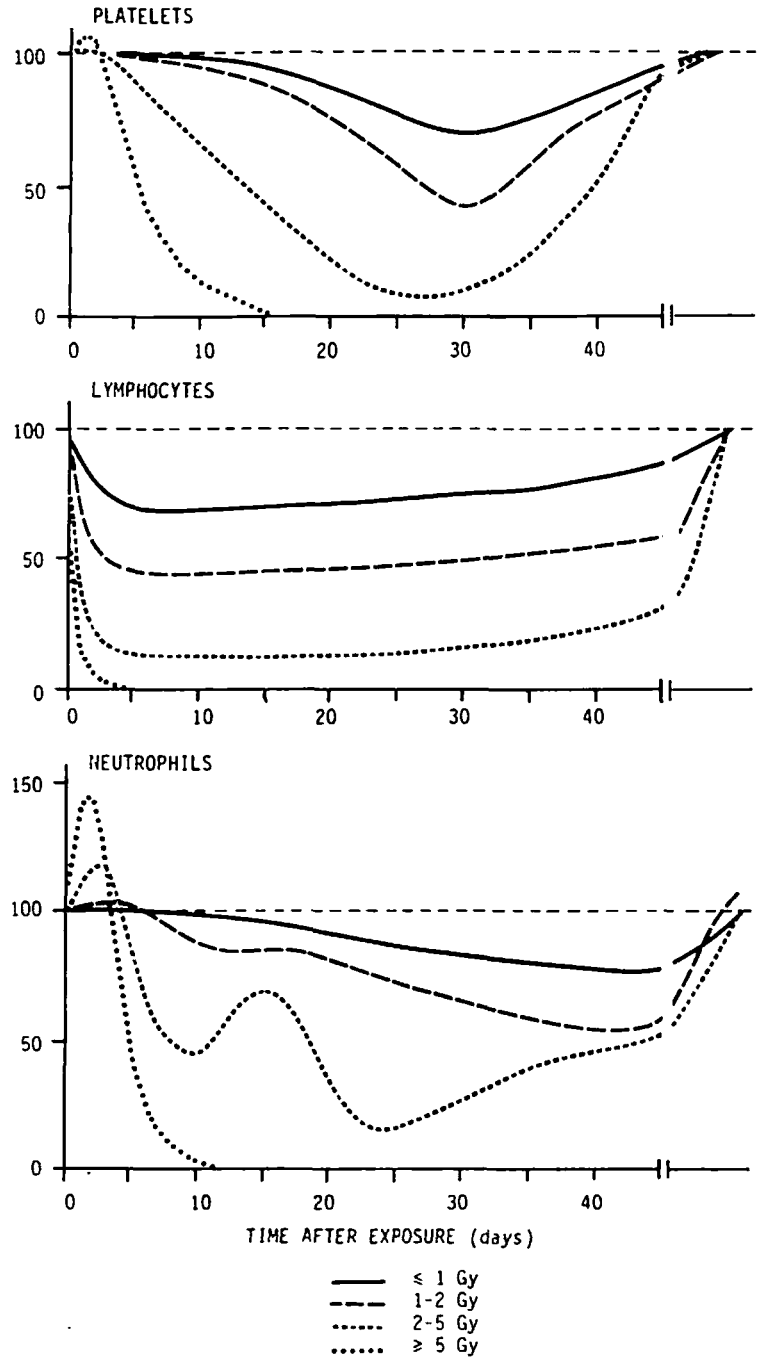


Figure IX. Schematic picture of average time courses for various cells in the blood, after various doses of radiation in man, derived from accident cases.
(Redrawn from [W2].)

with higher effective doses to the patients, because after the Y-12 accident the individuals receiving the higher doses, compared with those receiving low doses, had a greater fall in granulocytes but earlier regeneration reaching higher levels by day 60 [A2]. The greater response in the transplantation patients is difficult to explain, although it should be noted that many of the patients were anaemic and they had a short expectation of life. Different marrow doses, differences in the uniformity of dose, the contribution from neutrons in the accident cases and the confounding influence of concomitant disease have all been suggested as contributory factors [T10, T11, T12].

47. A greater-than-expected haematological response was also observed in patients with chronic granulocytic leukaemia [A1] exposed to 0.25 Gy and 0.5 Gy (midline doses) whole-body irradiation, in spite of the low exposure rate of 0.0012 to 0.0076 Gy per minute (at the midline). The rate of recovery of blood cell counts was slower than in the transplantation cases discussed above. These differences have been taken to indicate that data pertaining to irradiated patients suffering

from haematological diseases are not applicable to healthy individuals [A1] (except, perhaps, those data pertaining to patients in remission) [B41].

48. Figure IX shows that the lymphocyte count is the most sensitive index of radiation injury in the blood, in the sense that, for the same dose, nadir levels are reached earlier than for other cell types. Lymphocytes die in interphase, and doses of 1-2 Gy cause their numbers to decline to about 50% of normal by 48 hours. Decreases can also be observed during irradiation. For example, at the end of a 4-hour period during which 10 Gy was delivered to leukaemic patients in remission, the lymphocyte count was 50% of pre-irradiation levels, and it subsequently declined with a half-time of about 30 hours [D22]. A plateau was then reached which is dose-dependent, remained for about 45 days and was followed by a slow recovery over several months. The dose-dependence of the plateau level has been estimated in two reports from accident cases [W2, P13], and the results of the two reports are fairly consistent, one with another (Figure X and Figure A.II.b).

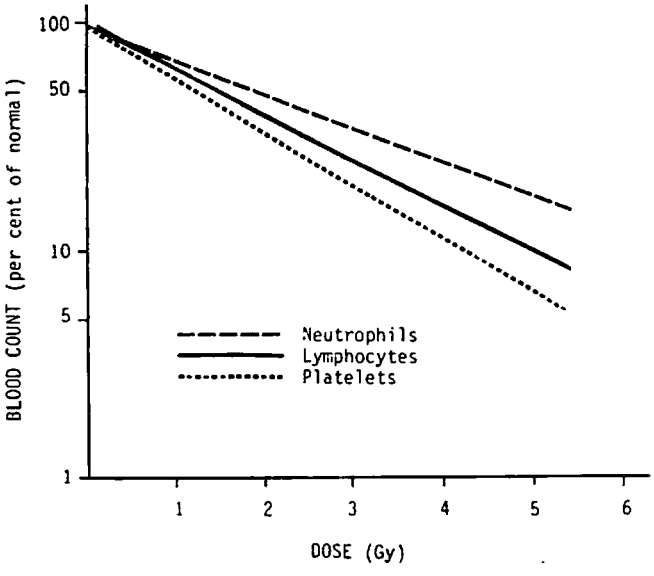


Figure X. Idealized average dose-response curves for nadir levels in blood cell counts.
[W2]

49. Neutrophils show an initial increase in number over the first few days after doses of 1-2 Gy or higher, and this "abortive rise" is greater after larger doses (Figure IX). Immediately after the delivery of 10 Gy in 4 hours to leukaemic patients in remission, the granulocyte count rose by a factor of 2-4 [D22]. A significant increase was noted as early as 10 minutes into the irradiation, when only 1.2 Gy had been given. The rise is probably due to a transient mobilization of cells from marrow and/or extramedullary sites and to accelerated maturation of precursor cells [B16]. This initial phase of granulocytosis is followed by a decline in the number of white cells, the rate and extent of which are dose-dependent. At day 10 after doses of 2-5 Gy there is the beginning of a second abortive rise, due to recovering haemopoiesis from precursor cell populations; this extends to about day 15 and is followed by a second decline to about day 25, due to a lack of recovery in the stem-cell population. The absence of a second rise in granulocytes is indicative of the failure of haemopoiesis to recover permanently [B16]. The second abortive rise is not seen after doses higher than 5 Gy (Figure A.V (left panel)).

50. With whole-body doses in excess of 6 Gy the critical level of neutrophils is reached in 7-9 days; after 4.2-6.3 Gy, it is reached in 10-20 days. With doses lower than 4 Gy, the critical level is generally reached after 20 days or more [U6]. The dose-dependence of the white cell count is shown in Figure A.V (left panel), which depicts the time to the minimum number of granulocytes; alternatively, Figure A.V (right panel) shows the time to reach the critical level of 500 granulocytes per μl (see below). From Figure A.V (right panel) it can be seen that after about 6 Gy, the granulocyte level would be reduced to 10% (from 5,000 to 500 per μl) in 12-14 days. In Figure X, the nadir is also 10% after 6 Gy, but it is reached somewhat sooner, after about 7 days (Figure IX).

51. The times between days 20 and 30 are critical for fever and infections. The period during which agranulocytosis is observed coincides with a period of fever both in animals [B17] and in man [T11, T12, Z2]. Studies of the correlation between granulocytopenia and the onset of fever showed that the latter was better correlated with the time of the minimum number of granulocytes (Figure XI [B37]) than with the absolute number of granulocytes at the start of the fever (Figures XII [B37]). Fever and granulocytopenia are also associated with intestinal injury [B31].

52. The degree and extent of leukocyte depression [J1] and bone marrow aplasia [I10] were shown to be correlated with mortality in the Japanese exposed to the atomic bombs. The chance of survival was very small in individuals having leukocyte counts of 1,000/μl

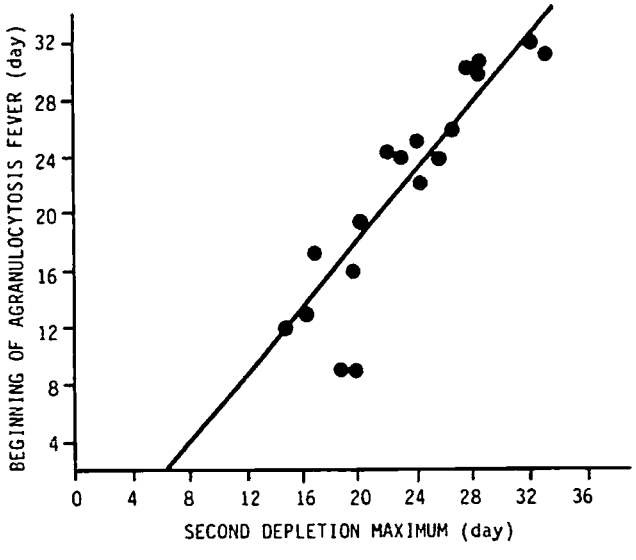


Figure XI. Time of the second depletion on the granulocyte count curve, corresponding to the beginning of agranulocytosis fever in man.
[B37]

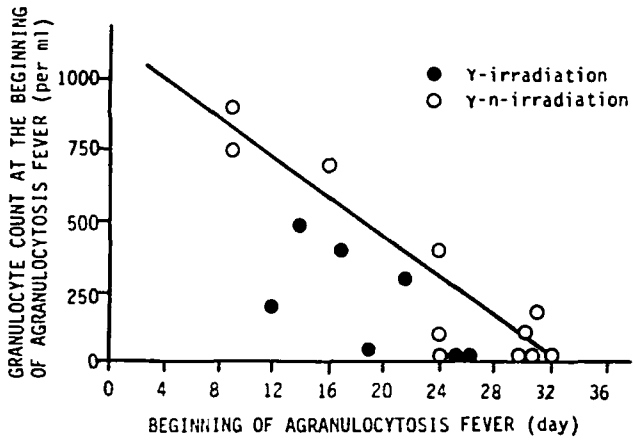


Figure XII. Granulocyte count at the beginning of agranulocytosis fever.
[B37]

in the third and fourth weeks after exposure, and the correlation of leukocyte counts with survival was best in the third week. Counts of less than 3,000/μl were not so hazardous in the fourth and fifth week as in the third week. The studies also showed that mortality was greater in Hiroshima than in Nagasaki for equivalent blood count levels. A possible reason considered at the time related to the estimated greater contribution to dose from neutrons in Hiroshima, associated with injury in other tissues contributing to death; this explanation is now unlikely because revisions in dosimetry have markedly reduced estimates of the neutron components of that dose.

53. The time course of the thrombocytopenia is broadly similar to that of granulocytopenia (Figure IX), but there is no second abortive rise. The dose-response relationship for the nadir of platelets shows a slightly more sensitive response than for that of lymphocytes (Figure X). After about 1 Gy, a decrease in platelets to 100,000 per μl is observed by day 30. The higher the dose, the earlier and greater is the reduction; after doses greater than 6 Gy, a minimum level of 10,000 per μl is observed by days 10-15. A thrombocytopenia below 30,000-50,000 per μl may be associated with bleeding, which can be prevented by transfusions of fresh platelets [F3]. Experience in treating patients suffering from bone marrow syndrome indicates that the critical level of thrombocytes requiring platelet transfusion is 20,000 per μl (see Appendix). Haemorrhages are also associated with the development of infections [J4, O5]. Owing to the long lifetime of the radioresistant red blood cells, anaemia is observed acutely only when bleeding has been substantial [B16].

54. The effects of radiation upon the immune response were reviewed by UNSCEAR in 1972 [U3]. As noted above, lymphocytes are especially susceptible to the acute effects of irradiation. Since this cell type is an integral part of the immune system, profound abnormalities of immune function would be expected as a consequence of whole-body exposure. This appears to be the case, although data pertinent to man are limited [C48, V19, M53]. The paucity of information is due in part to the fact that most of the relevant observations were made before many of the concepts that underlie current thinking on cellular immunology had been developed, in particular the concept that lymphocytes are heterogeneous in terms of structure and function. The situation is further complicated by differences in the radiosensitivities of those subpopulations of cells whose co-operative activities result in an immune response [A19, A21, A33, M53, M54, W26].

55. An increased susceptibility to infection has been well documented in persons exposed accidentally and therapeutically to doses in the low- to mid-lethal range [A3]. These infections may be caused by either endogenous (normal flora) or exogenous organisms. However, when assessing the role of an altered immune response in the presence of these infections, it is important to keep the following points in mind: (a) radiation at these dose levels may cause an increase in permeability of the vasculature, which may allow the normal bacterial flora to enter the circulation, and (b) when employed therapeutically, whole-body irradiation is generally administered to persons with haematological disorders, often in conjunction with high-dose chemotherapy and bone marrow transplantation. Even with bone marrow from an identical twin, the confounding effects of the primary disease (often leukaemia or aplastic anaemia) and other therapies on the immune response are considerable. Despite these cautions, however, there can be little doubt that whole-body irradiation causes marked acute alterations in the immune response of man.

56. Support for the above statement comes from several sources, the first of which is the whole-body irradiation of experimental animals, especially mice, whose immune response is remarkably similar to that of man. The consequences of such exposure in mice are profound, even with whole-body doses of less than 1 Gy [A19]. The effects on the immunological system are dose-dependent and may result in an augmented or a suppressed response to the same antigen, depending on the dose and the time between irradiation and the introduction of the antigen [A20]. This discrepancy in response appears to relate to differences in the radiosensitivity of effector and suppressor cells. Suppressor T cells ($CD8^+$) are more radiosensitive than helper T cells ($CD4^+$), and B cells have an intermediate sensitivity [A21, S47]. In addition, whole-body irradiation with doses as low as 0.5 Gy results in marked impairment of the normal recirculation of lymphocytes [A22, S22].

57. The second source of evidence is the results of graded doses of radiation administered in vitro. With some antigens, it is possible to evaluate the response of immuno-competent cells completely in vitro. These in vitro responses are strikingly similar to the corresponding in vivo reaction. Irradiation of one or several of the component T- and B-cell populations prior to introduction of the antigen results in dose-related abnormalities in function, abnormalities that by and large would have been predicted from complementary experiments in laboratory animals [A19, A23].

58. A third source of information is the results of partial-body exposures administered therapeutically.

Extensive immunological assessment has been carried out in persons given total lymphoid irradiation [TLI] for Hodgkin's disease [M53, V19] and in other persons irradiated regionally. Although the extent and the character of these changes appear to depend on the region of the body that is irradiated [B39], the results in general correspond to what would have been predicted from experimental animals. One of the best-studied groups of patients receiving regional irradiation are women who have received local radiation therapy for carcinoma of the breast. These and related studies support the notion that lymphocyte subpopulations differ in their depletion and repopulation after irradiation [P15, W3]. The following abnormalities were noted in individuals who had received 45 Gy regional irradiation over five weeks before or after mastectomy, in comparison with individuals treated by surgery alone [R16, W17]: (a) surface markers: there was a significant reduction in the total lymphocyte count, which returned to a suboptimal plateau by seven months after irradiation. The plateau persisted for at least 10-11 years after radiotherapy. The reduced recovery level was due primarily to a reduction in T-cells (lymphocytes binding to sheep erythrocytes and reacting with the monoclonal antibody Leu-1 (CD5)). There was a significant reduction in T-cells of the helper/inducer phenotype (detected by anti-Leu-3a (CD4)), and this persisted at one year and 10 years after irradiation. Normal numbers of T-cells of the suppressor/cytotoxic phenotype (stainable with anti-Leu-2a (CD8)) were found between one year and 10 years after irradiation. Induced IgG and IgM synthesis was also reduced after irradiation, with later recovery. In a related experiment, Job et al. [J9] showed a reduction in the helper/suppressor ratio in patients receiving adjuvant radiation therapy for primary breast cancer and in patients receiving brachytherapy and external beam radiation therapy for carcinoma of the cervix or corpus uteri. This change began during therapy and was due to a decrement in helper T cells detected by the OKTB monoclonal antibody. These alterations persisted for at least 18 weeks after irradiation. Similar observations have been made in patients receiving total lymphoid irradiation for rheumatoid arthritis [K10]: (b) mitogen and antigen responses: no significant changes in response of T-lymphocytes to PHA were found, but the reactivity to PPD tuberculin was markedly decreased after irradiation and gradually restored during the subsequent six months. The reactivity to allogenic lymphocytes (MLC reaction) was also reduced, but had reconstituted three months later; (c) cytotoxic functions: lectin-dependent cytotoxicity was unaffected by irradiation, but antibody-dependent cytotoxicity was reduced after irradiation, recovering by three years. Natural killer cell activity was unaffected when tested against one tumour cell type, but affected with another. The latter decrease was restored by three months.

D. EFFECTS ON OTHER TISSUES

1. Skin

59. Effects in skin are important. Because they are dose-dependent and because they are readily detected by eye, they can provide an approximate measure of injury with prognostic value. Attention must be paid, however, to the type of radiation used, because with higher photon energies, there is a build-up of dose in the surface layers and the maximum dose may be delivered to the dermis or deeper. In these cases, estimates of dose in deeper tissues derived from effects in the epidermis could be underestimated.

60. The thickness of human epidermis ranges from 40-50 μm on the trunk to 370 μm on the fingertips [I6, K15]. The average time for all basal cells to reach the stratum corneum was measured to be 17.7 ± 4.2 (SD) days [E6]. A review of these times at different sites in the body gave 32-36 days for the palm of the hand, 17 days for the upper limbs and 29-30 days for the lower limbs [R10]. The transit time through the stratum corneum is between six and 21 days, depending on the body site [B1]. A summary of cell kinetic data for human epidermis, averaged over various sites in the body, is given in Table 5. The hierarchy of cell population types in the epidermis is shown diagrammatically in Figure XIII.

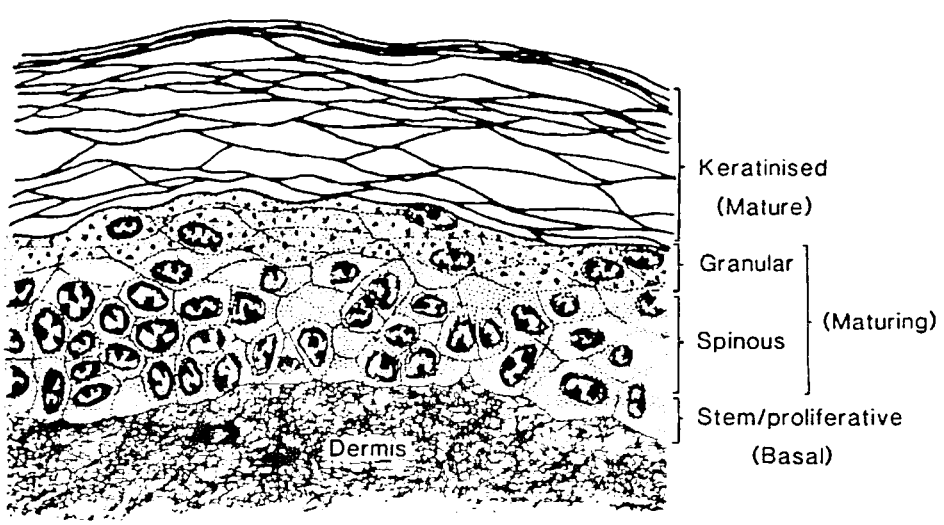


Figure XIII. Diagrammatic representation of the hierarchy of cell population types in the epidermis, drawn from a vertical section through normal human epidermis. (Adapted from [P28].)

61. The effects in skin are very dependent on the dose and on the area of skin irradiated (e.g., [A34, E12, H19, P28]). Erythema proceeds in waves. After doses greater than 10 Gy, there may be an initial phase, which reaches a peak around day 1, followed by a second wave between one and four weeks. Higher doses produce erythema of increasing severity, and the latency interval is shorter. After very high doses, erythema can appear and disappear several times. Erythema was used as a biological dosimeter in the early days of radiotherapy, and the "threshold erythema dose" varied with energy, dose rate and field size [E12]. Erythema is less easily recognized in pigmented skin and in exposed skin areas. The dose resulting in a visible erythema reaction within four weeks in 50% of individuals (not the initial transient erythema appearing within hours) after an acute single exposure with 200 kVp x rays over a 10×10 cm^2 field on the medial surface of the forearm is about 5.7 Gy [D6, L4].

62. In patients given radiotherapy to a 3 cm diameter area of the scalp with 100 kVp x rays the percentage of abnormal hairs increased between days 4 and 10 [V4]. The incidence of abnormal hairs rose above 10% only after doses to the hair roots of 0.75 Gy or more. The incidence was about 50% on day 10 after 1.5 Gy, and doses above 2.5 Gy resulted in abnormality in 100% of hairs [V4]. Temporary epilation is produced after doses of 3-5 Gy and is most severe in the second and third weeks [D2], as noted, for example, in patients receiving whole-body irradiation prior to kidney transplantation [T11, T12]. Similar time courses were observed in the survivors of the atomic bombs, and if regrowth of hair occurred it was observed by 12-14 weeks after irradiation [O5]. Epilation may be permanent after doses greater than about 7 Gy. Hair on the scalp is more sensitive than the beard or body hair.

63. Desquamation reactions appear following marked erythema, after acute radiation doses greater than about 12 Gy. The severity of the reaction depends on the anatomical location, the vascularity and oxygenation of the skin, and the genetic background, age and hormonal status of the exposed individual [R12]. Dose-time and dose-incidence relationships have been studied in radiotherapy patients receiving doses to relatively small fields. Moist desquamation is produced in 2-3 weeks in 50% of individuals after a dose of about 20 Gy to areas of 35-80 cm^2 [A4, E2, J6, L4, P2]. The maximum reaction occurs at about three weeks. After whole-body irradiation with such doses, the individual will have died from the intestinal syndrome before the desquamation reactions occur, except when the irradiation is poorly penetrating, as in the treatment of skin diseases or in direct skin exposure to short-range fallout radiation.

64. Desquamation reactions in skin are due primarily to the killing of cells in the basal layer of the epidermis and its associated appendages [P11, P28]. Measurements of the sensitivity of epidermal clonogenic cells in situ in man have been made after fractionated doses [A5] but not after single doses. However, the sensitivity has been assessed using human skin samples irradiated and assayed in vitro [D10]. The survival parameters were $D_0 = 0.7$-0.9 Gy, $n = 10$-16 (Table 1). The keratinocytes were more sensitive than epidermal clonogenic cells assayed in situ in mice or in pigs.

65. The time to full depletion of the epidermis after high doses corresponds to the transit time from the least-differentiated committed progenitor cell in the basal layer to the surface in unirradiated epidermis [P8]. This was deduced using a model applied to different types of epithelia, in which it was assumed that the clonogenic stem cells were sterilized after high doses, and also that the few divisions of committed proliferative cells, together with the processes of differentiation, maturation, and migration, were very radioresistant and hence unaffected. The normal turnover time of the epidermis would be expected to be longer than the above transit time by an amount equal to the lifetime of the stem cells in the basal layer. The time to full depletion of the epidermis after irradiation would be shortened where there is an acceleration of cell depletion as it proceeds after irradiation [P8, P28].

66. The degree of skin desquamation is markedly dependent on the area of skin irradiated. This has been studied in radiotherapy patients [C6, E2, J6, J7, M1, P2, V8], and some of these findings are summarized in Figure XIV and in Table 6. Some investigations were confounded by the use of various degrees of reaction acceptable as "tolerance" in different field sizes, e.g. [J6], as discussed in [H19]. In general, the effect of field size is similar for single or fractionated doses and can be described by either of the formulas:

$$\text{Dose} = k(\text{area})^{-0.16}$$

$$\text{Dose} = k(\text{diameter})^{-0.33}$$

where k is a constant [C7, V8].

67. The extrapolation of the above formulae to areas greater than 400 cm^2 is uncertain, because evidence for very large areas relates only to the use of lightly

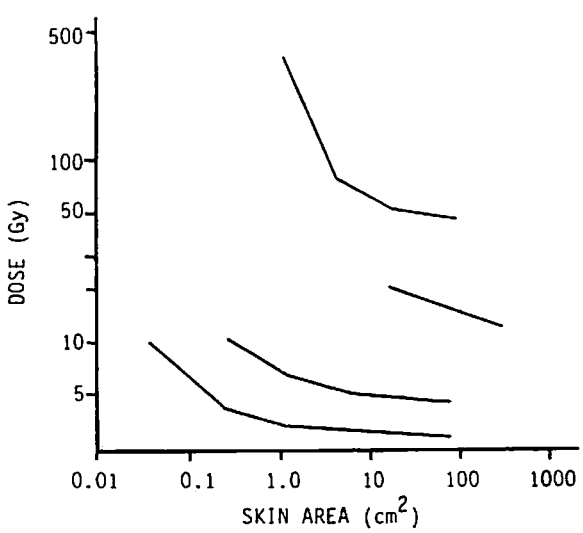


Figure XIV. Relationship between iso-effect dose for skin tolerance and field area using single doses (bottom two curves) or fractionated doses (top two curves) in man.
[H19]

penetrating electron beams for the treatment of diffuse diseases of the skin, and it is not known if these diseases predispose to increased radiosensitivity. However, it has been concluded that there is little effect of changes in area for areas above 400 cm^2 [S15]. The 50% erythema dose was estimated to be about 3 Gy for a single dose of electron radiation to the total body surface [S15, W6], corresponding to about half the dose required for areas of 100 cm^2.

68. It is the dose to the basal layer of the epidermis that determines the degree of early skin desquamation, and concomitant doses to the dermis have little influence. This was shown by experiments in pigs [M21], where various isotopes were used to irradiate to different depths a 1 cm diameter circle of skin. Surface doses to produce transient desquamation varied enormously with the energy of the radiation from the isotope but the relative doses to the basal layer, at a maximum depth of 90 mm, were much more similar (Table 7). Further experiments have been carried out with pigs, comparing irradiation by strontium-90 and thulium-170 [P32]. The percentage of the dose reaching the epidermal basal layer was similar for the two isotopes, but only about 10% of the surface dose reached the base of the dermis using thulium-170, compared with about 50% using strontium-90. These studies concluded that there was no effect of field size for epidermal reactions with thulium for areas between 5 and 19 mm in diameter, but a marked effect of field size was observed with strontium. This was considered to be due to the contribution to repopulation from hair follicles, spared more by thulium than by strontium.

69. The severity of desquamatory skin reactions may be decreased by post-irradiation treatments using corticosteroids, but erythema is not decreased [H26]. Standard procedures of cleanliness during the healing period will prevent infection. Skin haemorrhages (petechiae) in monkeys can be prevented by antibiotic treatment [S3], suggesting that infection may be involved in their initiation. However, once petechiae have appeared, their development continues because of thrombocytopenia.

70. The effects of cell depletion in the dermis are manifested later than in the epidermis and in the epidermal-associated hair follicles, primarily because there is a slower rate of cell turnover in the constituent cell types of the dermis. The dermis contains connective tissue, sebaceous glands, muscle fibres, nerve plexuses and nerve fibres, sweat glands and blood vessels. The thickness of the dermis varies markedly over the body, but is generally 1-2 mm [I6]. The effects on the blood vessels after high doses are observed initially as erythema and later as haemorrhages. Haemorrhages on the skin appear as small (petechiae) or larger (purpura) lesions. Purpura can appear as early as day 3, but the peak onset occurs in the third or fourth week after irradiation, predominantly on the upper half of the body [O5]. The duration of purpura varies according to the severity of the injury, and in fatal cases the lesions remain until death. Purpura occurs concomitantly with epilation in many cases, and it has been described in nearly all people who died 3-6 weeks after the atomic bombings [O5]. Hence, although the dose-incidence curve is not accurately known, the effect is produced by doses of 4-6 Gy.

71. Irradiation of the dermis with high doses produces a second wave of erythema (at 10-16 weeks in the pig and the rat). This is dusky red/mauve in colour and is considered to be due to damage to the deep dermal plexus of blood vessels [H18]. It is followed by dermal necrosis, ulceration and sloughing of the dermis.

72. Pain is an important feature of the exposure of skin to high doses of radiation, particularly in the case of deep lesions after exposure of the extremities. Pain is experienced during the first few days, it lasts several hours per day and it may persist for long periods [N1]. The period of maximum pain corresponds to the appearance of vascular lesions.

73. Effects on sebaceous glands were observed when treating facial acne with superficial x rays [S13]. There are 400-900 glands per cm^2 on the head. After 3 Gy, glands are reduced in size by 20% at two weeks. After 4 Gy, the glands are reduced in size by 25-50% at two weeks, with recovery by four weeks. After 8 Gy, the gland size is 50% of normal at one week, with further reduction at two and three weeks, and recovery to normal size by six weeks. After 15 Gy to a 2 cm circle, the glands are severely atrophied by two weeks, and there are only a few small glands present up to two months later [S13].

74. Interesting clinical information about the skin reaction after beta-irradiation is contained in reports on the Japanese fishermen irradiated on board the Lucky Dragon [K4] or on people irradiated in the Marshall Islands [C16]. The frequency and intensity of skin reaction were highest in individuals on the island of Rongelap, where radioactive fallout was also highest. The period of appearance of skin lesions and epilation in these people is described in [C16]. The skin reactions to beta-irradiation observed during the accident at Chernobyl are described in the Appendix.

2. Oral mucosa

75. Information relating to the effects of radiation on oral mucosa comes from observations on atomic bomb survivors [O5] and from radiotherapeutic treatments [P12, U9]. With the former, who received whole-body irradiation, oropharyngeal lesions occurred on all mucous membranes but were more prevalent on lymphoid areas than elsewhere [O5]. The tonsils, pharynx, nasal passages and tongue were frequently involved. The lesions were concomitant in many cases with epilation and purpura. The time of onset varied from a few days to five weeks, with a peak in the fourth week and a mean of 22 days. The initial symptoms were pain in the throat or gums associated with swelling and inflammation. This rapidly progressed to bleeding, ulceration and, in many cases necrosis. Ten per cent of survivors had severe ulceration. Healing was generally completed in 2-3 weeks,

with the lymphoid areas being the last to heal. Antibiotics greatly assisted healing [O5]. Necrotic gingivitis occurred in 10% of the 20-day survivors with oropharyngeal lesions in Hiroshima and in 6% in Nagasaki [O5]. This was characterized by redness, swelling and haemorrhage, and there was ulceration of the gums in fatal cases. Healing occurred slowly by re-epithelialization. The doses needed to precipitate these lesions are not accurately known, but are approximately in the range that cause epilation, purpura and some deaths, i.e., 3-5 Gy.

76. Injury to the mucosa of the mouth and throat is greatest in the cheeks, soft palate and hypoglossal area; it is less in the gums, hard palate, nose, posterior wall of the throat and tongue. Other areas, including the larynx, are less responsive [P12]. After local irradiation, accidental or radiotherapeutic, with doses of 5-10 Gy, hyperemia appears on day 1 and spreads to nearly all sections of the oral and nasal cavities. By day 4-5 there is oedema in the posterior wall of the throat, in the soft palate and the mucosa of the cheeks and nose, with pain in the mouth. These effects become more marked by day 10-15 and spread to the gums, tongue, and the hard palate. If there is necrosis, it appears at 8-12 days, followed by re-epithelialization. Recovery of the mucosal surfaces after doses up to 10 Gy occurs by 2-3 weeks after irradiation. After doses of 10-20 Gy, erythema extends to the larynx, there is virtually no latent period, there is pain and oedema in the mouth, and extensive mucosal necrosis begins on day 4-5. The recovery of the mucosa is slow and lasts for 1.5-2 months. Infectious complications occur together with local haemorrhages, and the effects are severe if there is also leukopenia [B36, G14, K7, K8, V13, V14]. Oral mucositis was noted at 5-7 days after whole-body irradiation of leukaemic patients (about 10 Gy, 0.05 Gy per minute) [D17].

77. Salivary glands are very responsive to irradiation, but recovery is possible even after high (fractionated) doses. Parotitis was observed after the Chernobyl accident, predominantly in those individuals receiving more than 6 Gy (see Appendix). This was coupled with an inability to salivate and a high level of amylase in the blood from day 1 to day 4 after irradiation. Studies in monkeys have shown that these effects in salivary glands are due largely to the high sensitivity of the serous cells, which undergo rapid interphase death after irradiation [S32]. In man there is also a loss of taste, experienced after doses as low as 2.4-4.0 Gy [C49]. In patients given daily radiotherapy, a 50% reduction in parotid gland secretion was noted at 24 hours after the first dose of 2.25 Gy, and the secretion was at negligible levels 24 hours after a second dose of the same amount [S45]. This effect was coupled with a transient tenderness and swelling of the glands, which was more severe after high doses. Doses of 15-28 Gy produced a dry mouth at 2.5 hours, on average, and pain and tenderness at 4.5 hours, reaching a maximum between 12 and 24 hours [K20]. The symptoms disappeared by seven days. In leukaemic patients treated with whole-body doses of 6-10 Gy, parotitis occurred in many cases about eight hours after the start of irradiation, and it persisted to 2-3 days [B32, D17].

3. Eye

78. The effects of low-LET radiation on the eyes of various species of mammal, including man, were reviewed by Merriam [M15]. Information concerning early effects in man derive mainly from the treatment of eye tumours by radiotherapy, and they are summarized in Table 8. For the superficial ocular tissues (particularly the conjunctiva and cornea), 10-15 kVp x rays were used; in other cases, 120-250 kVp x rays were used. Eyelid skin appears to be more responsive to irradiation than skin at other sites, the minimal erythema dose for eyelid skin was quoted as about 2 Gy, with hyperemia of the skin observed after 12-15 hours. Single doses of 3 Gy produced slight hyperpigmentation, and doses of 4-6 Gy gave marked hyperpigmentation in a few weeks. A dose of 4-6 Gy led to hyperemia after 6-8 hours, oedema and haemorrhages on day 2 and erythema by 2-4 weeks in about 50% of cases. Partial epilation of the eyebrows and eyelashes can occur [Z1]. After 6-10 Gy there may be erythema after 1-3 hours, together with oedema and pain. Partial epilation of eyebrows and eyelashes may persist for a few weeks, the eyelid skin becomes dry and atrophic, and telangiectasia develops. Necrotic changes in the eyelid skin and underlying tissues occur at doses above 10 Gy. After 4-10 Gy, keratitis is observed at days 20-40 in the upper epithelial layer of the conjunctiva. After 15-20 Gy, there is lacrymation and pain in the eyes, with irritation of the cornea and the iris. In the absence of infections these may last for three to four months.

79. A decrease in tear production was noted in leukaemic patients following whole-body irradiation (about 10 Gy, 0.05 Gy per minute) [D17]. The Japanese fishermen who received whole-body doses of 2-7 Gy and much higher surface doses from radioactive ash after the nuclear test explosion at Bikini developed acute keratoconjunctivitis by two weeks after irradiation [K4].

4. Lung

80. The pathogenesis of radiation injury to the lungs has been described by several authors [W4, V1, P5], and the radiobiology of the lungs has been discussed in [T32, U4]. The target cell population responsible for pneumonitis after irradiation remains unknown, but type-2 alveolar cells are implicated and vascular injury may be contributory [D26, T27].

81. After the thymus, the lung is the most radiosensitive organ in the thorax. Because lung tissue has a lower density than other soft tissue, a nominal 8 Gy corresponds to doses 8-15% higher to lung tissue using cobalt-60 gamma rays and 5-8% higher using 8 MV x rays [M9]. Hence 8 Gy becomes 8.6-9.2 Gy (cobalt-60) or 8.4-8.6 Gy (8 MV). The earliest signs of radiation injury in the lungs are oedema and changes in blood circulation followed by pneumonitis, which appears after a latent period of 1-3 months after doses greater than about 8 Gy. After whole-body irradiation with such doses, marrow failure may intervene before severe signs of lung injury appear, unless successful

marrow transplantation is performed. In some of the Chernobyl accident cases receiving the highest whole-body doses, the terminal period was characterized by the development of pneumonitis and pronounced respiratory insufficiency [U6]. Also, lung injury develops after high doses when the lower half of the body is shielded, as in the half-body treatment of lung metastases by radiotherapy [F12, V3].

82. Threshold doses and dose-incidence relationships for pneumonitis can be deduced from whole-body radiotherapy treatments of leukaemia prior to marrow transplantation, or half-body treatments for metastases. The effects are variously confounded by the concomitant use of cytotoxic drugs, e.g., cyclophosphamide. A survey was made of 15 centres in Europe giving whole-body irradiation before marrow transplantation to a total of about 400 patients [B32]. The dose rates ranged from 0.025 to 0.35 Gy per minute, and the lung doses from 6 to 10.5 Gy. The incidence of pneumonitis increased above 8 Gy and was dependent on the dose rate. Included in this survey were patients from the Royal Marsden Hospital in London, and a separate report described 107 of these patients with acute leukaemia given whole-body irradiation resulting in 9.1-10.5 Gy to the lungs at a dose rate of 0.025 Gy per minute. Eleven (10.3%) developed interstitial pneumonitis and five (5%) died of it [B49]. Sixty of them were irradiated and received a bone marrow transplant when they were in their first remission, and they were considered to be in a good clinical condition.

83. Irradiation to the upper half of the body was given to 245 patients for the palliation of disseminated cancer [F12]. The dose rates ranged from 0.5 to 4.0 Gy per minute. The results of these treatments, together with those given to a further 58 patients, were analysed subsequently in terms of corrected doses to the lung. Patients with significant previous and subsequent lung irradiation, with previous lung disease or with known tumour masses in the lung were excluded

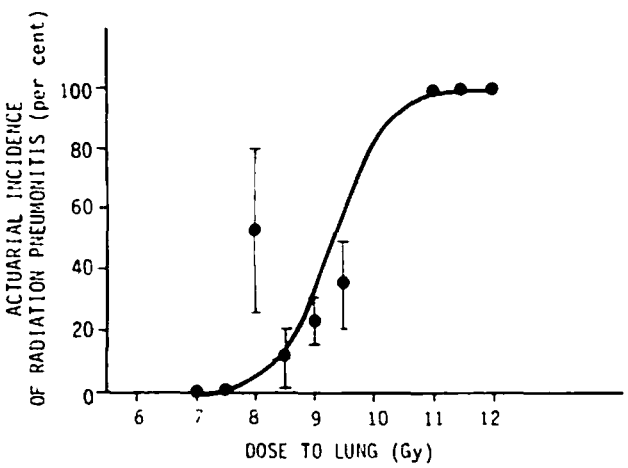


Figure XV. Incidence of pneumonitis versus dose to lung in man. Best fit sigmoidal complication curve using probit regression analysis. The point to the left of the curve was based on only four patients and hence has a large uncertainty. Based on patients excluding significant additional irradiation, previous lung disease, carcinoma in lung. Standard deviations do not apply for 0% or 100% incidence.
[V3]

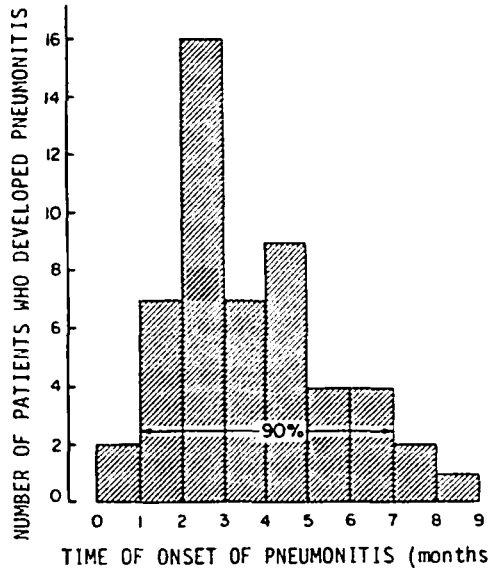


Figure XVI. The frequency distribution of the time of onset of radiation pneumonitis for 52 patients who developed the complication.
[V3]

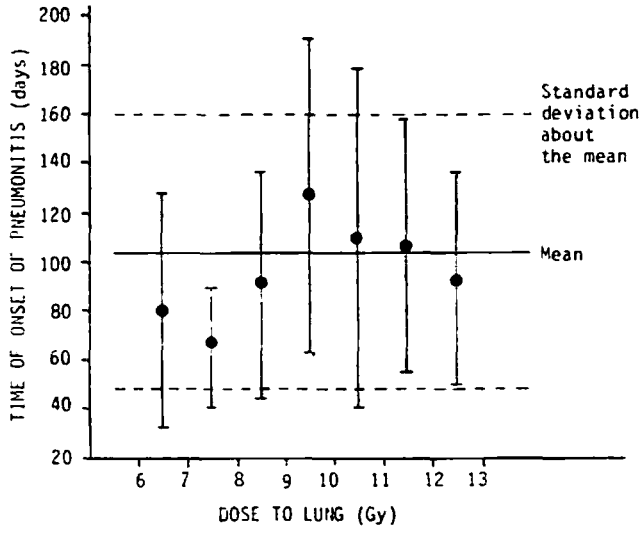


Figure XVII. Time of onset of radiation pneumonitis versus dose to the lung for 52 patients who developed radiation pneumonitis. Error bars represent standard deviations.
[V3]

from the analysis. A dose-incidence relationship for pneumonitis was presented by Van Dyk et al. [V3]. The doses to lung tissue needed to produce pneumonitis in 5% and 50%, respectively, of the cases were about 8.2 Gy and 9.5 Gy (Figure XV). The steepness of the dose-response curve could be interpreted by a D_0 value of ~ 0.6 Gy for the unknown target cells responsible for pneumonitis [T32]. The dose-incidence data are in agreement with other data for upper half-body irradiation [S17], where an incidence of pneumonitis of 10-20% was observed after lung doses estimated to have averaged 8.8 Gy [V3]. The frequency distribution of the time of onset of pneumonitis in 52 patients who developed the signs is shown in

Figure XVI: in about 90% of these patients pneumonitis appeared between one and seven months. Figure XVII shows that the time of onset was not significantly dose-dependent between 6.5 and 12.5 Gy, but this may reflect the limited sample size. Other data for humans [S17] and dogs [M40] indicate a decrease in latency interval with an increase in the dose. Lung fibrosis begins to develop at the end of the pneumonitis phase after high doses.

5. Testis

84. The kinetics of spermatogenesis in different species have been described by Bianchi [B11], and the information available on the kinetics of spermatogenesis in unirradiated man is summarized in Table 9. The testis is very responsive to radiation because the early differentiating forms of spermatogonia are extremely radiosensitive [B11, U4]. Spermatogonial cell necrosis can be detected in man at 4-6 hours after local testicular irradiation, with loss of these cells by 12 hours [H8]. The more mature cells composing the second and third phases of spermatogenesis (from preleptotene spermatocytes through meiosis and including the spermatids) are unaffected by doses below 3 Gy. These cells mature normally after such doses, and they therefore maintain the normal sperm count for about 46 days, which is the time of development from preleptotene spermatocyte to spermatozoa. The sperm count begins to drop after 46 days, approaching azoospermia at about 10 weeks after doses greater than 1.0 Gy (Table 10). Oligospermia is induced by lower doses down to 0.15 Gy. The sperm count drops earlier after doses between 1 and 4 Gy, when the spermatids also become affected. Below 3 Gy, there are no morphological alterations in the spermatozoa. Changes in sperm count at various times after different x-ray doses are shown in Figure XVIII [H8].

85. Concomitantly with the histological changes, changes in testicular hormone levels are also observed. Plasma and urinary levels of follicle-stimulating hormone increase after doses to the testis of greater than 0.1 Gy [R11], and the increase after 0.75-6 Gy may be as much as four times over the control level. Plasma levels, but not urinary levels, of luteinizing hormone are elevated after doses greater than 0.2 Gy, and the levels may be two times higher than the pre-irradiation value after 6 Gy. The levels of urinary oestrogen, urinary testosterone and plasma testosterone are not changed significantly.

86. In mice, there is a correlation between the level of stem cell killing, the sperm count at a fixed time of recovery after irradiation, the final plateau level of recovery and the length of the infertile period [M46]. In man also, the spermatogonial stem cell is considered to be the target for long-term sterility [M46]. Doses inducing temporary or prolonged sterility in men have been reviewed by UNSCEAR [U4] and ICRP [I9]. Acute doses of up to about 4 Gy cause temporary or prolonged sterility in some men [G4, H27, H29, O1]. Higher doses may cause permanent sterility, and the dose inducing permanent sterility in 100% of men is greater than 6 Gy (Table 10). After 6 Gy, long-term histological recovery has been reported at 7.5 months, with sperm appearing in seminal fluid at 24 months [R11]. The number of Leydig cells increased 90 days after 6 Gy [R11].

87. The few data for accidental exposures of the testis are consistent with the above controlled study by Rowley et al. [R11]. The acute accidents include two men who received estimated doses of 1.7 Gy and 1.8 Gy [H6]; one man who received about 3.9 Gy of mixed neutrons and gamma rays [O1]; one man who received 0.6-1.0 Gy to the testis from iridium-192 gamma rays [R7]; and 23 Japanese fishermen who received doses of 2-7 Gy over two weeks (1.5-4.5 Gy in the first day) after the nuclear explosion on Bikini Atoll in 1954 [K4].

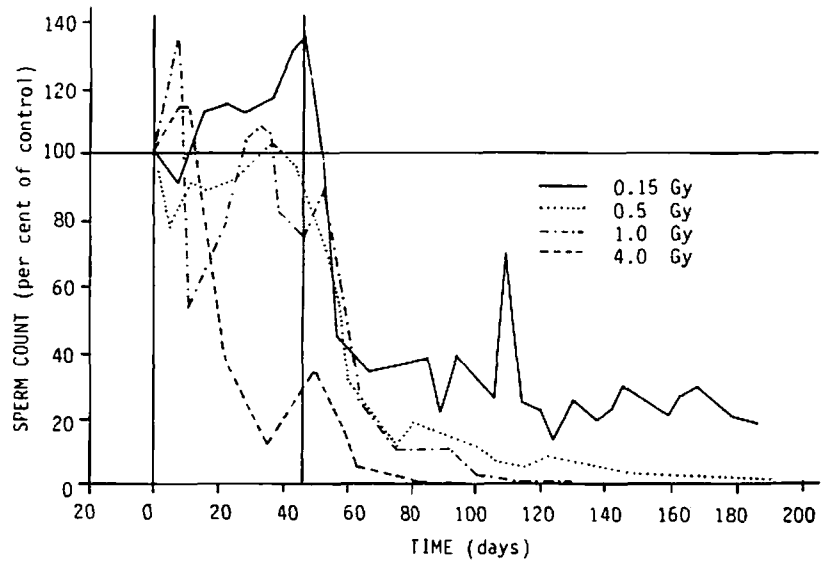


Figure XVIII. Time course of sperm-counts of normal men following exposure to various doses of 190 kVp x rays.
[H8]

6. Ovary

88. There are a total of about 2 million germ cells in the human ovary at birth, of which 50% are atretic (degenerating) [B2, B4, K3]. The mean number of follicles declines from an average of 382,000 at age 12-16 years, to 150,000 at 18-24 years, 59,000 at 25-31 years and 8,300 at 40-44 years [B14]. This decline is due to atresia since only about 400 oocytes are ovulated during a reproductive lifetime of about 35 years [B4]. Germ cells killed by radiation become pycnotic and are removed by phagocytosis within a few days. Primordial oocytes are more resistant than oocytes in growing follicles [B3]. The germ-cell content and the radiosensitivity of the ovary in different species were reviewed in the UNSCEAR 1982 Report [U4] and by Bianchi [B11].

89. Observations on ovaries and ovarian functions come from patients treated locally in the past with low doses of radiation to the ovaries to treat infertility, higher doses to induce an artificial menopause, and doses delivered incidentally during the treatment of abdominal tumours. Doses inducing temporary or permanent sterility in women were reviewed by UNSCEAR [U4] and ICRP [I9]. Acute doses of up to about 4 Gy cause temporary sterility in some women, and doses of 3 Gy up to 10 Gy cause permanent sterility in an increasing proportion of women [G4, L1, P2, P3]. Older women are more susceptible, probably because the number of follicles decreases with age.

II. DOSE-RESPONSE RELATIONSHIPS IN MAN

A. ACUTE DOSES

1. The $LD_{50/60}$

90. For many purposes, particularly the planning of protection from accidental or other acute exposures to radiation, it is customary to think in terms of the probability of survival following a dose of radiation over the whole body. One would need to know the form of the dose-response relationship for death over a given time or, at least, the value of the 50% intercept of such a curve, which is most simply and reliably defined as the lethal dose for one half of the irradiated population (LD_{50}) over the given time; say, 30 days or 60 days ($LD_{50/30}$ and $LD_{50/60}$, respectively). While the concept of LD_{50} is quite clear and widely applicable in experimental work, it is a difficult concept to appiy in the context of human irradiation. For example, the final effects will always be modified to a greater or lesser extent, depending on the cause and the conditions of exposure by the nursing or therapeutic procedures applied after irradiation. These procedures will presumably increase the value of the LD_{50} relative to its value in the absence of such procedures. Also, the state of health of the irradiated human beings may not be representative of the average state of health in the population, at least not under all conditions of irradiation. For example, the exposure of patients will produce effects that may interact with the effects of the diseases requiring irradiation or with the effects of other forms of therapy, decreasing the value of the LD_{50} relative to its value for normal individuals. The exposure of nutritionally-deprived individuals, e.g., the Japanese in the Second World War, might also produce lower values of LD_{50}. Previous estimates of the $LD_{50/60}$ are listed in Table 11, along with the factors that may increase or decrease it. Thus, when data from different groups are combined, the resulting values of the LD_{50} will, to different degrees, depart from the value obtained without complicating circumstances or treatments, and they will be affected by a variability larger than that applying theoretically to the LD_{50} of a normal human population. This variability will tend to lessen the slope of the overall dose-response curve.

91. Ideally, data on dose-mortality relationships should be derived from groups of individuals receiving doses homogeneous to within a few per cent. In practice, however, this condition is met only in the case of radiotherapy patients, and their response may be confounded by the underlying disease or by other cytotoxic treatments. In accidents, exposure is usually inhomogeneous, and this confounds the analysis of dose-effect relationships: for example, values of $LD_{50/30}$ at the midline are 20% higher for unilateral than for bilateral irradiation of large animals. Most of the individuals irradiated by the atomic bombs in Japan received unilateral prompt exposure, accompanied by fallout irradiation, and some of them were partially shielded. The population of the Marshall Islands and the Japanese fishermen exposed in the 1954 nuclear test explosion received substantial but non-lethal doses of fallout irradiation, mainly in the first two days; they are probably the largest groups of healthy individuals exposed to near-homogeneous doses, albeit over a two-day period.

92. Doses quoted in the literature are usually those at the midline, and they depend to various extents on radiation quality. Some depth-dose curves for different types of radiation are given in Figure XIX. In that figure, the depth dose is shown as tissue/air ratio, which is defined for tissue dose versus kerma at the same point. It is, therefore, independent of the inverse-square law and dependent only on photon energy, depth in tissue and field size. The most relevant parameter for death following bone marrow failure is the marrow dose, and this is usually estimated as the mean dose in an annulus between 0 and 6 or 7 cm below the body surface. It corresponds to about 0.75-0.8 of the free-in-air tissue kerma for multilateral irradiation with ^{60}Co or ^{137}Cs gamma rays [I5] (see Figure XX). The midline dose is about 10% less than the marrow dose for ^{60}Co gamma rays, and the difference is greater for less penetrating radiations, e.g., for low-energy x-ray beams or neutrons (Figure XIX). Values of midline doses related to exposure for various radiation energies and species have been published [B6].

93. The form of the dose-mortality relationship for the $LD_{50/60}$ in man is expected to follow approximately a normal (Gaussian) distribution. The relationship will be sigmoid on a linear plot of per cent mortality versus dose. There is a threshold region where doses

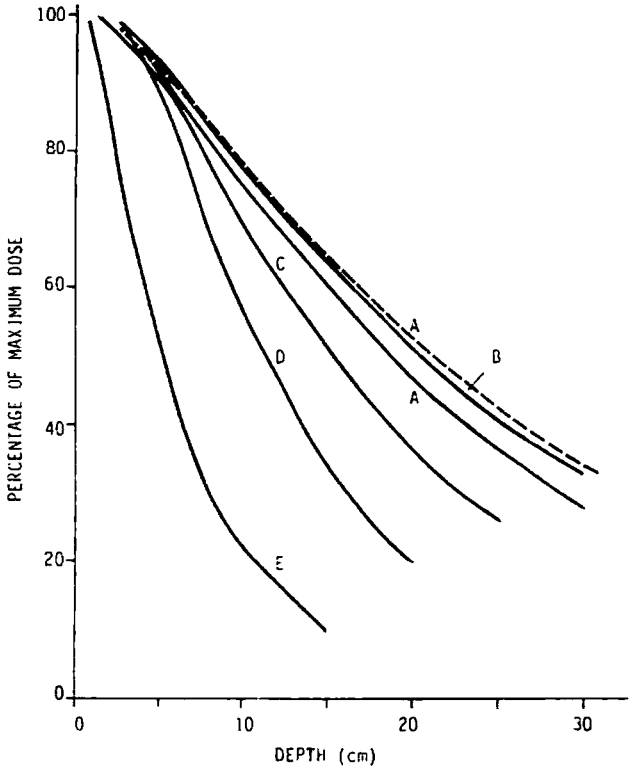


Figure XIX. Depth-dose curves for different radiation qualities. [B20, S4.] Data are tissue-air ratios (corrected for the inverse-square law) expressed as a per cent of the maximum dose:

	Radiation type	*SSD (cm)*	*Field size (cm × cm)*
Curve A	^{60}Co γ rays	80	20 × 20 and 35 × 35
Curve B	4 MV x rays	Infinite	20 × 20 or 35 × 35
Curve C	^{137}Cs γ rays	40	20 × 20
Curve D	230 kVp x rays	50	20 × 20
Curve E	^{235}U fission neutrons	500	6 × 8

cause no mortality, followed by a sharp increase in mortality with progressively higher doses, reaching a plateau at 100% mortality after still higher doses. Doses causing very little mortality are generally quoted in the range LD_{1-10}, and those causing high mortality in the range LD_{90-99}. To estimate these doses directly would require analysing large groups of individuals exposed homogeneously to the same dose. For example, with 100 individuals, the accuracy of estimates for 10% and 90% mortality would be, respectively, 3% and 30% of the mean (binomial standard sampling error). With 1,000 individuals, the accuracies would be 1% and 19%, respectively. Since there is no experience with such large groups, the doses must be estimated from dose-response relationships, where the most accurate parameter that can be calculated is the $LD_{50/60}$. These doses apply to the average individual in a population and not to a specific individual, who may have a response different from the average. The $LD_{50/60}$ will be considered first. As has already been noted, previous estimates of $LD_{50/60}$ reported in the literature are given in Table 11.

94. The $LD_{50/60}$ has been estimated from the data on mortality following the atomic bombings of Japan in the Second World War. A value for $LD_{50/60}$ of 1.5 Gy (marrow dose) has been deduced for people exposed inside Japanese-style houses at Hiroshima [R20]. This was calculated by first ascertaining the distance from the hypocentre at which there had been 50% mortality, and then converting this distance into dose. The distance was deduced to be 892 ± 11 m from a survey of 201 documented individuals who died between one day and two months after the explosion. This distance was given later as 887 m [H44]. At the distance of 892 m, revised estimates of free-in-air tissue kerma were used [K16], together with shielding factors [E9], to calculate a cumulative marrow dose of 1.5 Gy from gamma rays and neutrons. Similar calculations of dose at other distances enabled a dose-mortality curve to be deduced. The revised dosimetry (DS86) has caused the estimate to be increased from 1.5 Gy to 1.8 Gy [F15]. Further, a total dose of 2.4 Gy at 892 m was quoted in an analysis using individual transmission factors [F15]. A recent re-assessment of such data [F15] concerning deaths versus distance at exposure has produced a value for $LD_{50/60}$ in the range 2.7-3.1 Gy (see Table 11 and Figure XXI).

95. The mortality in known numbers of individuals exposed to the bomb irradiation at particular places is being further studied [e.g., F15]. For example, a

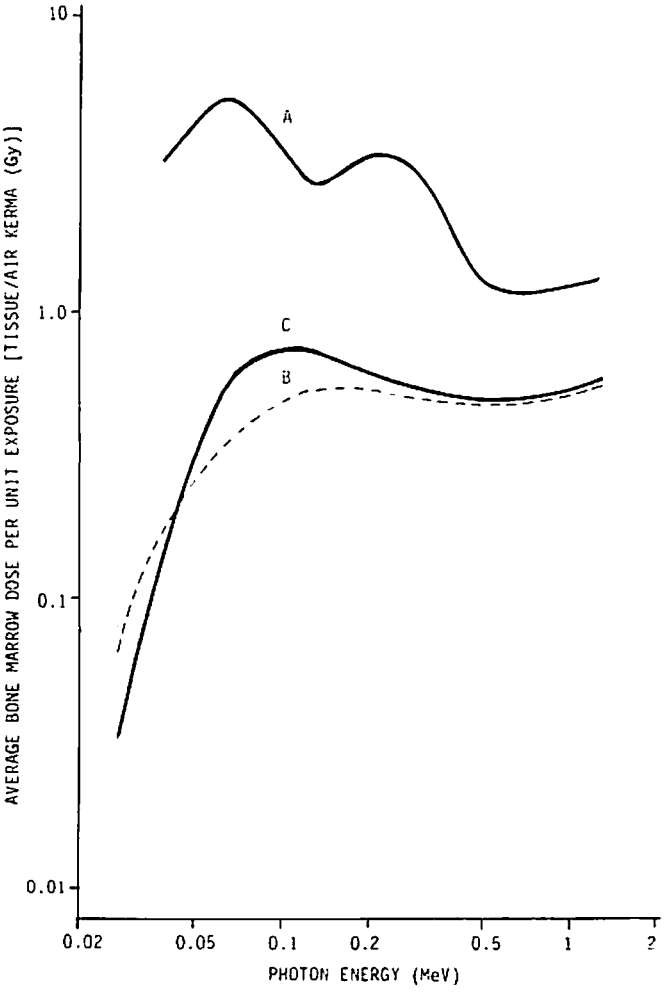


Figure XX. Average dose in bone marrow per unit exposure measured by a personal dosimeter on the front of the trunk (curves A and B) and per unit exposure measured in free air at the position of the centre of the body (curve C). Curve A: irradiation from the back only. Curve B: irradiation from the front only. Curve C: rotation during exposure, simulating irradiation from all sides.
[15]

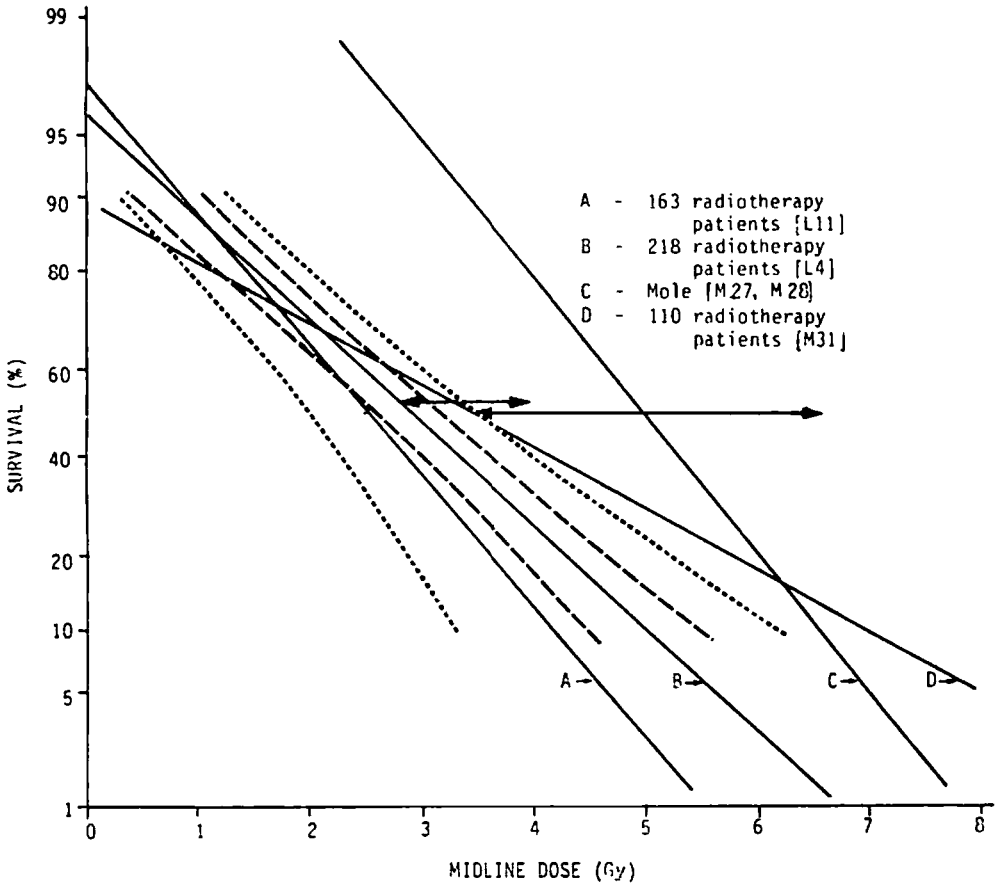


Figure XXI. Estimates of dose-survival curves for man, at 60 days. Curves A and B: Standard error limits (dotted curves at both sides of curve A, dashed curves at both sides of curve B) calculated using probit analysis; Curve C: The line is drawn assuming a coefficient of variation of 0.24, derived using several species of large animal [M28]. The left arrow extends to approximately the LD_{50} calculated using lower levels of survival at the 90% Poisson probability level, and the right arrow is included speculatively for completeness. Curve D: Arrows denote standard error limits. The dose-survival curve estimated from the population in Hiroshima receiving atomic bomb irradiation is expected to lie in the range between curves A and B.
[L4, L11, M27, M28, M31, R20]

group of 159 labourers were exposed when shielded by wooden buildings about 1,000 m from the hypocentre at Hiroshima, and of these 58.5% died between day 20 and 38 [O5]. Using the revised doses, as in the preceding paragraph, the tissue kerma of about 2.4 Gy multiplied by a factor of 0.79 gives marrow doses of 1.9 Gy, and possibly 2.1 Gy if prompt and delayed radiation components are considered separately [F15]. Also, of 193 workmen exposed unshielded at 1,000 m from the hypocentre, only 10 survived a marrow dose currently estimated to have been about 3.3 Gy [F15].

96. Other groups of individuals were exposed inside concrete buildings. Ninety 15-year-old girls were exposed in the Central Telephone Office of Hiroshima at 550 m from the hypocentre. Of the 59 who survived to 24 hours, 29 (49%) died between one and ten weeks after exposure. The majority (20) died in the fourth, fifth and sixth weeks. From measurements made years later of chromosomal aberrations in the T-lymphocytes in the survivors [F15], the dose was estimated to have been 6.5 Gy. Although this is similar to the value of 6.0 Gy according to the T65D estimates, the revised estimates of dose are lower, perhaps as low as 4 Gy [F15]. None the less, the survival rate of these girls was higher than that of adults irradiated in other buildings who had apparently lower doses.

97. A recent detailed analysis of weighted data concerning deaths versus distance from the hypocentre, including those occurring on the first day after exposure, has given a value for $LD_{50/60}$ of 2.1-2.5 Gy marrow dose, the value depending on the mathematical model used to fit the data. A probit fitting of the data gave a value for $LD_{50/60}$ of 2.2 Gy, and for $LD_{95/60}$ of 5.8 Gy [F15]. It was considered that the value of $LD_{50/60}$ may be too low by up to 17% because of the contributions to mortality estimates from deaths on the first day and of the severely injured. This was estimated in a separate smaller study of 184 individuals, 84 of whom died on the first day, where the $LD_{50/60}$ was calculated using probit analysis to be 3.2 Gy or 2.6 Gy marrow dose when the above early deaths were respectively excluded or included. Hence the true value of $LD_{50/60}$ may be around 2.5 Gy or higher. The true value of $LD_{05/60}$ is even more uncertain. It may also be higher, or lower down to 4.5 Gy [F15]. In view of these uncertainties, it is concluded that the dose-survival curve for the Japanese exposed to atomic bomb radiation in Hiroshima is

likely to be similar to curves deduced for ill radiotherapy patients receiving whole-body irradiation (in the range of curves A and B, Figure XXI).

98. There have been many radiation accidents involving single individuals or a few individuals, often with very inhomogeneous doses from gamma rays or x rays, or mixed radiation, including a neutron component. These accidents have been summarized by several authors [B7, B31, D24, D25, L12, M19, U4]. The most comprehensive listing is probably the REAC/TS Radiation Accident Register [L12]. Between 1944 and October 1983, 188 accidents were recorded involving 928 persons, 22 of whom died from acute effects [F10]. One hundred and forty-four individuals received whole-body doses greater than about 0.25 Gy, and eight of these died; 46 received, in addition, local irradiation with doses greater than 6 Gy, and eight of them died; 62 received high internal doses, and four of them died; 110 Marshall Islanders received both internal and external irradiation, and one of them died. In March 1987 these numbers were updated to 284 accidents involving 1,358 persons, 33 of whom died from acute effects (excluding Chernobyl) [L38].

99. Before the accident at Chernobyl ([U6] and the Appendix), the accidents involving the largest number of individuals, and therefore the most useful for analysis, were those in 1958 at Oak Ridge, United States [O2, H23] and at Vinca, Yugoslavia [H22, I1, J4, M49]. The doses received by these individuals are still a matter for debate; some recent estimates are reported in Table 12. The various estimates depend on the assumptions about the position and orientation of the individuals, and the dose and RBE of the neutron component. None the less, there was only one death, individual V at Vinca, who received a marrow dose estimated recently to have been approximately equivalent to 4.5 Gy of low-LET radiation (Table 12). Although he had marked haematological responses, these were not the primary causes of death. Two of the individuals irradiated at Oak Ridge received antibiotic treatment for respiratory infections, whereas the Vinca cases had barrier nursing, and a series of antibiotics, platelet and red cell concentrates, and later marrow cells. In one report, recalculation of the doses broadened the possible ranges of dose so that they overlap, depending on the uncertain aspect of exposure, particularly at Oak Ridge, i.e., from the side or from the front [B7]. Another report concluded that the data from Oak Ridge were more reliable than those from Vinca, because in the latter accident the exposures may have been more inhomogeneous [M19]. From a re-analysis of the measurements of sodium activation, it has been suggested that the doses at Oak Ridge should be increased by about 10% and at Vinca, by about 30% (column 8, Table 12) [M26]. This could remove the apparent difference in clinical effect for estimated equivalent doses in the two accidents in earlier reports.

100. In the accident at Chernobyl, described in the Appendix, 115 persons received doses ranging from approximately 1 to 16 Gy (Table A.3). In most cases the individuals received antibiotics and were hospitalized, if necessary under aseptic conditions, and platelet and red-cell infusions were administered when considered necessary. Thirteen patients received allogeneic marrow transplants and six received embryonic liver transplants. In the lowest dose group, 31 individuals received relatively uniform bone marrow doses of gamma-irradiation of approximately 1 to 2 Gy. In the second dose group, 43 individuals received marrow doses between 2 and 4 Gy; in many cases higher doses to the skin from beta-irradiation were also received. None of the patients of this group died up to 60 days after irradiation, but one died at day 96. A further 21 persons received marrow doses between 4 and 6 Gy. Seven of them died between 16 and 48 days after irradiation, and six of these seven had severe skin injuries, which contributed greatly to their death. In the highest dose group, 20 individuals received doses between 6 and 16 Gy. One person died at day 10 after irradiation, 17 died between 14 and 48 days, and two died at days 86 and 91, respectively. The individuals in this dose group had severe skin injuries to 40-90% of the body that were probably lethal, as well as severe signs of radiation sickness (Table A.7). These observations, in particular the survival of 43 individuals in the dose group 2 to 4 Gy surviving more than 60 days, suggest that the $LD_{50/60}$ for this irradiated population was at least 4 Gy.

101. Various groups of cancer patients have been given acute whole-body irradiation. Some were relatively ill people with advanced disseminated cancer and others were relatively healthy individuals irradiated while in remission or when bearing metastasizing solid tumours. A group of 19 children and adolescents with Ewing's sarcoma and one with leukaemic infiltration of bone received 3.0 Gy from whole-body irradiation with cobalt-60 gamma rays given in 15 minutes (dose homogeneity to within ± 10%) [R6]. None of these 20 individuals died within one year. They were given antibiotics when infection arose, blood infusions at about day 30 to replace haemoglobin, and barrier nursing during the phase of pancytopenia. According to Poisson statistics, zero deaths in a sample of 20 individuals might be observed 1 in 20 times (i.e., a 5% probability) if the true number of deaths on average among many such samples was 3.7; that is, if the true mortality was 3.7/20 = 19%. Hence it is possible that, although no mortality was observed in this particular sample, the average mortality level characteristic of this 3.0 Gy dose could be as high as 19%, but not as high as 50%.

102. An attempt was made to extend this type of analysis to a total of 27 individuals, by including subjects A, C and D in the Y-12 accident, and subjects V, M, D and G in the Vinca accident [M28]. Estimates of the total (n + γ) marrow doses that were used ranged from 2.8 to 3.3 Gy (column 7, Table 12). The one death (case V at Vinca) was attributed to marrow failure for this exercise, in order to provide a maximum value to the observed mortality. Using these 27 individuals, the statistical exercise in the preceding paragraph gives virtually the same result, with the possible average mortality being 21%. A further point is that if the true average mortality was 50% at these estimated doses of about 3 Gy, as is suggested by an analysis of mortality in radiotherapy patients (see the

next paragraph), or more (column 8, Table 12), there would be a 5% probability of as few as six deaths out of a random sample of 27 individuals, in contrast with only one death observed. Hence the data for the Ewing's sarcoma patients, with or without the inclusion of these accident cases, are inconsistent with an $LD_{50/60}$ as low as 3 Gy. The revisions in the dosimetry for the accident cases that increase their respective doses [M26] strengthens this conclusion, as does the survival to 60 days of all 43 individuals receiving doses estimated to be between 2 and 4 Gy in the Chernobyl accident (Table A.3).

103. One group of 163 relatively ill cancer patients was irradiated to the whole body with acute doses of low-LET radiation [L11]. The estimated dose (with its standard error) giving 50% deaths within 60 days was 2.5 (+0.98 −0.51) Gy, calculated using a normal distribution, and 2.35 (+5.06 −0.87) Gy using a log-normal distribution. The data were corrected for a death rate of 4% in unirradiated patients, and average doses were given for a 26 cm diameter sphere in the epigastric region. A similar analysis of 218 patients irradiated within an overall period of one day, gave an $LD_{50/60}$ of 2.86 ± 0.25 Gy [L4]. These two calculated dose-mortality curves (A and B) are shown in Figure XXI.

104. An analysis of 110 patients receiving whole-body irradiation from 1 to 10 Gy, either for various cancers and leukaemia or prior to kidney transplantation, indicated an $LD_{50/60}$ of about 4.0 Gy [M31]. The Committee's probit analysis of these data produced an $LD_{50/60}$ of 3.4 ± 0.5 Gy (curve D, Figure XXI). The data for the patients with malignancies were not significantly different from the data for the (fewer) patients with kidney debilities.

105. Smaller groups of patients have also been given whole-body doses of up to 3 Gy, without bone marrow transplantation. For example, one patient with metastatic bronchogenic carcinoma given about 3.8 Gy (midline dose) using ^{60}Co died on day 20 after irradiation, and one with generalized neuroblastoma given about 2.6 Gy survived to 4 months after irradiation [K18]. Of seven patients with advanced colon and lung cancer irradiated with 2.0 Gy (midline dose) using ^{60}Co, two died within 60 days (at 28 and 56 days) [S27]. Three patients, in a series of 18, in remission from acute leukaemia were given 3 Gy midline dose using ^{137}Cs, and they survived more than 60 days [K23]. They received antibiotic therapy and transfusions of platelets and red cells when considered necessary.

106. Since the $LD_{50/60}$ for ill cancer patients is confounded by their disease and other concomitant treatment, it may be lower than the $LD_{50/60}$ for healthy people. The data in the last three paragraphs suggest that for ill cancer patients treated with conservative supportive medications and blood-cell infusions when necessary, the $LD_{50/60}$ is about 3.0-3.5 Gy (marrow dose).

107. The cancer patients considered relatively healthy at the time of irradiation were those with Ewing's sarcoma. Whole-body irradiation was given to these children and adolescents to sterilize the metastases. Three patients with localized disease given 3 Gy (midline dose) survived more than 60 days without needing supportive medications [M34]. Ten patients with localized disease given 3.0 Gy (midline dose) survived more than 60 days [J17]. A larger series of 20 patients was described, one of whom was diagnosed subsequently to have had instead a leukaemic infiltration of bone [R6]. All 20 survived more than 60 days. This indicates that the $LD_{50/60}$ of relatively healthy people is greater than 3.0 Gy, although it is not known if these young people were more resilient to irradiation than adults. The apparently high doses tolerated by the schoolgirls irradiated by the atomic bombs [K17] would support this idea.

108. Attempts have been made to use experiments with animals in order to predict the dose-mortality relationship for man. Two similar approaches have been described. The first approach [L4, M28] relies on the similarity of the coefficient of variation (CV) of the LD_{50} [i.e., the inverse slope (probit width) divided by the mean] among different species of large animals. The CV for irradiated cancer patients was 0.58, which is much larger than the CVs calculated for dogs (0.15) and monkeys (0.21) [L4]. This greater variability was attributed to the marked heterogeneity of responses among patients. The mean CV for dogs and monkeys (0.18) was applied to the data for 218 irradiated cancer patients [L4], where the $LD_{50/60}$ was 2.86 ± 0.25 Gy, to calculate the doses for 10% mortality (2.2 Gy) and 90% mortality (3.5 Gy). Mole [M27, M28] calculated the weighted mean CV for five different species of large animal (dog, sheep, goat, pig, donkey) to be 0.24. This value was used, together with pertinent but sparse information for mortality in "healthy" humans, to deduce a value for the LD_{50} in man of about 5 Gy (Figure XXI). The information just referred to came from the 27 individuals described in paragraph 102 [M28].

109. The second approach was to take the ratio of the doses that produced measurable, very low or very high mortalities [B6]. The ratio of LD_{95}/LD_5 or LD_{90}/LD_{10} from 34 experiments in six species of large animal was close to 2.0. This ratio was used, together with the data for the Ewing's sarcoma patients, to consider the LD_{90} or LD_{95} for man. The two approaches are consistent with one another, and they suggest that the dose that would kill "few" healthy humans is about 3.0 Gy, the dose that would kill "most" is about 6.0 Gy [B6] and the LD_{50} is 4.5-5.0 Gy [M27, M28].

110. Estimates of dose-survival curves for various animal species are given in Figure XXII. Data from many published experiments were reviewed by Baverstock [B6], and were re-analysed to obtain a mean curve for each species [T24]. Doses in each experiment for a given species were multiplied by the ratio of the LD_{50} for that experiment and the mean LD_{50} for all experiments. This assumed that variations between experiments were due to dose-modifying influences, e.g., to changes in dose rate or LET. The data for mice were reviewed and analysed separately [H32].

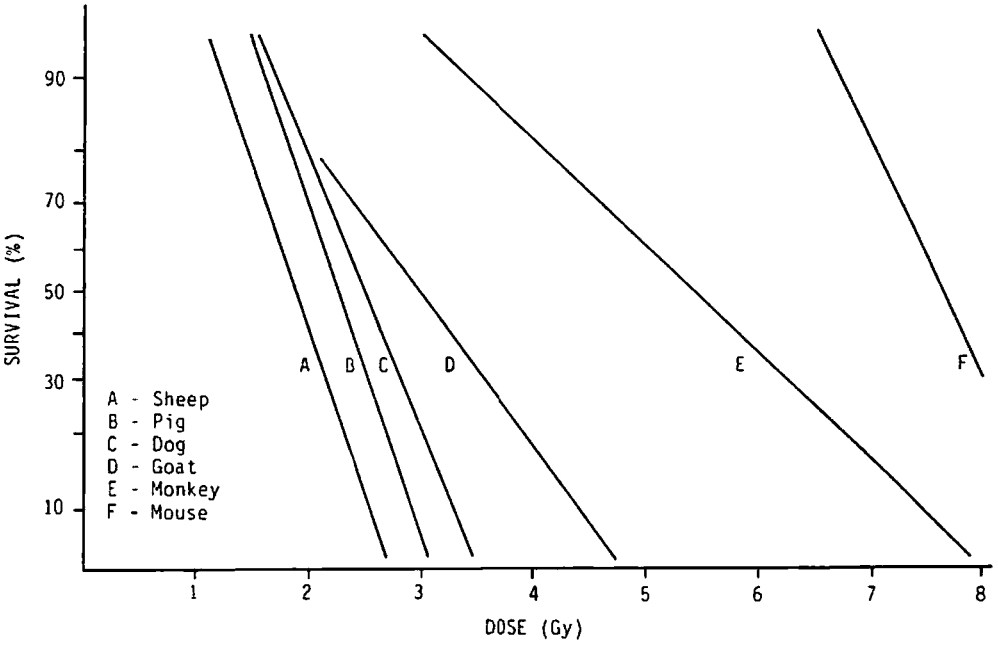


Figure XXII. Midline dose-survival curves calculated from various published experiments using different species of animal irradiated bilaterally.
[T24]

111. In the data for large animals reviewed by Baverstock [B6], lower slopes correspond with higher values of LD_{50} (see curves for goat and monkey in Figure XXII). This indicates that heterogeneity in the irradiated population is greater for species showing a higher LD_{50}, possibly because variations between individuals are greater in species with a high LD_{50} or because the sensitivity of their target cells is less. Evidence for the latter possibility is that the D_0 for granulocyte-macrophage colony-forming cells is generally reported to be much lower in dogs (~ 0.7 Gy) than in mice (~ 1.8 Gy) or in humans (~ 1.5 Gy) [H11]. Data are not available for other species.

112. It is concluded from the preceding discussion that the $LD_{50/60}$ for acute irradiation is likely to be around 3.0 Gy marrow dose in the case of humans receiving no or little medical treatment, as deduced from recent analyses of the results of the atomic bombings in Japan. A similar value pertains to some groups of ill cancer patients receiving good medical care. The $LD_{50/60}$ for healthy humans receiving good supportive medical treatment after irradiation (e.g., barrier nursing, antibiotics symptomatically and blood cell infusions) is likely to approach or equal 5.0 Gy, in particular for children. This is deduced from the lack of mortality in the young and relatively healthy Ewing's sarcoma patients given 3.0 Gy marrow dose, the presence of only one death out of the seven individuals receiving the highest doses in the Vinca and Y-12 accidents; the survival of all 53 individuals receiving doses of 2-4 Gy and of 14 out of 21 individuals receiving 4.2-6.3 Gy in the Chernobyl accident; and the data on dose-response relationships available for other large animals. It should be noted that the $LD_{50/60}$ can be further increased markedly by successful marrow transplantation, probably up to about 9 Gy acute single dose. After these higher doses there may be some cases of pneumonitis occurring in the second month, unless the lungs are shielded. At even higher doses (>10 Gy) acute gastrointestinal injury will become more prevalent.

113. A summary of the effects and their time courses after whole-body irradiation of man, prepared by the Committee, is given in Table 13. The Table lists possible therapies for the responses. More detailed summary tables of symptoms and signs after various ranges of dose are available, for 0.5-1.0 Gy, 1.0-2.0 Gy (Table 14), 2.0-3.5 Gy (Table 15), 3.5-5.5 Gy (Table 16) and higher doses [Y7].

2. Doses for very low and very high mortality in man

114. The dose-mortality curves for ill cancer patients are shown in Figure XXI, together with a curve for healthy humans described by an $LD_{50/60}$ of 5.0 Gy and a coefficient of variation of 0.24 [M27, M28]. The slope of the latter curve is consistent with the conclusion of Baverstock [B6], using data for various species of large animals, that the ratio of doses (LD_{90}/LD_{10}) or (LD_{95}/LD_5) was about 2. Also, the probit width, for this curve, of 1.20 Gy (i.e., 5.0 × 0.24 Gy) would correspond to a D_0 value for the bone marrow target (stem) cells of 1.20/1.2, or 1.0 Gy, using the Poisson model described by Gilbert [G3]. From Figure XXI it may be seen that a dose of 2 Gy would be unlikely to kill more than about 1% of a healthy population (curve C). This is compatible with the Chernobyl experience (Table A.3) but not with the atomic bomb data [F15]. By contrast, a dose of 2 Gy could kill up to 30-40% of a population of very ill cancer patients (curves A, B and D). Similarly, a dose of 7 Gy would probably kill about 95% of healthy people (curve C) but 5-6 Gy to ill cancer patients could probably achieve the same level of mortality (curves A and B).

3. Geometry of exposure and depth-dose distributions

115. Large animals irradiated unilaterally exhibit greater LD_{50} values than those irradiated bilaterally, by about 20% for dog, sheep, pig and goat [M28] (Table 17). Further information on the effect of exposure geometry is that the LD_{50} for goats irradiated dorsally is 0.63 of the value for ventral irradiation [B7]. Also, higher values for the LD_{50} of goats are obtained when parts of the vertebrae are specifically shielded from direct dorsal irradiation [B7]. Similar effects would be expected for man, but no data exist on this subject.

116. The interpretation of the differences in LD_{50} with the direction of exposure relates to the exponential relationship between cell survival and dose. Irradiation of a cell population with a non-uniform dose is always less effective than a homogeneous irradiation with the average dose of the distribution [B18]. In the case of bone marrow, the effects depend on the depth-dose curve for the particular radiation used and the distribution of active bone marrow along such a depth-dose curve. Models for these effects have been described [B18, B19, T3].

117. Depth-dose distributions are shown in Figure XIX for a variety of radiation qualities [B20, S4]. These are expressed as percentages of maximum tissue-air ratios, independent of the inverse-square law, and measured for large radiotherapy fields (> 20 cm $\times$ 20 cm) and source-to-surface distances (SSD) greater than or equal to 40 cm. No ideal comparison exists wherein a full range of radiation qualities has been used with the same large field size and the same SSD. Hence the curves shown in Figure XIX would change slightly, depending on the particular irradiation set-up. For 4 MV x rays, the highest energy considered, the surface dose for well-collimated beams and short SSD is between 40% and 50% of the maximum dose. For low energies and non-collimated beams, the surface dose is a greater percentage of the maximum dose. With non-collimated beams and very long SSD, the surface dose may be equal to the maximum dose. For fission neutrons, a significant build-up effect would be unlikely.

118. The average dose in bone marrow per unit exposure is shown in Figure XX [15]. These curves were obtained by calculation and measurement using a phantom. Similar curves are available for the average doses in the intestine and the gonads [J10].

119. With low-energy x rays there is an additional dose at bone surfaces due to the greater photoelectric effect with the high-atomic-number elements (e.g., calcium and phosphorus) in bone. The greater dose depends on the x-ray energy and on the thickness of the bone, and the effect decays within a few hundred microns of the bone surface. The increase in dose is as great as 50% on the bone surface using 250 kVp x rays [E5], and on average about 20% for a single layer of cells situated against the bone surface. This effect in the mouse would increase the dose to the marrow on average by about 9% compared to the dose in soft tissue remote from bone, and it should be reflected in the $LD_{50/30}$ if the concentration of marrow cells critical for survival is constant in all regions of active haemopoietic tissue. Since higher concentrations (about twice as high) of critical stem cells have been detected close to bone surfaces in the mouse [L7], the above figure of 9% may be slightly higher. No information is available on the measurement of such effects in large animals, apart from the lower concentration of granulocyte-macrophage precursor cells close to bone surfaces in human ribs [T33].

4. Dose inhomogeneity, shielding and bone marrow distributions

120. The effect of dose inhomogeneity on the haematological response and survival in rodents and dogs has been reported in papers submitted by the delegation of the USSR [D28]. In mice, the $LD_{50/30}$ was about 5.5 Gy for whole-body irradiation, about 14.5 Gy when only the front half of the body was irradiated and about 8 Gy when only the rear half was irradiated. The corresponding values for dogs were about 2.8 Gy and 3.8 Gy. Different critical organs were probably responsible for death after these types of irradiation. Thus, for both mice and dogs, larger average doses to the body could be tolerated when only the front half was irradiated, and smaller average doses when only the rear half was irradiated, compared to uniform irradiation. Also, these average dose differences were reduced when both halves of the body were irradiated.

121. When small portions of the body containing active marrow are shielded during irradiation, the LD_{50} can be markedly increased. This would also apply to some degree in the case of non-uniform irradiation. Shielding the right legs of mice increased the $LD_{50/30}$ from slightly less than 7 Gy without shielding (7 Gy gave 70% mortality) to about 12 Gy [C2]. Shielding the right leg below the hip joint gave 73% survival at 30 days after 10.5 Gy, in contrast to 0% without such shielding [D1]. Shielding the leg only below the knee joint, or below the tibia, did not cause survival to drop below 70% [D1]. These phenomena are due partly to the migration to and repopulation of irradiated marrow by progenitor cells from the shielded marrow and partly to the ability of the shielded marrow to increase its normal rate of producing maturing haemopoietic cells. In mice, a persistently reduced complement of only 10-20% of stem cells remaining during chronic irradiation [L2] or after repeated irradiation [H14] can maintain a normal output of mature haemopoietic cells into the blood for many months.

122. In dogs, shielding the skull reduced lethality after 4-5 Gy from 100% to 20%, and shielding sternum, pelvis or skull doubled the LD_{50} [A31]. Also, shielding, separately, the head and neck, chest, abdomen or pelvis gave no deaths in separate groups of 20 dogs each given 6 Gy, a 100% lethal dose if given to the whole body [L31]. Shielding smaller volumes of marrow in dogs has also been shown to markedly increase survival [D28]. Shielding one or two vertebrae was found sufficient to protect dogs from an otherwise fatal exposure to radiation [S41]. Shielding the limb

epicondyle resulted in 100% survival after doses three times the $LD_{50/60}$, but shielding only the third and fourth ribs was insufficient [C34].

123. The above data suggest that in man, the shielding of perhaps as little as 10% of the active marrow, while the remainder of the body receives a dose close to the $LD_{50/60}$, may reduce the number of deaths to zero. The efficacy of shielding different parts of the body in man depends on the distribution of active bone marrow.

124. Various estimates of the percentage of active marrow residing in the different bones of man have been calculated from histological measurements using ^{59}Fe uptake. The values for humans aged around 40 are compared with those for other species in Table 18. Some of the values for humans were calculated from the absolute weights of total marrow in the bones of 11 cadavers [M12]. These weights separately for each cadaver were multiplied by the proportion of marrow that was active. This proportion has been estimated by various investigators on the basis of marrow cellularity and uptake of ^{59}Fe, and the values given by Cristy [C12] for humans aged around 40 were used by Woodard [W8]. The absolute weight of active marrow in a given bone was expressed as a percentage of the total weight of active marrow. Finally, the average of these percentages was calculated over the six males and five females investigated. The averages differ in many cases from the values presented by Ellis [E4], as used by ICRP [16] for reference man. The largest differences are evident in the values of 3.9% for the sternum (3.6% in females), given as 2.3% by Ellis [E4], and 7.7% for the sacrum (7.4% in females), given as 13.9% by Ellis [E4]. Also, the percentage of active marrow in the total marrow, 27.5% (28.5% in females), was given as 50% by ICRP [16]. The values from Woodard [W8] probably apply quite well for ages above 20 years but not so well for younger people, because the skull has a higher proportion of active marrow than other regions of the skeleton [C12]. In diseased patients there may be significant extramedullary haemopoiesis, which would modify the normal distribution.

125. The distribution of active marrow in Japanese adults was measured by weighing the marrow in each bone of seven male and three female cadavers, aged between 26 and 41 years [M30]. The red-marrow component of the mean weight of marrow in each bone was assessed histologically. The values are given in Table 18. These values are the mean for each bone, an approach similar to that used by Ellis [E4], rather than the mean of the proportions for each individual, the approach preferred by Woodard [W8].

126. The less-detailed distributions in man measured using ^{59}Fe uptake and scanning techniques [S43, A32] are in broad agreement with an anatomically derived distribution [M30]. A particular difference between these distributions and others based on earlier anatomical assessments [W8] is the significant amount of active marrow in the lower limbs (as is found in other species, Table 18); 8.7% [S43], 7.9% [A32] and 10.6% [M30], versus 0% [W8].

5. Radiation quality

127. Most accidental human whole-body exposures to high-LET radiation have involved both fission-spectrum neutrons and gamma rays. Exposures from the atomic bombings also involved gamma rays and fission neutrons, but the revisions in dosimetry [K16] have reduced the estimate of the neutron component, particularly at Hiroshima (Figure XXIII). For example, at 890 m from the hypocentre in Hiroshima, where about 50% of individuals irradiated inside Japanese-style houses have been considered to have died from marrow failure, the contribution to the dose from neutrons has been calculated to be only about 2% of the total marrow dose [R20]. Thus the contribution from doses of neutrons to early effects in the population at Hiroshima is now considered to be much less than had previously been thought and approaches the level calculated for Nagasaki.

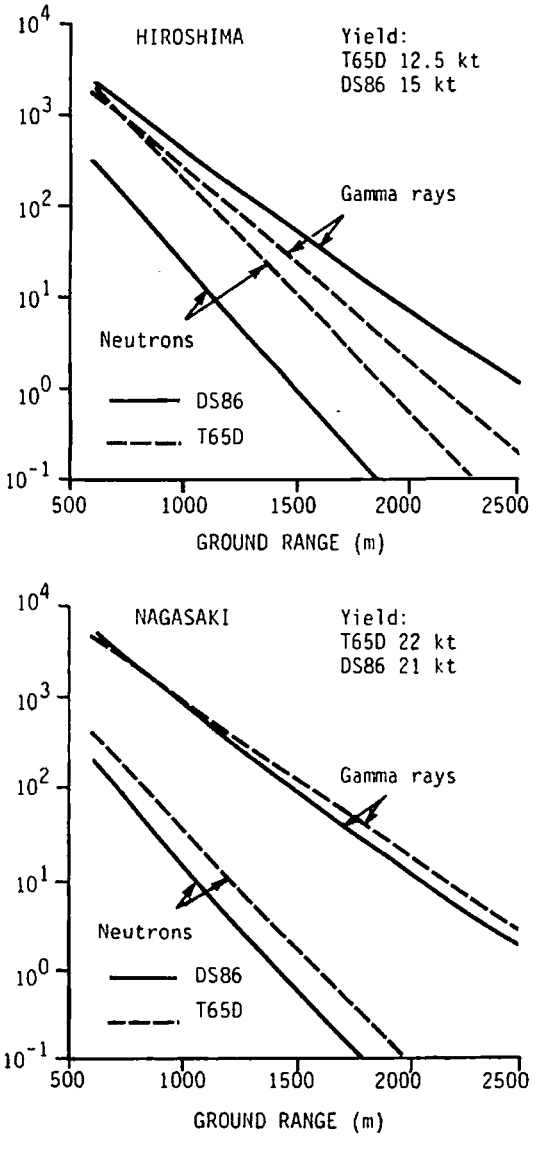


Figure XXIII. Comparison of 1965 estimates (T65D) and revised 1986 dosimetry (DS86) for initial nuclear radiation in Hiroshima and Nagasaki.
[K16]

128. RBE values for fission neutrons are 2-3 for $LD_{50/30}$ in small rodents, e.g., 2.0 for guinea pigs [B5] and 2.7 for mice [G1]. Similar values are obtained for haemopoietic stem-cell survival [C1]. Although in general RBE is a function of dose, RBE values for haemopoietic stem cells are not markedly dependent on the dose in the range under consideration for components of dose in the $LD_{50/30}$ [C1, C3]. Also, it has been considered valid to assume that there is no interaction between doses of neutrons and doses of gamma rays, so that the effects of combined simultaneous exposures to neutrons and gamma rays can be calculated on the basis of the estimated separate components of dose and RBE values measured at high doses, as, for example, in the accident data reviewed by Mole [M28]. There is, however, some evidence that doses from various components do interact, giving greater effects than expected for cells in vitro [H16, M11], and also for haemopoietic stem cells when the neutron component of dose is small [C3].

129. The RBE for exposures with fission neutrons appears to decrease with an increase in body size. For example, the RBE for LD_{50} in bilaterally-irradiated pigs has been reported to be 0.4-0.54 [W9, B25], 0.73 for $LD_{50/30}$ in goats and 0.83 for sheep [E1]. There are several reasons for this apparent reduction in RBE with an increase in body size. First, neutrons are attenuated more rapidly in tissue than are gamma rays, so the dose at greater depths is less and is due increasingly to gamma rays. Second, in small animals, a large part of the dose from neutrons is from charged particles, whereas with large body masses neutron capture reactions dilute the dose from charged particles with dose from knock-on protons. Third, the dose from neutrons in and near bone is less than in soft tissue remote from bone. The dose from neutrons to a one-cell-thick layer on the bone surface can be up to 20% less as a consequence of this effect [B22]. When the doses in the experiments with sheep were expressed as average doses in a 7-cm-thick outer annulus, the RBE value increased to 3, as expected for haemopoietic failure [E1].

130. The doses to the individuals in the Oak Ridge and Vinca accidents are calculated generally by summing the three components: (a) gamma ray dose due to emission from the source; (b) gamma ray dose from neutron capture in a 6 cm annulus of a 30 cm cylinder; and (c) first-collision, charged-particle dose multiplied by an RBE factor of 0.8-1.0 [M28]. All of these components have uncertainties, in particular the RBE. None the less, when the calculations are performed and doses are estimated for the various individuals (Table 12), the low mortality in this small number of exposed individuals is consistent with that in the Ewing's sarcoma patients irradiated with similar doses of low-LET radiation [M28, B7].

131. With small animals, the RBE of neutrons for the gastrointestinal syndrome is often higher than for the bone marrow syndrome [B22], and the ranges of dose resulting in the two syndromes often overlap. Thus, interpretation of the appropriate RBE values must take into account the times of death characteristic of each of the syndromes. With an increase in body size, the RBE for the gastrointestinal syndrome does not decrease as it appears to do for the bone marrow syndrome, but remains at 2.5-3, as shown for dogs [A10, A11] and for sheep [A12]. This is partly due to the confounding influences on the marrow dose of the precise distribution of active marrow and of target cells within the active marrow, and the effect of the presence of bone, which influences do not apply to the intestine. However, in the case of the intestine, a high dose to a small segment may be sufficient to lead to death (this is not true in the case of marrow). Also, because of the greater RBE values for the intestine, the contribution of gastrointestinal injury, i.e., haemorrhages and infections, to the bone marrow syndrome is greater with neutrons than with low-LET radiation, particularly after doses slightly above the $LD_{50/30}$ [E1, B23, B24]. With unilateral irradiation, severe injury to the skin can contribute to deaths after neutron doses slightly higher than the $LD_{50/30}$ [B25].

6. Modification of the $LD_{50/60}$ by post-irradiation treatments

132. The management of persons after irradiation or combined injuries has been discussed in a number of publications (e.g., [B57, C50, C51, W28, W29]). However, the value of routine medical treatments after irradiation in man is uncertain because there are no suitable groups, treated or untreated, with which the treated groups can be compared. The populations in Hiroshima and Nagasaki received minimal medical treatment owing to the destruction of the already sparse supplies and facilities [O5]. The Y-12 subjects at Oak Ridge were treated conservatively, and were admitted to hospital two hours after the accident [B29]. Prophylactic antibiotic treatment was not given, nor were bone marrow transplants. Antibiotic treatment was given to two patients for mild oropharyngeal infections. The subjects at Vinca were treated more extensively [J2]. They received antibiotics on the day of the accident and thereafter when necessary. They were barrier-nursed and given injections of packed red cells and transfusions of platelets when necessary. The patient who later died had received a transfusion of foetal and later adult haemopoietic tissue. The other four subjects apparently showed a favourable response to the transplants, with parallel changes in the blood and bone marrow and clinical condition. However, because the haemopoietic cells were given at 27-36 days after irradiation, when endogenous recovery would be expected to have begun, it is difficult to judge the degree of efficacy of the transplants. The radiotherapy cases [R6] were barrier-nursed, with antibiotics given for febrile infections. Platelet transfusions were given on two occasions. The Appendix describes the extensive treatment, including marrow transplants, given to the victims of the accident at Chernobyl.

133. The use of antibiotics is reported to have been beneficial in large animals. Monkeys, whose $LD_{50/30}$ is 6.0 ± 0.2 Gy without the use of antibiotics, were given doses of 8.2 Gy, which would normally result in less than 5% survival [B30]. Antibiotics given between 1.5 days before and 14 days after irradiation increased the survival rate to 28% (7/25), and the addition of

typhoid vaccine to the treatment protocol to hasten marrow regeneration gave a survival rate of 36% (5/14). The estimated $LD_{50/30}$ was increased to 7.5 Gy, i.e., by a factor of 1.25. The medications prevented most of the diarrhoea and anorexia observed in the monkeys who had been irradiated but not treated. These latter animals died between days 10 and 13 of septicaemia due to enteric organisms.

134. Other studies with animals have combined antibiotics with other supportive treatments such as fluid replacement and blood or marrow transfusions. In dogs, platelet transfusions, together with antibiotics, were successful in overcoming the critical period of haemopoietic failure between day 10 and day 20 after doses near the LD_{50} [S9, P4]. Mortality after three dose levels, where the mean survival times were about 14 days, was decreased from 9/10, 5/5, and 5/5 to 2/10, 2/5, and 1/5, respectively [P4]. Six of 12 monkeys, irradiated with lethal doses of 8-8.9 Gy and treated with autologous marrow and routinely with antibiotics, survived to seven weeks, and five of these survived to at least one year [S2]. All seven monkeys receiving the autologous marrow but antibiotics only symptomatically died between days 7 and 23. Also, six monkeys irradiated with 8.5-9.5 Gy then treated by autologous marrow 2.2-12.9 10^6 cells survived over 50 days, in contrast with a mean survival time of 14.5 days for six irradiated monkeys that had not received the graft [C19]. Only three of 18 monkeys similarly irradiated but receiving 8 10^5 homologous bone marrow cells survived to 30 days. All of these monkeys developed graft-versus-host disease; 14 out of 18 showed recovery of haemopoiesis from the donor cells, but only two survived to 30 days. Another series of experiments with monkeys showed that the $LD_{50/30}$ could be increased by a factor of about 1.8 using injections of 2-4 10^8 autologous bone marrow cells per kilogram body weight, and thrombocyte concentrates, erythrocytes and prophylactic antibiotics when necessary [B23].

135. From the limited evidence available, the efficacy of post-irradiation treatments after neutrons appears to be similar to their efficacy after low-LET radiation. The increase, by a factor of about 1.8, in $LD_{50/30}$ for monkeys, brought about by injecting 2-4 10^8 autologous marrow cells per kilogram body weight after irradiation, was found for both x rays and fission-spectrum neutrons [B23].

136. When the platelet level falls markedly below 20,000 per μl of blood, transfusion of platelets will help prevent bleeding. Infusions of granulocytes would be expected to help combat infections, but the short half-life of these cells (6.7 ± 1.4 hours in man [M6, A9, B16]) makes this procedure difficult to realize in practice.

137. The studies with large animals described above demonstrate that conventional supportive medications and transfusions of blood elements after irradiation can increase the $LD_{50/30}$ by as much as 1 Gy [B30, P4, S9]. Although this dose increment may appear small, the corresponding survival rate would increase quite markedly because of the steepness of the dose-response curve (Figure XXI).

138. Clinical data on the efficacy of bone marrow transplantation after irradiation refer mainly to the treatment of leukaemia; the results are confounded by the disease itself, concomitant cytotoxic treatment and other supportive measures. By extrapolating to man the relationship between body weight and the number of injected autologous marrow cells required for rescue of 50% of animals after LD_{100}, a value of 2 10^7 cells per kg was deduced for man [V11]. For 100% rescue, 4 10^7 cells per kg was estimated. The minimum cell dose for rescue after lethal whole-body doses to leukaemic patients using HLA identical allogeneic bone marrow cells is approximately 1 10^8 per kg body weight [T6]. This is compatible with the above extrapolations for healthy individuals, because in mice and dogs approximately four times as many allogeneic as autologous marrow cells are required for rescue [V9, V10]. Foetal liver is also an important source of haemopoietic stem cells for transplantation purposes, e.g., [W30]. The experience with bone marrow and foetal liver transplantation to the victims of the Chernobyl accident is described in the Appendix.

B. EFFECTS OF DOSE PROTRACTION

139. Protracted or fractionated doses are usually less injurious than are single doses, for two main reasons. First, cells are capable of repairing sublethal radiation damage. This process is complete in six to eight hours, and the attending increase in survival is generally greater after higher doses than after lower doses. Repair of sublethal damage can also be described by an increase in the total dose required to achieve a given level of cell killing or tissue injury. The sparing effects of protracted or fractionated irradiation are much less important after high-LET radiation, because such radiation produces much more irreparable damage than low-LET radiation.

140. Second, repopulation of cells may take place during the overall time of irradiation. The time of onset of compensatory proliferation is specific to a given tissue. It occurs once depletion of the normal complement of mature cells has been recognized. In the intestine, repopulation usually commences within a few days of the beginning of irradiation and in skin it commences after about two weeks. The doubling time of regenerating clonogenic cells is usually about one day, and may be less. The doubling time is longer than the cell cycle time because of concomitant differentiation of the clonogenic cells and hence their loss from the precursor cell pool. The cycle time during regeneration is much shorter than before irradiation, and there can be an increased number of divisions in the amplifying proliferative populations, leading to a transient overshoot in the mature cell populations. Low dose rates, 0.4-2.7 Gy per hour, can block cells in the cycle and prevent cell division [M36].

141. Of the main tissue responses described in this Annex and occurring within a few months of irradiation, the lung shows a greater sparing effect of dose protraction or fractionation over a week or two than the intestine or skin [T24, U4] (the marrow shows a lesser effect). Empirical formulae have been devised to

describe the increase in dose that is tolerated with protraction or fractionation in radiotherapy. With dose protraction, the increase in iso-effective dose with increasing irradiation time, T, can be described by the formula

$$\text{Dose} = \text{Constant} \times T^{m}$$

where m is the exposure-time coefficient. Alternatively, $D = \text{constant} \times R^{m/(m-1)}$, where R is the dose rate. The formula is applicable over a limited range of exposure times, which, like the value of the coefficient, varies between tissues [T24]. For human skin tolerance, m is about 0.29.

142. The most widely used description of fractionation effects is the Ellis formula [E3], in which the number of fractions and the overall time are variables. According to Ellis,

$$\text{Total dose} = \text{NSD} \times N^{0.24} \times T^{0.11}$$

where NSD is the nominal standard dose and the exponents 0.24 and 0.11 apply to early skin reactions. The formula is generally considered valid for between 4 and about 30 fractions, and it is recognized that different exponents apply to different tissues [T24]. Variations on the formula that consider partial tolerance, time-dose factors and cumulative radiation effects (CRE) have been described [T24, U4].

143. An alternative to these power-law relationships has been described more recently; the linear-quadratic relationship [D20]. It is considered to be more representative than the Ellis formula of the relationship between total dose and fraction size over a larger number of fractions, when the overall time is less than a few weeks and does not influence the dose required for a given effect [F4]. The effect E in a tissue of a series of n fractions each of dose d is given by:

$$E = n(\alpha d + \beta d^{2})$$

where α (Gy^{-1}) and β (Gy^{-2}) are constants. When n and d are changed from n_1 and d_1 to n_2 and d_2, total doses D_1 and D_2 resulting in the same effect E are related by

$$D_2/D_1 = n_2d_2/n_1d_1 = (\alpha/\beta + d_1)/(\alpha/\beta + d_2)$$

The ratio α/β is tissue specific. Lower values indicate a greater sparing effect of fractionation, e.g., $\alpha/\beta \sim$ 2-4 Gy for pneumonitis, and higher values indicate less of a fractionation effect, e.g., $\alpha/\beta \sim$ 10-20 Gy for early skin reactions [T24].

1. Prodromal responses

144. Comparatively little is known about the effects of dose rate or fractionation on prodromal responses, but there is some decreased effect due to dose protraction. The information comes mainly from radiotherapy treatments, and different centres have used different dose rates. Even when the same dose rate is used, the severity of prodromal symptoms after a given dose has differed between centres. For example, only two out of eight patients with haematological malignancies given 10 Gy (0.05 Gy per minute) had nausea during irradiation, with vomiting after 5-7 Gy [C35]. In contrast, all seven patients with a similar condition treated by Thomas et al. [T17] developed nausea, and six out of seven vomited towards the end of irradiation. Prodromal symptoms were more severe when the dose rate used to give 3 Gy to patients with Ewing's sarcoma was 0.3 Gy per minute [R6], compared to 0.03 Gy per minute [M34]. With whole-body irradiation prior to marrow transplantation, it was noted that the onset of nausea and vomiting was related to total dose, but not to the rate at which the dose was given, except possibly in the case of dose rates of less than 0.06 Gy per minute [B32]. The incidence of vomiting was about 10% in the 64 Rongelap natives exposed to fallout doses estimated to have been about 1.75 Gy, where the dose rate decreased from about 0.055 Gy per hour at the start of irradiation to about 0.016 Gy per hour after 50 hours [C16]; vomiting appeared in slightly less than 40% of accident cases and radiotherapy patients after estimated acute doses of similar magnitude (Figure V). There is no accurate information concerning high-LET radiation.

145. In monkeys, the latent period to retching or vomiting after 4.5 Gy was increased by a factor of 3 (from 30 to 90 minutes) when the dose rate was reduced from 1.2 to 0.07 Gy per minute [H35, H36]. Most of the increase occurred between 0.5 and 0.15 Gy per minute. In dogs, routine emesis during irradiation with 18 Gy could be avoided by reducing the dose rate from 0.18 to 0.05 Gy per minute [H38].

146. In the radiation accident in Mexico City in 1962, the individual receiving the highest dose delivered at 3.0 Gy per day for seven days and 0.25 Gy per day for a further 17 days, had anorexia and vomiting only after the seven days of exposure at the higher dose rate [M3]. In the individual receiving the lowest dose of about 1 Gy over 106 days of exposure at 0.09-0.16 Gy per day, fatigue was reported on day 36, but there were no intestinal symptoms.

147. An extensive series of studies was performed on patients receiving abdominal radiotherapy with 45-55 Gy (midline dose) given in 2 Gy fractions, five per week [B56]. Nausea and vomiting appeared after the first few sessions. These symptoms were highly variable in severity and they lasted for about a week. The effects were more frequent and intense after either the upper half of the abdomen or the epigastric region had been irradiated. Diarrhoea occurred during the third week when the total accumulated dose had reached 25-30 Gy, particularly in women where the field included the lower abdomen. Gastric pain was experienced by men irradiated in the epigastric region, but only rarely did diarrhoea occur in those who received irradiation to the lower abdomen and pelvis. The apparent sex differences may reflect technical differences in the irradiations.

148. Retrospective studies on 2,000 patients receiving whole-body irradiation showed increases in ED_{50} values when doses were protracted over eight days or more (Figure XXIV) [L9]. In 1,085 patients given small, daily whole-body exposures, 20-30 R (about 0.15-0.20 Gy to the stomach) per day for 30 days or

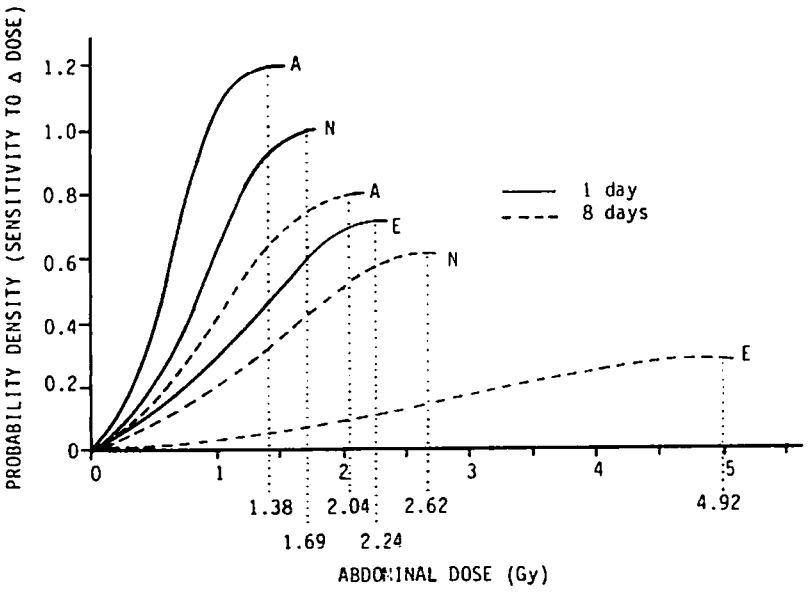


Figure XXIV. Changes in incidence of prodromal symptoms for fractionated doses in man. Fractionation of total body dose over eight days increases the doses required to produce the same incidence of various prodromal symptoms in the exposed population of patients by 1.5 for anorexia (A), 1.6 for nausea (N) and 2.2 for emesis (E).
[L9]

more were required to cause prodromal symptoms. Exposures from 10-20 R (about 0.075-0.15 Gy to the stomach) per day produced nausea infrequently, even when these exposures were delivered rapidly at approximately daily intervals for 3-4 weeks, and exposures of 5-6 R (about 0.04 Gy to the stomach) per day produced no symptoms [L9]. Patients irradiated at very low rates (less than 1.5 R, about 0.01 Gy per hour to the stomach) and receiving less than 30 R (about 0.20 Gy to the stomach) per day also showed a lack of prodromal symptoms, except fatigue [R5].

2. Intestinal responses

149. Studies of human intestinal mucosa have been made during and after x-ray therapy, using serial biopsies from patients irradiated to the abdomen for malignant disease [T9]. Exposures of 2,000-3,300 R (15-20 Gy to the intestine) delivered in daily fractions of about 1-2 Gy produced during the treatment a decreased mitotic activity in the crypts, a decrease in the absorptive surface area of the bowel, and an increased infiltration of the lamina propria by inflammatory cells and plasma cells, with occasional crypt abscess formation. Both the mitotic activity in the crypts and the mucosal surface recovered by two weeks after the end of treatment. Gastrointestinal malabsorption was reported in patients during irradiation to the abdomen with daily fractions of 1-1.5 Gy, to a total of about 30-40 Gy in five weeks [P1], or fractions of 2 Gy to a total of 45-55 Gy [B5]. Biopsies of rectal mucosa taken from radiotherapy patients receiving a total dose of 42.5 Gy in 10 fractions for bladder and cervix cancer showed a depression in total cells per crypt during treatment, with recovery to control values by day 70 after the last fraction [W10]. The number of fibroblasts in the crypt sheath was depressed by the end of the fractionation schedule; this was followed by recovery, but there was a subsequent depression at days 360 to 800 after irradiation.

150. Low-dose rate or multifractionated radiation spares the intestine in all species quite substantially [U4]. With radiotherapy treatments, tolerable fractionated doses are in the middle of the range accepted for all tissues in the body [R12]. The small intestine, rectum, colon and stomach, in this order, are the most responsive regions [F2] and will tolerate not more than 40 Gy delivered over four weeks to large volumes of tissue. Dose-mortality relationships for man due to protracted intestinal irradiation in man are unknown.

3. Haematological responses

151. Protracted irradiation is generally less efficient than acute irradiation in reducing the number of blood neutrophils. However, this effect was not detected when the overall exposure time was relatively short, as in the case of the Marshall Islanders given 1.75 Gy over 50 hours, where the haematological responses were those expected after a similar dose delivered acutely [C15, C13]. With further protraction, there is less effect per unit dose. For example, in the Mexican accident [M3], the one survivor received between 9.8 and 17 Gy over 106 days, and his lowest recorded blood cell counts were 2,000 white cells per μl and 70,000 platelets per μl.

152. The dependence of the nadir in WBC count on total dose and exposure time was described by Yuhas et al. [Y2], who analysed data for 121 patients with non-

haematological malignancies receiving fractionated whole-body irradiation over various periods of time. The relationship was:

$$\text{Per cent WBC} = K \times 100 \times D^{-b_1} \times T^{b_2}$$

where K is a constant, required for extrapolation to the ordinate at zero dose because no effect was seen below 25 R (about 0.15 Gy to the marrow); D/0.0075 is the total marrow dose in Gy; b_1 is the slope of per cent WBC on D/0.0075; T is the time of protraction in days; and b_2 is the slope of per cent WBC on T. For the patients with non-haemopoietic malignancies and with normal initial levels of WBC, $b_1 = 1.04$, $b_2 = 0.63$. The contribution to the observed effect from the diseases of these patients is unknown.

153. The recovery of marrow in irradiated leukaemic patients was slower than in patients with non-haematological malignancies [Y2]. This was deduced from 2,000 case histories where fractionated treatments had been given. Values of the coefficient b_2 were markedly different from the value of 0.63 for patients with non-haematological diseases, being 0.392 for patients with chronic myeloid leukaemia (CML), 0.221 for chronic lymphocytic leukaemia (CLL) and 0.231 for lymphosarcoma (LS). The values of b_1 were not markedly different from one another, being 0.999 (CML), 0.91 (CLL) and 1.119 (LS). The analysis indicated that the greater sensitivity of WBC levels in leukaemic than in non-leukaemic individuals was associated more with dose protraction and recovery phenomena than with total dose.

154. A study was made of patients in remission receiving fractionated whole-body irradiation over four days prior to cyclophosphamide and bone marrow transplantation for acute lymphocytic and non-lymphocytic leukaemia and chronic myeloid leukaemia [S5]. From blood samples taken during the fractionated course of irradiation, an effective D_0 of 3.7-5.4 Gy was deduced for lymphocytes and about 10 Gy for granulocytes. This confirmed that the greater radiosensitivity of lymphocytes (relative to granulocytes) applies also to fractionated doses. The values of sensitivity refer to cell numbers measured within a few hours of a dose fraction and not to the later nadir levels. In a similar study using 11 fractions of 1.2 Gy given over four days, the decline in lymphocyte numbers during irradiation was characterized by $D_0 = 1.2$ Gy [D22]. Further, the decline was similar for B- and T-cells and for the OKT4 and OKT8 lymphocyte subsets. Low whole-body doses of 0.1-0.15 Gy, given twice weekly to a total dose of 1-1.5 Gy for the treatment of generalized lymphocytic lymphoma and lymphosarcoma, produced a drop in the white cell and platelet counts, both of which reached a nadir at 4-5 weeks after completion of the irradiation [J20, J21].

155. A continuing decrease in granulocyte/macrophage colony-forming cells (GM-CFC) in bone marrow and blood during irradiation, followed by regeneration after irradiation, was reported in patients treated for malignant lymphomas using whole-body doses of 0.1 Gy delivered three times per week to a total of 1.1 Gy [L19]. In contrast, studies of the concentration of GM-CFC in the blood of five patients receiving whole-body irradiation (1.5 Gy in 15 days) for various metastatic cancers showed an increase around day 10 during the irradiation [T18].

156. GM-CFC have also been measured in patients receiving fractionated partial-body irradiation. After irradiation of 16%-30% of the total marrow in patients with various malignancies (carcinomas of the cervix, lung and rectum), a significant decrease in GM-CFC per millilitre of blood took place between days 5 and 14 after the start of treatment, by which time the cumulative doses were between 4 and 14 Gy [B48]. Between days 15 and 24 after termination of therapy delivered over several weeks, the GM-CFC per millilitre of blood were about 12% of normal and thereafter increased slowly to 24% on day 45. After doses of 30-40 Gy (five 2-Gy doses per week) delivered to 25-45% of the marrow of patients with Hodgkin's disease or non-Hodgkin's lymphomas, the ablated marrow repopulated slowly over a period of months (the repopulation was faster in larger irradiated volumes) [D16, M37].

4. $LD_{50/60}$ in man

157. There have been few instances where the number of individuals exposed to near-homogeneous protracted irradiation has been sufficient to allow an estimate of the change in $LD_{50/60}$ with dose protraction. The only information relates to a few accidents, from groups of individuals receiving irradiation from atomic bomb tests, and to radiotherapy patients receiving low-dose-rate or fractionated whole-body irradiation. Some examples of protracted whole-body exposures are given in Table 19. The 64 individuals exposed to doses of about 1.75 Gy from fallout radiation received most of their dose in the first few hours. The average exposure rate over 50 hours was about 0.03 Gy per hour, decreasing according to $t^{-1.2}$. The haematological responses were those expected for similar doses given at high dose rate, and hence any dose rate effect was small [C15, C13]. The other individuals in Table 19 received exposures over 5-115 days.

158. An accident occurred in Goiania, Brazil in 1987 [123] which resulted in initial acute whole-body external exposures followed by low dose rate chronic whole-body exposure from internally deposited ^{137}Cs chloride (from a damaged teletherapy source). In addition, many persons received acute localized radiation injuries (beta/gamma) to the skin and deeper tissues. Twenty-one persons required intensive medical care. Ten persons were critical with dose estimates (cytogenetic dosimetry) ranging from 3-7 Gy. Four persons died as a result of their exposures. In addition to good nursing care, antibiotics and platelet transfusions, the experimental drug granulocyte-macrophage colony-stimulating factor (GM-CSF) was administered to eight patients suffering from the acute radiation syndrome. Four of the patients who received GM-CSF subsequently died as a result of their radiation insult. The efficacy of using GM-CSF was not demonstrated.

159. Several formulae have been proposed to calculate equivalent doses for mortality when the dose is protracted, and these are empirical guides to changes in dose. One of the first to be proposed involved the equivalent residual dose (ERD), which was the dose required to cause equivalent injury in an unexposed individual.

$$ERD = D_1[f + (1 - f)e^{-t/r}]$$

where D_1 is the dose delivered in a single exposure, f is the fraction of the total injury that is irreparable, t is the time in days that has elapsed since exposure and r is a constant equal to the repair half-time in days divided by 0.693 [L4]. The ERD during protracted exposure at a constant dose rate is calculated as

$$ERD = D_d[ft + r(1 - f)(1 - e^{-t/r})]$$

where D_d is the dose rate and t is the exposure time in days [L33, N12]. The recovery half-time in man was postulated to be 15-35 days and f to be 10%, on the basis of sparse clinical results and extrapolation from animal data [L4, N12].

160. A power function was proposed [L9]:

$$\text{Iso-effective (fractionated) } LD_{50} = LD_{50}\text{(1 week exposure)} \times t^{0.26}$$

where t (in weeks) is longer than one week. This formula was deduced from the whole-body irradiation of cancer patients, where the LD_{50} (one-week exposure) was taken to be 3.45 Gy. It was suggested that the exponent 0.26 might be 2 or 3 times higher for healthy people.

161. Another formula has been proposed more recently for calculating accumulated iso-effective doses up to 10 Gy [B10, B21, M19] and up to 100 days exposure [M2, H47]. The operational equivalent dose (OED) for acute exposures is expressed by the formula

$$\text{OED (Gy)} = \text{total accumulated marrow dose (Gy)} - 1.5 - 0.1\,t\text{ (days)}$$

The formula was deduced from a large number of dose rate and fractionation experiments in various animal species including mice, guinea pigs, sheep and swine [M19]. The dose of 1.5 Gy in the formula represents the average amount of dose recovered in the first day among all species, and thereafter an extra dose per day (dependent on species) is required to counteract repopulation. A dose of 0.1 Gy per day is assumed for man. In view of the differences in the values of the constants between species, the formula is considered suitable only as a guide and not as an accurate assessment [M13]. It is intended for application in circumstances where a large initial dose is given. The maximum value of OED is transformed into the expected mortality using the dose-mortality curve for an acute exposure. Negative values have no meaning. Also, the relationship applies only to mortality from marrow damage.

162. The above formulae are consistent with the data for single and fractionated exposures in man (Tables 11 and 19), but they should be taken as only a very rough guide.

5. Skin

163. Information on the response of skin to fractionated doses of irradiation comes mostly from radiotherapeutic experience. This information was reviewed in detail in the UNSCEAR 1982 Report [U4], and is summarized here, together with more recent information.

164. Dose-incidence curves for erythema using fractionated doses (Figure XXV) have been measured using reflectance spectrophotometry [T21]. The measurements were made on patients irradiated using two parasternal fields, each 5 × 12 cm.

165. A dose-survival curve for epidermal clonogenic cells in situ was measured in patients receiving fractionated radiotherapy to an area 22-24 cm × 15-18 cm on the chest wall [A5]. The total doses ranged between 63 and 72 Gy and were given in 34 to 48 fractions. Cell sensitivity was characterized by $D_0 = 4.9 \pm 1.5$ Gy for these fractionated doses, a value compatible with predictions from extensive information in mice.

166. Data obtained by various radiotherapists since about 1930 were reviewed and analysed by Cohen [C7, C21], and these data formed the basis for the Ellis formula [E3]. The nominal standard dose (NSD) is about 18 Gy for skin tolerance when areas of 35-100 cm^2 are irradiated. The exponents of N (number of fractions) and T (overall time) also apply if the end-point is erythema, because the slopes of the iso-effect curves are similar, but the doses are lower. Also, the same exponents apply for different field sizes, where the values of NSD differ according to the formula given in section I.D.1.

167. For early skin reactions, the α/β ratio is generally considered to be in the range 10-20 Gy. For erythema on the chest wall, ratios of 8.4 Gy, 21.9 Gy and 21.5 Gy were determined at incidences of erythema of, respectively, 16, 50 and 84% [T21].

168. The influence of dose rate on skin reactions is known from the results of radiotherapy. Curves relating total dose and dose rate to produce "tolerable"

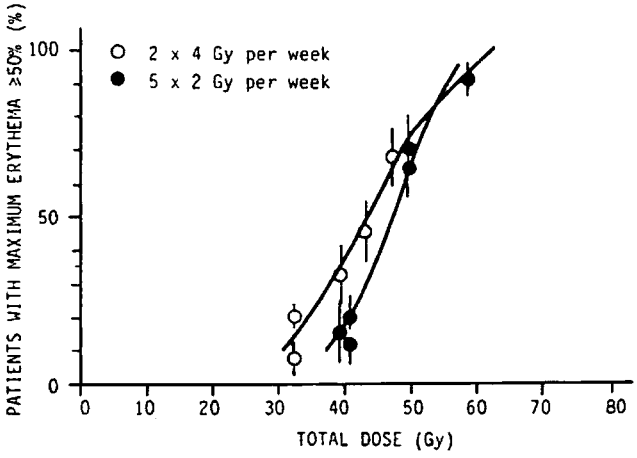


Figure XXV. Dose-incidence curves for human skin erythema. [T21]

reactions, mainly in skin, were presented by Hall [H1]. An equation describing the shape of these curves [O3] has the form

$$T = 2.1 \times 10^4 \times r^{-1.35}$$

where T is the treatment time in hours using dose rate r (Gy per hour × 0.01).

169. The time course of skin reactions is similar after neutrons or after x rays [F1]. The RBE value for single doses of neutrons (16-MeV D on Be) producing erythema on 5 cm × 4 cm areas of thigh skin is about 3.0, with reference to 8-MeV x rays [F1]. Earlier work using 200-kVp x rays as the reference radiation [S11, S12] also gave a value of about 3.0 when the original "doses" were converted to Gy [B15] and 3.5 when using (8-MeV D on Be) neutrons. For fast neutrons, the exponent of N is reduced to 0.03 [F1].

170. Radiation-induced skin injury was evident in 19 patients involved in the Goiania accident (1987). Lesions were present on hands, feet, legs, armpits and numerous small areas on chest, abdomen, face, arms and the anterior medical aspects of the legs. Skin injury was due to beta radiation from contamination (external) and to deeper underlying tissues from penetrating gamma radiation. Beta injuries healed within 3 months after exposure followed by expression of gamma injuries to deeper tissues. None of the local injuries among Goiania victims were as extensive as in the victims of Chernobyl. The clinical interpretation of this difference was that the Russian victims suffered from combined-injury disease including thermal and beta burns while the Brazilian ones were from radiation only.

6. Lung

171. The lung is spared by the use of low-dose-rate or fractionated irradiation [B32, K1, M38]. The dose for 5% incidence of pneumonitis can be increased from 8.2 Gy to about 9.5 Gy using 0.05 Gy per minute instead of 0.3-0.5 Gy per minute [K1]. The patients receiving low dose rate also received chemotherapy, which may have reduced lung tolerance. Hence the effect of reducing the dose rate may be greater than observed. This possibility is suggested by experiments with mice, where the ratio of LD_{50} values at the two dose rates was 1.8 without the use of chemotherapy [H17] and 1.3 when cyclophosphamide was given [L32].

172. With fractionated irradiation the total dose can be increased even further [M38, P5]. With prophylactic lung irradiation in the treatment of osteosarcoma, no pneumonitis was seen in 40 patients given 20-25 Gy to the lung in daily dose fractions of 1.5 Gy [N8]. This was in contrast to seven patients in whom pneumonitis was observed after 30 Gy delivered at 3 Gy per day. There was no pneumonitis in a further 14 patients given 24-25 Gy in 13 daily doses to the lung [N9].

173. In the Ellis formula [E3], the combined exponents of N and T were estimated to be 0.43, with an NSD of about 9 Gy equivalent (7 Gy with concomitant actinomycin D) [P5]. The combined exponent was deduced from a series of 26 patients treated to at least one whole lung for metastatic lung disease, using cobalt-60 gamma rays or 1-MV x rays, together with a series of fractionation data using lethality in rats after lung irradiation. Although the separate exponents of N and T have not been estimated for man, in mice the exponent of T is about 0.07 [U4]. The exponent of N is about 0.39 between one and eight fractions, and about 0.25 between 8 and 30 fractions [U4]. Alternatively, iso-effective doses for different fractionation schedules using short overall times can be calculated using the α/β formulation, where α/β for mouse lung is 2-4 Gy [T24].

174. A recent analysis has been made of 54 patients with various thoracic malignancies given irradiation to various lung volumes in daily fractionated doses [M38]. No previous treatments had been given. The incidences of pneumonitis in five groups of these patients are given in Table 20. The groupings were made on the basis of biologically equivalent doses in different schedules. No significant differences were observed in the incidence of pneumonitis for patients given irradiation to less than one quarter of the total lung volume and patients with irradiations of between one quarter and one half of the total lung volume.

7. Gonads

175. Contrary to what happens in other tissues, fractionated doses to the testis are more effective than single doses in damaging spermatogenesis in animals [U4], because of the progression of cells into sensitive stages. This is also observed in man [L13]. Compared to single doses of the same total amount (5 Gy), 20 doses of 0.25 Gy produced a more rapid drop in the number of sperm cells, and more time was required for recovery [L13].

176. Most of the quantitative data on the effects of fractionated irradiation on the testis come from the treatment of malignant disease by radiotherapy [I9, U4]. Fractionated doses of 0.5-1.0 Gy produce temporary aspermia beginning at about three months [S1]. Fractionated doses of 2-3 Gy produce long-lasting aspermia at 1-2 months [S1].

177. The few measurements of testicular hormone levels in accident cases receiving protracted irradiation are consistent with the planned study referred to earlier using single doses [R11]. After accidental exposure to iridium-192 gamma rays for various periods of time in a seven-day period, the levels of serum follicle-stimulating hormone were constantly elevated and luteinizing hormone was variably depressed after a dose estimated to have been 1.75 Gy to the testes [W1].

178. The total doses of fractionated radiation needed to cause temporary or permanent female sterility are higher in some studies than in others using single doses [I9, U4], but it is difficult to assess accurately the increase in the total doses. In mice, fractionation clearly has a sparing effect on fertility [R13]. Fractionated doses of 4-7 Gy to the ovaries of older

women induced artificial menopause in the majority of cases. Higher doses, 12-15 Gy, were required in young women [A8].

179. Serum gonadotrophin levels were unaffected by doses of up to 1.5 Gy given in fractions over 28 days to a series of patients treated for Hodgkin's disease by oophoropexy followed by irradiation [T7]. A series of patients received pelvic irradiation for carcinoma of the cervix, to a total dose of 60 Gy using doses of 9-12 Gy per week in 3-5 fractions [B12]. Levels of follicle-stimulating hormone rose immediately following doses of 5.6-24 Gy among different patients; the levels of luteinizing hormone rose after doses of 11.3-26 Gy. The levels of oestradiol in the peripheral blood decreased after doses of 6-12 Gy.

C. INTERNAL EMITTERS

180. Large amounts of internal emitters are required to produce early effects in man. Amounts this large would be received in therapeutic treatments and, possibly, in accidents or from nuclear fallout [D23]; in the case of nuclear fallout, however, external irradiation might provide the majority of the dose and could therefore be responsible for producing the early effects. Large amounts of internal emitters have been used to treat certain cancers. The dosimetry is complicated by tissue distribution, decay rates and clearance rates. More uniform distribution of dose to the body is produced by elements that are not taken up by specific organs (e.g., iodine by the thyroid, phosphorus by the marrow), and the whole-body dose depends on the circulation time before uptake. Dose rate, cumulative doses, spatial distribution of dose and the effects of internal emitters on tissues in animals were discussed in detail in the UNSCEAR 1982 Report [U4].

181. Haematological injury has been reported in man after the therapeutic use of colloidal gold, radioiodine, radiophosphorus and radiosulphur. Radiocolloids have been used to irradiate serosal surfaces following the accumulation of fluid and disseminated tumour cells. An activity of 550 MBq colloidal ^{198}Au in 40 ml saline injected into the peritoneal cavity resulted in total doses to the retroperitoneal lymph nodes, the omentum and the peritoneal serosa of 77.5, 67.5, and 47.5 Gy, respectively. Mild radiation sickness and haematological complications such as persistent leukopenia, were reported [H7]. The dose to the marrow from this treatment is unknown, but it would have been very inhomogeneous. Overdosage using 7,400 MBq of ^{198}Au resulted in estimated doses of 73 Gy to the liver and spleen and 4.4 Gy to the marrow [B40, S23], giving rise to pancytopenia, with a tendency towards recovery by day 60-70. However, on day 69 the patient died of cerebral haemorrhage, with concomitant severe thrombocytopenia.

182. The use of radioiodine to treat metastatic thyroid cancer is limited generally by the dose to bone marrow. Doses of 3,700 MBq of ^{131}I delivered in excess of 0.5 Gy to the plasma and caused sialadenitis in about 50% of patients. Bone marrow depression may be observed after multiple doses or large single doses of ^{131}I; however, this can be avoided by administering doses with individual activities not exceeding 5,500 MBq at intervals of two or more months [H2]. The accumulated dose to the blood can be as high as 5 Gy [S8]. In a large series of patients, about 3 Gy were delivered to the blood from ^{131}I-sodium iodide; after nausea, the most frequent serious complication was depression of the bone marrow [B9].

183. Detailed immunological studies have been performed on 34 patients treated with 1-3 doses of 300-350 MBq ^{131}I for toxic or atoxic nodular goiter [W27]. Blood lymphocyte counts were reduced to 60-80% at both one week and six weeks after treatment, and the frequency of lymphocytes expressing receptors for C3 (EAC-rosette-forming cells) was also reduced. At six weeks there was a small increase in the frequency of T-cells, identified by Leu-1 monoclonal antibodies; this was due to an increased proportion of helper/inducer T-cells, identified by Leu-3 monoclonals. The ^{131}I also decreased the capacity of lymphocytes to secrete IgM when stimulated with pokeweed mitogen. Less effect was seen for IgG and IgA. The mitogenic responses of lymphocytes to PHA and ConA were not changed significantly.

184. Radiophosphorus (^{32}P) has been used widely for the treatment of polycythemia vera. Single or multiple doses are given to reduce the polycythemia, and the activity per treatment, 140-220 MBq, delivers a cumulative dose to the marrow of about 1.4 Gy [S10]. The dose rate decays with a half-life of 6.7 days. Overdosage was reported with a patient who received 14.8 MBq per kg body weight [C33]. This patient showed a mild and reversible pancytopenia. Two patients were given 1,850-2,220 MBq, which delivered a cumulative dose of about 10 Gy to the marrow [G16]. Three weeks later there was agranulocytosis, severe thrombocytopenia and marrow aplasia. Haemopoiesis recovered spontaneously from day 40 after treatment. Blood counts returned to normal in one patient, but mild thrombocytopenia persisted in the other.

185. An immunological study was carried out on 16 patients receiving a single dose of 150-305 MBq ^{32}P for polycythemia [W27]. Blood lymphocytes were reduced 40% by 12 weeks after treatment. The B-cell component was reduced most, but lymphocytes expressing T-cell markers were increased. PHA reactivity was increased, but Ig secretion in response to pokeweed mitogen was reduced.

186. The treatment of chondrosarcoma and chordoma by ^{35}S is limited by the latter's haemotoxicity. In 13 patients, the cumulative activity administered as sequential amounts of 185-222 MBq per kg body weight, was 370-1,780 MBq per kg of body weight, giving a total dose to the marrow of about 9.9 Gy [M7]. The first dose had minimal effects in most patients, but with each successive dose, marrow depression increased and recovery decreased. Thrombocytopenia, leukopenia and, later, anaemia developed progressively and were dose-related.

187. Severe acute injury to the intestinal mucosa has not been reported from internal emitters in man. The highest radiation dose would be received by the large intestine, because the contents of the gut have a long residence time at in this site. The critical cells are the stem cells in the crypts, the dose at this position is the most important. Experiments in dogs indicated an $LD_{50/8}$ corresponding to 130 MBq per kilogram body weight of ^{106}Ru-^{106}Rh, which delivered approximately 40 Gy to the mucosa over about 18 hours [C18]. Comparisons were made of doses from ^{147}Pm or ^{106}Ru-^{106}Rh resulting in death from gastrointestinal injury. These isotopes have widely differing beta energies, and it was calculated that a dose of 35 Gy of either isotope to the crypt cells resulted in the death of 50% of the dogs [S14]. This dose is comparable to a dose of about 13 Gy of external irradiation delivered acutely [B16]. Values of 35-40 Gy in these experiments with dogs are compatible with similar doses of multifractionated external irradiations in man, which are considered to be tolerance doses in radiotherapy [R9].

188. In the few cases where doses to the human lung from internal emitters have resulted in symptoms of pneumonitis, the inhalation has been very protracted and the doses uncertain. For example, a chemist who had been involved in the separation of radium and mesothorium compounds and who had inhaled radioactive compounds over a long period showed signs of radiation damage to the lungs [D5]. Pneumonitis was reported in a man who had been employed for a long time in the luminous paint industry [R1]. Relationships have been described between the initial dose rate in the lung following inhalation of radioactive particles and their effective half-life in the lung, in relation to the survival of different animal species from pulmonary injury, extrapolated to man [W25]. It was deduced that death from lung injury could be expected in all individuals receiving as little as 7 MBq of an inhaled alpha emitter with an energy of about 5 MeV and an effective half-life greater than 100 days.

189. Effects of internal emitters in animals have been discussed in detail by ICRP [17] and UNSCEAR [U4]. For example, in experiments in which dogs were exposed to beta-emitting aerosols of fused aluminium-silicated particles labelled with ^{90}Y, ^{91}Y, ^{144}Ce, or ^{90}Sr, it was found that the dose to the lungs resulting in death from pneumonitis in 50% of the dogs could be increased by a factor of 5 (^{90}Sr) or 10 (^{91}Y) relative to the acute dose of external radiation [M8]. The dosage increase depends on the half-life of the isotope which governs the exposure time. With long-lived alpha-emitters, the clearance rate from the lung is most important.

190. Two individuals died after working with large amounts of tritium; they had received doses which were estimated to have been in total about 3 Gy over six years and about 10 Gy over three years [M39, S31]. A slow but continuously progressive anaemia was observed, rather than changes in the white cell count. Another individual, who received a lower accumulated dose of about 1.5 Gy over four years, showed only a slight hypoplastic anaemia.

191. Many cases of accidental ingestion of radionuclides have been reported [F10]. Bone marrow effects are particularly marked when the compound is taken up in the marrow (phosphorus) or the bones (strontium, radium) and when the half-life is long. Haemopoietic injury has been reported after the ingestion of, and chelation therapy for, 37 MBq americium-241 delivering 5.5 Gy to the bones over five years [T22] and after the ingestion of radium giving 2 Gy over six weeks and more than 50 Gy over three years [G14].

192. Extensive internal contamination with $^{137}CsCl$ occurred in 22 persons in the Goiania accident (1987). Internal contamination in these 22 individuals exceeded 85 mCi (3,100 kBq). One child in the Goiania accident had internal ^{137}Cs levels exceeding 30 mCi (1,100 MBq). Extensive ^{137}Cs internal contamination prompted the use of Prussian Blue for the first time in radiation accident history. Prussian Blue was effective in enhancing the foecal elimination of ^{137}Cs although high levels of internal contamination remain in many of these people.

193. The treatment of individuals following ingestion of large amounts of internal emitters has been discussed in various publications [D23, I2, I3, I4, N15]. The treatments are based on reduced absorption and retention, enhanced excretion or diminished translocation. Internal emitters reaching the gut can be removed to some extent by the use of emetics, lavage and precipitating agents. Colloidal ion-exchange carriers, e.g., zirconium citrate, are effective when administered within hours of exposure but are themselves toxic. Decalcification therapy, designed to increase bone resorption, enhances only slightly the elimination of radium and strontium, and it does not affect the non-alkaline earth elements, e.g., plutonium. Chelating agents such as DTPA and, more recently, LICAM C [M35] are efficient at complexing rare-earth elements and actinides.

D. BIOLOGICAL AND OTHER VARIABLES

194. Many biological variables are known to affect the response of tissues and whole animals to irradiation [U4]. In this section, only those variables will be considered that may contribute to important differences in the response of tissues in man after whole-body irradiation, namely, oxygen concentration, previous treatment by radiation or other cytotoxic agents, and genetic disorders in the general population.

195. The radiosensitivity of well-oxygenated tissues can be reduced by a factor of 2-3 by excluding oxygen at the time of irradiation. This was seen for skin reactions where a tourniquet applied to limbs enabled the dose given in radiotherapy to be at least doubled [V16]. There is evidence of a slight natural hypoxia in a few tissues in man, particularly avascular laryngeal cartilage, which was sensitized by about 10% by the use of hyperbaric oxygen [H15], and skin, which was sensitized by up to 40%, also using hyperbaric oxygen [V2]. The use of the chemical sensitizers metronidazole

and misonidazole in radiotherapy has not produced any sensitization of normal skin [D4], but there is one reported case of increased oral mucositis [A6].

196. Radioprotectors have been considered as one means of decreasing the effects of irradiation. These radioprotectors include thiol compounds, which must be administered before irradiation, and immunomodulators, which can be given afterwards [e.g., G32]. Many studies, for example those using the Walter Reed (WR) thiol compounds, have been carried out in animals. Protection factors of up to 3 have been reported for the bone marrow in mice, and values of between 1 and 2.5 for a variety of other tissues [D27]. This variation depends on the radiation dose, lower values being observed at higher doses, in part to differences in intrinsic oxygenation status among tissues, and perhaps also to their natural endogenous thiol content [D27].

197. Previous irradiation may influence the response to a second treatment if there has not been full recovery of the tissue. This is a well-known phenomenon in animal tissues, particularly in the skin [B26, H13]. At times greater than six weeks after a large first dose, the tolerance dose is reduced by about 10%, and it can be reduced further by repeated priming doses [H10]. In man, there is little quantitative evidence pertaining to skin, but some radical radiotherapy treatments to the larynx, performed up to 30 years after moderately high doses given for thyrotoxicosis, were tolerated remarkably well [H21]. Intestinal tolerance to second irradiations in man is uncertain. In mice, there is a higher resistance [H3], which is due to induced hypoxia [R4]. With bone marrow in man, there is a greater response to a second irradiation given a few months after the first irradiation [M18, T11, T12]. In animals, the $LD_{50/30}$ for a second irradiation can be greater than or less than the $LD_{50/30}$ for animals not having received any pre-treatment; this depends on the size of the priming dose and the time between irradiations [H10].

198. Many cytotoxic drugs decrease the radiation dose required for a given effect. The effect is achieved by the direct cytotoxic action of the drug and/or by synergistic interaction with radiation. This information was reviewed in part in Annex L of the UNSCEAR 1982 Report [U4]. Interaction effects are a major confounding factor in analysing the radiation response of ill cancer patients treated with other cytotoxic agents.

199. A very small sector of the population may be particularly radiosensitive because of inherited genetic disorders. The relevant data were discussed in Annex I of the UNSCEAR 1982 Report and in Annex A of the UNSCEAR 1986 Report [U4, U10]. The best documented of these disorders is ataxia telangiectasia (AT), which is an autosomal recessive disease. In this disease homozygotes may be present at a frequency of 1 in 40,000 and heterozygotes at a frequency of between 0.5 and 5% [S42]. The signs of AT are progressive cerebellar ataxia, conjunctival and cutaneous telangiectasia, frequent sino-pulmonary infections with sometimes abnormal immunity, a generally hypoplastic lymphoid system and a predisposition to cancer. Death often occurs before the age of 20, from either sino-pulmonary infections or malignancies.

200. Three AT patients, children aged seven, nine and 10, were reported to show unusually severe responses to cancer radiotherapy, particularly in respect to skin responses. Gotoff et al. [G5] described the case of a 10-year-old boy with palatal lymphosarcoma who received 30 Gy to the nasopharynx out of a total planned dose of 40 Gy. He developed marked erythema, severe dermatitis and subsequent deep tissue necrosis. It was concluded that an unusually high radiosensitivity was responsible for his death. A nine-year-old boy with Hodgkin's disease received 27.5 Gy out of a planned dose of 40 Gy to the mediastinum [M20]. He developed severe oesophagitis, the skin became pigmented and desquamated and he later died of respiratory problems. Cunliffe et al. [C20] reported a seven-year-old boy with a malignant lymphoma in the upper lobe of the right lung. After 20 Gy, dysphagia and erythema were noted, and after 30 Gy the treatment was stopped because of the severity of the responses. He died three weeks later. In addition, successful treatment of medulloblastoma in an AT patient was reported using conventional techniques but reducing the dose to one third of standard, in accordance with their findings that the sensitivity of the patient's bone marrow cells was three times normal [H49]. In a survey in 1982 of all known radiotherapy treatments of AT patients, five out of seven individuals were considered to be excessively sensitive to radiation [S30].

201. Cultured skin fibroblasts from AT patients were found to be more radiosensitive to gamma rays than those from normal individuals, by a factor of 2-3 [U4, T2]. With 14-MeV neutrons, this factor was 1.2-2 [P26, P27]. Heterozygotes have a sensitivity intermediate between that of homozygotes and of controls [C4, K2, T2], as detected for example between cell strains using low dose rates [P26, P27].

202. Peripheral blood lymphocytes from patients whose illnesses were associated with autoimmunity, such as rheumatoid arthritis, systemic lupus erythematosus and polymyositis, were found to be more radiosensitive by a factor of up to about 4 than lymphocytes from healthy volunteers or from patients whose illnesses were not associated with autoimmunity [H43]. The increased sensitivity was associated with deficiencies in DNA repair.

203. Other genetic disorders predispose to increased chromosomal injury and tumour induction after radiation. These include retinoblastoma [H4], basal cell naevus syndrome [T4, H4], Fanconi's anaemia [R3, B13], Down's syndrome [T2], xeroderma pigmentosum, Bloom's syndrome [U4] and Huntingdon's chorea [M22, K2, A7, T2]. Although the lymphocytes from some patients with Fanconi's anaemia were more sensitive to radiation-induced chromosome aberrations, fibroblasts from the same patients showed no increased sensitivity, using colony formation as an endpoint [D11]. No accurate estimates of increases in tissue radiosensitivity are available.

204. Other genetic factors in the general population that may affect radiosensitivity have been discussed in [P7]. These include familial deficiencies in glutathione metabolism and variations in genetic constitution mimicking the variations in radiosensitivity between mouse strains and mutants, particularly those with haematological disorders. Also, the radiosensitivity of natural killer cells in the immune system has been reported to be controlled by X-linked genes [B35].

205. Skin fibroblasts were taken from unirradiated sites in six patients who had an unusually severe skin reaction after radiotherapy. The fibroblasts were irradiated in vitro, and survival curves were plotted for colony-forming ability. The cells from five of the six patients showed greater sensitivity than cells taken from individuals whose skin response to radiotherapy was normal [S20].

206. In conclusion, it is believed that the proportion of individuals in the general population who, because of genetic disorders, are likely to show a significantly higher radiosensitivity for acute tissue effects is about 1%. While some of these individuals could of course occur in groups of irradiated individuals being used to calculate the $LD_{50/60}$, their rarity in the general population makes it unlikely that they would have any significant influence on the calculated values.

III. PROGNOSTIC INDICATORS AND BIOLOGICAL DOSIMETRY

A. PROGNOSTIC INDICATORS

207. In cases where persons are exposed to high doses, whether as a result of accidents or of irradiations for therapeutic reasons, it is essential to determine the prognosis as precisely as possible in order to be able to decide on the best treatment. The prognosis after near-lethal exposure is based on three types of data: dosimetric, clinical and biological.

1. Dosimetric data

208. Where doses to the body in general and to the bone marrow in particular can be determined with sufficient precision, it is possible to make a relatively accurate prognosis. This is the case with individuals irradiated for medical reasons or for those irradiated as a result of accidents where the distribution of the dose in the body and the dose rate are reasonably well known. Because all the dose-effect relationships suggested for mortality in man have very steep slopes, a very small shift towards lower or higher doses can cause a large variation in the probability of death. It is reasonable to assume that variability within a single species will be less than, or at most equal to, the variability between different species of similar body size. For different species of similar size, the LD_{50} varies by a factor of less than 2 ([U4] and Figures VII and XXII).

209. Taking estimates of $LD_{50/60}$ for all classes of individuals (healthy and sick), situated at the extremes of a probable range of $LD_{50/60}$ between 2.5 and 5.0 Gy (Figure XXI), and comparing them with an overall average $LD_{50/60}$ of about 3.75 Gy, for example, corresponds to probabilities of mortality of about 20% or 90%, assuming the same form of dose-effect relationship. Figure XXI illustrates these variations, showing, for example, that the 10% probability of death lies between approximately 0.5 and 3.5 Gy and the 90% probability, between 4 and 7 Gy. The large uncertainties preclude a formal prognosis only on an estimate of the dose to the bone marrow.

210. The intensive treatments to which exposed individuals are always subjected may completely change the prognosis. The treatments that are offered following accidental exposures are designed to combat intercurrent infections and aplasia, and they may increase the probability of survival. Those that are offered to patients suffering from neoplastic disorders often involve cytotoxic agents, and they may decrease the probability of survival. In the first case, the individuals are mostly healthy; in the second, the disease affecting the patients is an aggravating factor. In accidents, the higher the dose, the more intensive is usually the treatment; consequently, the slope of the dose-effect relationship may be less steep than the slope of the theoretical curve expressing $LD_{50/60}$ in the absence of treatment. It is possible that, after treatment, the $LD_{50/60}$ may be increased by a factor of (at least) 2 or (at most) 3 [L9, R6, T5].

211. The values of $LD_{50/60}$ are influenced by a variety of factors. The main ones are (a) sex: women appear to be slightly more resistant than men [F15]; (b) age: extrapolation from animals to man suggests that the LD_{50} at birth is lower than the LD_{50} for adults by a factor of 2; the value for adults appears to be attained at around puberty, with a subsequent decrease to minimum values in old age; (c) state of health: the LD_{50} is lower in individuals affected by other diseases, particularly if they relate to the bone marrow or if they reduce the natural immune responses; and, finally, the most important factor, (d) the protraction and/or fractionation of the dose with time.

212. In cases of accidental exposure, protraction and fractionation of the dose can have a very important effect; when irradiation is performed for medical reasons, whether it is whole-body irradiation or successive half-body irradiations, the dose is usually given over a few days to a few weeks. This may also be true with accidental internal exposure to long-lived radionuclides. If the dose is spread over a month or more, the LD_{50} may be increased to 10-20 Gy (see Table 19). The use of a model based on cellular responses and comparing single and multiple exposures used in radiotherapy would give a factor of 2, or an LD_{50} of about 7 Gy for protraction over two weeks [L9].

213. All these uncertainties make it very difficult to establish an accurate prognosis based solely on physical dosimetry. This is particularly true in the case of accidents, where, except for criticality accidents, the exposure time is very difficult to determine, giving rise to an additional error whose magnitude may reach

factors of 2-3 or more. Dosimetry is most valuable in the case of very low or very high doses because, whatever the possible error, one can at least establish whether the patient has been exposed in the non-lethal or the lethal part of the curve (broadly, doses up to 0.5-1 Gy or above 6 Gy).

214. The prognosis is related to the nature of the radiation involved. In accidents, one is generally dealing with penetrating radiation, since out of a catalogue of 98 accidents, 61 were caused by irradiators and 14 occurred as the result of criticality excursions in reactors [H20]. In whole-body medical irradiations, penetrating radiation is also usually involved, depending on which of the effects are desired. The prognosis is particularly difficult to establish in cases of criticality accidents with mixed gamma-neutron fields. There are two types of difficulty in reconstructing the dose: (a) the uncertainties in assessing the values of the neutron and gamma-ray components and (b) the choice of an RBE for the neutrons. The latter choice is particularly difficult, because the RBE varies according to the syndrome under consideration; in addition, the neutrons attenuate more rapidly with increasing depth than do the gamma-rays (see Figure XIX). Also, the simple addition of gamma doses and neutron doses multiplied by an RBE factor, may be an oversimplification and a source of additional error, as already discussed.

215. Another important element in the prognosis is the spatial distribution of the dose. In accidents, irradiation is never homogeneous. Therefore, the concept of average dose in the bone marrow, while useful for establishing an order of magnitude, is insufficient for making a precise prognosis. Relatively small volumes of bone marrow that have escaped exposure or have been only slightly irradiated because of the inhomogeneity of the exposure are sufficient to repopulate sterilized haemopoietic areas through cell migration, as long as the marrow stroma has not been damaged.

2. Clinical data

216. An accident victim will be rapidly admitted to hospital following a reactor accident (after an accident with an isolated irradiation source it may be later before the symptoms and signs of radiation injury are recognized, depending on the dose and the part of the body irradiated). At an early stage, the critical period may not yet have been reached, and prodromal symptoms may be of major importance. The prodromal phase, described in section I.C.1, lasts from the first to the seventh day; it precedes a latency period from about day 7 to day 20 after doses resulting in the bone-marrow syndrome (Table 21). The principal gastrointestinal prodromal signs are anorexia, nausea, vomiting and diarrhoea. The average 50% incidence dose is lowest for anorexia (slightly below 1 Gy) and highest for diarrhoea (between 2 and 3 Gy). Table 22, which summarizes the results of Table 2, may allow a quick prognosis for a patient presenting one or more of these symptoms. Vomiting is an easily detectable prognostic indicator, provided that no psychosomatic factor is involved. Figure V, which expresses incidence of vomiting as a function of dose, allows a preliminary assessment of the dose level and therefore of the prognosis. In addition to defining the dose-effect relationship, the intensity of these phenomena may have prognostic value: vomiting and diarrhoea may be isolated or profuse, and they may or may not increase in frequency. Their intensity and frequency are an indication of the severity. The other prodromal signs are indicators of neuromuscular reaction: fatigue, apathy, fever and hypotension (whether or not followed by hypotensive shock).

217. For doses around the $LD_{50/60}$, the most frequent prodromal indicators are anorexia, nausea, vomiting and fatigue. At supralethal dose levels, other indicators appear, such as diarrhoea, fever and hypotension [L4]. However, the prodromal indicators may occur without necessarily being followed by the death of the individual or by an acute irradiation syndrome. The latency period before their appearance is also a good prognostic feature. The earlier and more sustained is the prodromal indicator, the longer and more difficult is the return to normal, and the higher is usually the dose. Figure IV illustrates the times elapsing before appearance of the prodromal indicators; these range from a few hours for doses of around 1 Gy down to about 20 minutes or so for doses of about 10 Gy [B33]. The same data are set forth in Table 23 [I12], which also lists times of delay for the critical period (latency) and prognoses.

218. Fractionation and protraction of the dose influence the appearance and intensity of prodromal symptoms and signs. Fractionation over one to seven days increases the ED_{50} by a factor of 1.5-2.7, depending on the effect under consideration (see Figure XXIV) [L9]. Table 24 compares the ED_{50} values for the principal prodromal indicators after exposures over one day and over about a week [L9]. These doses are based on a retrospective study of 2,000 radiotherapy patients (whole-body irradiation) receiving doses above 0.3 Gy per day. The ED_{10} is estimated to be about one quarter of the ED_{50}. The mean factor for exposures over a week is approximately 2; by extrapolation, it could go up to 3 for longer periods.

219. The appearance of erythema during the prodromal phase is a bad prognostic sign, particularly if erythema covers extensive areas, as this indicates a high dose. The prognosis is poorer if the erythema appears at an early stage, in spite of the fact that the patient may still appear to be in good health. For whole-body irradiations with energies of 0.1-0.5 MeV, erythema becomes manifest after doses of 2-3 Gy; with much higher energies, it will indicate higher doses at depth because of the build-up of dose in the surface layers.

220. The absence of any prodromal symptom soon after irradiation indicates an excellent prognosis: the average dose in the whole organism is probably less than 0.5 Gy and certainly less than 1 Gy. A few isolated, temporary symptoms of moderate intensity

suggest a dose below 2 Gy. From the first days after the accident onwards, the presence of clinical indicators and the observation of their severity allows a more accurate prognosis, and therapeutic decisions can be taken without waiting for the acute symptoms of the later critical phase.

221. Once the critical phase begins, the prognostic elements are much easier to interpret than they were in the prodromal phase. An excellent indicator is the time elapsing before the appearance of the critical phase; the shorter the latency time, the less favourable the prognosis. All cases of accidents involving whole-body irradiation have shown this [H20]. During the critical phase, the appearance of new clinical indicators, an increase in their severity and persistence are bad prognostic signs. Table 25 lists the principal signs that may appear, classified in order of increasing severity, but not necessarily in chronological order of appearance [N4].

3. Biological data

222. The haematological syndrome presents the most serious problem for clinicians. The gastrointestinal and neurological syndromes appear at considerably higher doses: 10-15 Gy in the digestive tract and 50 Gy or more in the central nervous system are required to trigger these syndromes in one week and in a day or two, respectively. In the case of uniform whole-body exposure, the haematological syndrome occurs without fail below 10 Gy and down to a few Gy.

223. The earliest haematological indicator is a reduction in the concentration of blood lymphocytes. The speed at which this phenomenon begins is directly related to the mean bone marrow dose. In general, once the fall has started, its rate, estimated over the first three days, is a good prognostic indicator [H20]. Figure XXVI shows the lymphocyte reduction in six subjects irradiated in the course of three accidents [I13].

224. Other signs are also useful, although later, biological indicators. The fall in granulocytes concentration in the circulating blood to very low levels is an important feature to be monitored, because granulocytopenia is responsible for intercurrent infections, which may cause death. Also important are the thrombocytes, which help prevent haemorrhages. Daily blood counts are the basis for the immediate prognosis and for decisions about transfusions of blood cells.

225. An important element in prognosis is the minimum level of the various blood cells and the date on which this minimum is reached (Figures X, A.II.b and A.V). In three accidents, with doses ranging from 3 to 12 Gy, the time taken to reach the nadir varied from about 4-7 days in the case of lymphocytes and from 10 days to about a month in the case of granulocytes and platelets [N5]. Quantitative data are set out in Table 26, together with the clinical outcome or the prognosis.

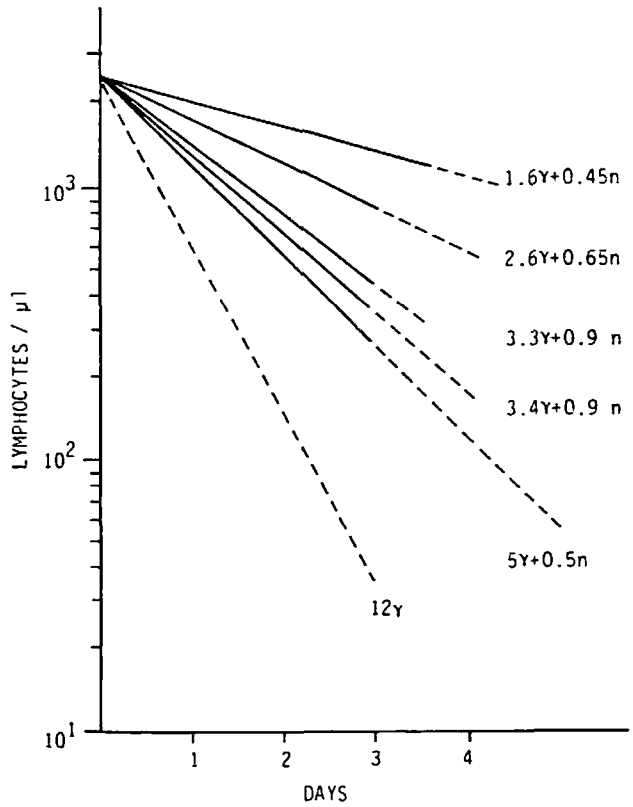


Figure XXVI. Approximate reductions in lymphocytes following accidental exposures to inhomogeneous doses. Data from six individuals exposed in three accidents: Brescia, Italy (1955): 12 γ; Mol, Belgium (1965): 5 γ + 0.5 n; Vinca, Yugoslavia (1958): 3.4 γ + 0.9 n (4.38); 3.3 γ + 0.9 n (4.18); 2.6 γ + 0.65 n (3.38); 1.6 γ + 0.45 n. Original estimates of doses (Gy) related to gamma rays and neutrons are presented separately. Values of dose in parentheses are revised equivalent low-LET marrow doses (see Table 12).
[I13]

226. After prolonged exposures it is difficult to assess the time taken for the haematological syndrome to appear, because it is difficult to fix a starting point for the irradiation period. The minimum values have the same significance for the prognosis as in the case of acute exposure; the length of time at the minimum level is more difficult to interpret for purposes of prognosis, since it seems to be related to the exposure period [H20]. Table 26 indicates minimum values of the same order of magnitude as those in Table 27 [N5].

227. During the critical phase, the duration of marrow aplasia is an important feature. A low blood count lasting for a long time is a bad sign, probably indicating not only a high but also a relatively uniform bone marrow dose and, possibly, a prolonged exposure. In the case of prolonged exposure, the depression is long-lasting and the repopulation rate is particularly slow, a mirror image of the initial slow reduction [H20]. During the phase of recovery, the reappearance of cells (whether mature or not) in the circulating blood, and their gradual increase, are good signs. Lymphocytes and platelets are generally slower in returning to normal than are granulocytes. Very often, all blood-cell types fluctuate considerably around

their normal concentrations when they return to levels within the normal range, but this phenomenon has no prognostic importance.

228. The appearance and persistence of immature cells in the circulating blood is a good sign, because it indicates a good bone marrow response. The cells most frequently found belong to the granulocyte lineage: pro-myelocytes, myelocytes and meta-myelocytes. As a rule, they are present only in small numbers. It is their continuing presence over a period of days, rather than their absolute number, that is the basis for a favourable prognosis. Likewise, the number and variation over time of reticulocytes are important.

229. Other conditions such as a rare blood group, repeated transfusion problems (shock, etc.), sudden anaemia indicating haemorrhage, or leukocytosis indicating an infection, are unfavourable prognostic signs [H20].

230. A detailed examination of the bone marrow is essential for several reasons. A number of marrow punctures in widely scattered areas selected according to the conditions of the accidental irradiation (that is, the subject's position in relation to the source and the part of the body that has probably been most exposed) will give information about the uniformity of the irradiation. The severity of marrow aplasia is directly related to the distribution of the dose [I14]. Marrow punctures give a much more reliable picture of the bone marrow state than does the circulating blood. However, the prognosis is not necessarily poor if the samples all show a severely depleted marrow; it requires only a few stem cells to repopulate the marrow, and direct examination with differential counting of the bone marrow cell types is an insufficient basis for a reliable medium-term prognosis. It is not unusual for an apparently depopulated marrow to become repopulated to a normal level.

231. It is then necessary to perform further tests on the bone marrow cells; the cells (e.g., CFU-MIX) closely related to the stem cells should be cultured, because their existence indicates the likelihood of subsequent bone marrow restoration. There is, however, a practical problem in that such cultures take quite a long time to grow (around one week), and they require fairly elaborate techniques that cannot normally be carried out on a large scale [I14]. Furthermore, it is debatable whether they are useful in patients with extensive aplasia, in view of the relatively large marrow samples needed for the examination. Since it is exactly those individuals exposed to the highest doses who require a precise prognosis, this culture technique has its limitations.

232. Because it is easier to take blood samples than marrow samples, cultures are generally made of the circulating progenitor cells (GM-CFC). This technique has been used in cancer patients receiving partial-body or whole-body irradiation to assess injury and recovery in haemopoietic progenitor cells [T18]. There are difficulties here too, however. These circulating cells have a low concentration, the culture techniques are elaborate, and the number of GM-CFC in the blood may not adequately reflect the concentration of stem cells in the marrow. For the time being, therefore, this method remains qualitative and its true value uncertain.

233. Quantitative marrow scintigraphy can be used to evaluate the regions of the bone marrow that are still functional; this technique produces quantitative findings quite rapidly [I14, P16]. The pattern can be followed for about a week. It is possible to study, in each region of the bone marrow, the degree of iron turnover (incorporation by the erythrocytes and release by the reticulocytes) and its uptake. It is also possible to distinguish extra-medullary haemopoietic regions and to measure their relative effectiveness. However, studies such as these relate to the erythroid lineage, not to the most important granuloid lineage, and transient erythroid recovery may occur in the absence of stem-cell recovery.

234. Cytogenetic dosimetry, which may be performed in a few days, allows an estimate to be made of the mean dose in the body. Counting the number of abnormalities in circulating blood lymphocytes (mainly dicentrics, rings and fragments) and comparing this number with reference values gives an accurate estimate of the mean dose. This approach has its limitations in cases of highly inhomogeneous acute exposures during which only some of the lymphocytes are irradiated and for which the dilution factor is not known, as well as in cases of prolonged exposure. Study of the electroencephalogram is equally useful but has the same limitations with regard to prolonged exposure. All the above techniques are discussed in section III.B.

235. Urine analysis is useful from several standpoints (see section III.B.2). It evaluates the state of the irradiated individual's renal function, which is essential to outlasting the critical, life-threatening period. It may confirm hidden haemorrhaging, by indicating haematuria, or renal malfunction associated with glycosuria or proteinuria. It is not, however, essential for determining the actual radiological damage and its consequences.

236. Biochemical analysis may throw some light on metabolic disturbances. These include disturbances that affect the regulation of the water balance, which, if extensive, can jeopardize survival. The prognosis will depend on the quality of the treatment utilized, and daily checks are indispensable. A routine check must cover (a) renal functions (urea, creatinine, calcaemia, phosphoraemia and blood ionogram); (b) liver functions (lactic dehydrogenase, transaminase, alkaline phosphatase and bilirubin); and (c) nutritional indicators (electrophoresis of peptides and proteins, and serum iron).

237. A thorough bacteriological check would make it possible, in the event that infection is discovered, to take measures that would allow a favourable prognosis, at least in the short term. The aim here would be to detect any latent infections (especially dental, otorhinolaryngeal or urinary) or opportunist infections, which are frequent in subjects with immune deficiencies. Again, the prognosis will depend on the effectiveness of the treatment. Septicaemia, fungal

infections and infections involving bacteria that are particularly pathogenic and/or resistant to antibiotics present special problems.

238. Sperm analysis is also a useful prognostic indicator. Changes attributable to irradiation are discussed in section I.D.5, and Figure XVIII shows sperm counts as a function of dose and time. The prognostic value of a sperm count is great, because the changes are very sensitive indicators at relatively low doses [I9]. A first sample must be obtained less than 40 days after the accident and a second sample, after the second month. Table 10 shows the effects of irradiation on spermatogenesis and the prognosis for fertility [L4]. It should be noted that the threshold dose for permanent sterility does not rise significantly when the dose is fractionated over some days or a few weeks. This is attributed to differentiation of the spermatogonia, which pass from relatively resistant early stages to type B, more sensitive, with a D_0 value in the region of 0.2 Gy [U4].

239. As is clear from the foregoing discussion, a prognosis founded on only one parameter or one class of parameters (dosimetric, clinical or biological) is bound to be very uncertain. To be valid, a prognosis must be founded on an entire range of data, and the wider the spectrum of these data and the better their coherence, the more refined will be the predictions. Table 23 summarizes the kinds of data that are useful in prognosis [I12], and Table 28 recapitulates the threshold levels of the signs and symptoms that can be detected by specialist teams and that appear after low doses [N5].

B. CLINICAL AND BIOLOGICAL DOSIMETRY

240. The many ways of estimating dose can be divided into two main kinds of investigation: (a) clinical dosimetry, which compresses the observation discussed in sections I.C and I.D (the relative value of these observations is discussed in section III.A) and (b) biological dosimetry, which comprises all the laboratory examinations that might allow an evaluation of the dose received by the individual, its distribution in the body, the time span of dose delivery and the quality of radiation involved. Biological dosimetry relies on haematological, biochemical, cytogenetic and neurophysiological examinations [E10, I13, J11, J12, K19, N14], which have different degrees of dosimetric value. Some are only qualitative (biochemical examinations, for example), others have considerable prognostic value (cytogenetic and neurophysiological examinations, for example); the majority are difficult to interpret in cases of protracted or fractionated exposures.

1. Dosimetry based on haematological data

241. Quantitative morphological haematology (cell count, differential count, platelet count etc.) is discussed in sections I.C.4. and III.A. Irradiation causes changes in the circulating blood components (cells and plasma) and in the haemopoietic tissues, and the examinations have to be more rigorous than routine examinations.

242. The morphology of the cells can be changed by irradiation. Frequently, the number of binucleate lymphocytes is higher than normal [H20, J13, J14, R17]; however, their appearance is generally delayed, so this measure is of little interest for immediate diagnostic purposes. Other abnormal features have been observed in the peripheral blood lymphocytes of persons irradiated with high doses, including (a) nuclear changes; (b) nuclear pycnosis; and (c) micronuclei, which are the result of chromosomal abnormalities but can be detected more easily and more quickly than karyotype abnormalities. These changes can be observed at relatively low doses, typically 0.25 Gy in vivo and 0.02 Gy in vitro [I15].

243. The number of peripheral lymphocytes displaying a defective nuclear structure has been shown to be related to dose for doses above a few Gy, administered in vitro and in vivo [W14]. This phenomenon has been studied in rats and in humans, but it cannot be readily used for dosimetric purposes because the damaged cells are trapped by the reticulo-endothelial system and rapidly disappear from the bloodstream; blood samples must therefore be taken soon after irradiation and incubated in a culture medium for several hours.

244. The incidence of nuclear pycnosis in lymphocytes irradiated in vitro is also related to dose. In animals (rats, rabbits) there is a linear relationship up to about 1 Gy, with a low threshold at about 0.05 Gy [I15]. However, this method is unreliable because pycnotic lymphocytes vary so widely among non-irradiated subjects. Moreover, it has been shown that in animals, pycnosis varies with the size of the cell and the nucleus/cytoplasm ratio [R18].

245. At doses between 1 and 8 Gy [I15], irradiation reduces the uptake of tritiated thymidine in vitro by the lymphocytes following treatment with phytohaemaglutinin. This explains the reduction in mitoses and cellular transformations during irradiation. Although it should be regarded as only semi-quantitative, this method is sometimes used in cases of accidental irradiation [W15].

246. Irradiation also affects the electrophoretic mobility of lymphocytes and the distribution of cellular volumes [S25]. This was noted in rabbits after doses of 2 and 4 Gy, where there was an increase in the number of large cells in the second week after irradiation. At the same time, in the categories of cells characterized by their degree of mobility, the category showing the first changes manifested the phenomenon only briefly, starting about five minutes after exposure and lasting for about 30 minutes. The explanations offered for this vary: an increase in cellular metabolism that produces functional changes in the lymphocyte or, alternatively, differences in the cell populations at the outset, with the most mobile groups able to undergo certain alterations outside the circulation and then to reappear with a different volume [I15].

247. Various immunological changes have been measured after regional irradiation [W17], in surface markers, mitogen and antigen responses and cytotoxic functions (see section I.C.4). Some of these changes persist for up to 10 years, but there have been no detailed studies of their potential use as biological indicators of radiation dose.

248. Leucocytes other than lymphocytes also show malformations after irradiation. There are few quantitative data, and such data as there are, are of little use in establishing a diagnosis or prognosis. These changes only confirm exposure; it is not so far possible to correlate them with dose or to know their relative importance. The most common changes are (a) giant polynuclear cells (hypersegmented polyploid granulocytes); (b) cells with small alterations in their nuclear structure, such as small chromatin adnexa; (c) the presence of immature granulocytes; and (d) mitotic abnormalities in erythroblasts and granulocytes [B16, F5, I15, I16]. These abnormalities occur only infrequently. The reduction in the number of monocytes may be related to the inhomogeneity of the dose, in the sense that when severe monocytopenia appears rapidly, it is a sign that a very large proportion of the bone marrow has been irradiated [I15]. Conversely, if a significant part of the haemopoietic marrow has escaped irradiation, there may be only a temporary reduction in the number of monocytes, or even monocytosis with an increase in the number of immature cells [I16].

249. Erythroblasts can be found in the blood stream after irradiation, always in very low proportions [B16, I15]. Reticulocytes are useful indicators in prognosis [H20]; a dramatic fall in reticulocyte count is often a sign of early fatality.

250. Serum glycoproteins increase in the presence of infection, inflammation or neoplastic and idiopathic disorders. The effect of irradiation on the concentration and distribution of protein-bound carbohydrates in the serum of mice and dogs has been studied [I15]. A considerable increase has been reported in animals exposed to lethal doses, while no notable change has been reported in animals receiving lower doses. It is not possible to treat all glycoproteins as one entity to be used as a dosimetric indicator. However, following the separation of various elements, changes in concentration have been noted for transferrin, haptoglobulin, the β_2 glycoproteins and the α_2 macroglobulins [E8]. A common feature of all these proteins is their richness in bonded carbohydrates.

251. Because the bone marrow function is extremely important for prognosis, all tests of its proliferative ability may a priori be regarded as useful dosimetric indicators. The analysis of bone marrow cannot in any case be a substitute for studies of the peripheral blood, which are currently the most reliable biological dosimeter.

252. The mitotic index in bone-marrow cells is one of the most accurate biological indicators, and also has a certain prognostic value. Changes in the mitotic index are related to the dose, but doses of 1 Gy or lower produce little change [K12]. After a few Gy in man, there is an initial drop in the mitotic index, recovery at about day 8 and a further fall before it returns to near normal by day 24 [F9]. In the Y-12 accident, the marrow of individuals who had received estimated doses of 2.4-3.7 Gy had practically no mitotic cells. For higher doses, the cell count dropped slightly from day 3 after exposure. The most commonly observed morphological change was the presence of giant neutrophil precursors from day 2 to day 16.

253. Some authors have proposed a test based on the marrow's capacity to respond to stimulation: injection of ethiocholanolon causes granulocytes stored in the bone marrow to migrate into the bloodstream [G18, I15, V15]. The ethiocholanolon is a testosterone metabolite, δ-4-androstane-3, 17-dionin. It is an androsterone isomer, with the configuration 5β-H (A:B cis); androsterone has the configuration 5α-H (A:B trans). The ethiocholanolon test was first used in patients suffering from malignant blood disorders; given the relationship between the responses to this test and the quality of the peripheral granulocytic cell pool, it was considered to be a good guide to the therapy of these blood diseases [V15]. Furthermore, ethiocholanolon affects neither the mononuclear nor the thrombocytic cell lines. The granulocyte outflow starts quickly and lasts for about 16 hours after the injection. Because of its low toxicity and the good reproducibility of the response, the method using ethiocholanolon is superior to methods using other leucocyte-mobilizing agents. A positive response indicates active medullary production of granulocytes.

254. Many of these methods have now been superseded by cell culture techniques for bone marrow. These techniques are at present fairly reproducible; mixed-cell colonies originating from cells closely related to the stem cells and granulocyte/macrophage colonies arising from granulocytic precursor cells may appear in cultures even using marrow punctures that have indicated, morphologically, a lack of haemopoiesis. Quantitative medullary scintigraphy, in conjunction with the iron-59 test, gives a picture that can be used to assess the impairment of the marrow.

255. Because the mature cells are resistant and have a long lifetime (approximately four months), it is difficult to use erythrocytes directly as early biological indicators of radiation damage, although the erythroid precursor cells are radiosensitive. Iron is incorporated only into the precursor cells, and the iron-59 test, in conjunction with the other tests of medullary function, allows the erythroid cell populations to be evaluated.

256. A greater denaturation of haemoglobin in erythrocytes by phenylhydrazine was reported in occupationally exposed persons, compared with the normal population [G22]. In patients with bronchial carcinoma given fractionated doses (1.2-1.5 Gy per day), an increased denaturation was observed when the cumulative dose reached 7-9 Gy [G22]. However, no increase was noted when erythrocytes from normal individuals were given doses between 1 and 8 Gy in vitro [G23].

2. Dosimetry based on biochemical data

257. Any changes in the blood biochemical parameters may be regarded as interesting signs. Glycaemia cannot be taken as a biological indicator because of its high degree of stability in the body. It is not unusual to observe hyperglycaemia from day 1, followed by pronounced hypoglycaemia (0.5 g/l) on about day 3 and a return to normal that takes about a week, after some fluctuation around the normal level [J13, J14, J15]. In the same way, fluctuations in plasma electrolytes and plasma proteins increase as the dose rises. However, the data are not accurate enough for these indicators to be used quantitatively [J15]. The features often noted are disturbances such as hypochloraemia, hyponatraemia or hypokalaemia during the first week [J13]. Electrophoretic analysis of the protein fractions shows the largest reduction (greatest at about two weeks) in the albumins. A dose-dependent appearance of a humoral factor in blood serum, which inhibits incorporation of ^{125}IUdR into cells in culture, was reported in mice [F7]. The technique has not yet been developed for man, partly because the thymidine concentration is only one-tenth that in mouse serum and partly because of other technical difficulties [F8, S29].

258. Hyperamylasemia can be produced by irradiation [B51, C38, K20, T30]. The pancreas is not very sensitive (doses up to 2 Gy have no effect), but amylase increases are detected if the salivary glands have received more than 0.6 Gy [W18]. The increases are maximal on day 1 after radiation, returning to normal by day 3, but a clear dose-dependence has not yet been established.

259. The variations in the chemical composition of the urine are of more significance than those of the blood. The urinary electrolytes may reveal changes in potassium excretion (extra-physiological fluctuations) and in the excretion of sodium and chlorine, which declines during the first few days after exposure [J13]. The 17-ketosteroids increase substantially during the first few days, before returning to normal by the end of the first week [J13].

260. After radiation exposure, there is a considerable enzymatic breakdown of nucleic acids and proteins, especially in lymphatic tissues [A29, H42, S38]. As a consequence, the urinary excretion of nucleosides and amino acids, as well as their metabolites, increases. A dose-dependent increase of deoxycytidine from normal low levels [I15] was observed in the urine of rats during the first day after a whole-body irradiation with 0.5-2.5 Gy [G30]. An enhanced excretion was also found in man after radiotherapy [B55, S39]. Similar effects were reported for the excretion of thymine in rats [Z5]; however, this was not seen in man [B55]. Thymine is metabolized to β-aminoisobutyric acid (BAIBA). The excretion of this substance is considerably increased in mice, rats and man after irradiation [S39]. After accidental human irradiation, an increase from 100-200 μmoles per litre of urine to 250-650 μmoles per litre was observed [G19, J14].

261. There is a general increase in the levels of amino acid in the urine of animals and humans during the first day after irradiation [S39]. The relative enhancement depends on the absolute excreted amount and on the metabolism of the specific amino acids. Because these factors are very complex and different for each amino acid (a decrease in urinary excretion can occur with some) no general rule is observed [S39, J14]. Accordingly, the excretion of amino acids is not usually an appropriate indicator.

262. There are, however, some amino acids or their metabolites that show a dose-dependent change in urinary excretion after irradiation. One of these is taurine, which is the metabolic end-product of cysteine. Its excretion increases 1-2 days after irradiation in the urine of rats and mice [K22, S26, S38]. Excretion increases with radiation dose in the range 0.75-2.5 Gy. In man, an enhanced urinary level of taurine was also observed after accidental irradiation [A29, J14]. It has been suggested that the increased excretion of taurine after irradiation may be related to intracellular taurine elimination due to changes in cell permeability [S26] and to the breakdown of lymphatic tissues [D12]. However, metabolic studies in mice show that the biosynthesis of taurine is also altered [H42, S40].

263. Some days after irradiation the urinary excretion of taurine decreases below normal values [L30]. This effect is due to metabolic changes of vitamin B_6-dependent decarboxylases and other enzymes which are decreased, as in the condition of vitamin B_6 deficiency [S38]. As a consequence of such metabolic alterations, the urinary excretion of kynurenic acid and xanthurenic acid (metabolites of the amino acid tryptophan) increases after irradiation of mice and rats [A29, H42, L30, S38, S39]. This effect was also observed in man [L29]. These changes occur in a dose range of 4-8 Gy, which, in mice and rats, causes severe radiation sickness prior to death [S39]. From these studies it can be concluded that biochemical indicators may be useful for certain dose ranges. Thus, the breakdown products of nucleic acids and taurine may be useful indicators in a lower dose range (0.5-3 Gy) and metabolites like kynurenic and xanthurenic acid, in a higher dose range (4-8 Gy).

264. Creatinine could serve as a measure of radiological damage to the irradiated muscles that are no longer able to metabolize it in the normal way [G20]. The level of creatinuria has never been correlated with dose, but it may confirm the uniformity of irradiation. In accidents where a relatively large portion of the body has not been irradiated, such as the Lockport accident in 1960, the level of creatinuria (creatine/creatinine ratio) scarcely increased. In accidents involving whole-body irradiation, such as occurred at Oak Ridge (Y-12) in 1958 and at Mol in 1965, the level was significant, with three conspicuous peaks in one instance (day 2, end of first week and end of second week) [J14].

265. In recent years a number of new biochemical indicators of severe radiation damage have been proposed. For example, a method has been suggested for the quantitative evaluation of damage to the

membranes of erythrocytes in the peripheral blood [M50, M51, M52]. Inhibition of the incorporation of labelled precursors of the DNA in bone-marrow cells has been used in a method worked out by Porschen et al. [P31]; the authors used their method to monitor irradiation even in small doses (some tenths of Gy). There are also data on determining the total content of desoxyribonucleotides in the blood and urine of patients irradiated for therapeutic purposes [S46, T31]. However, due to the paucity of such studies, it is difficult to evaluate the value of these indicators.

3. Dosimetry based on cytogenetic data

266. The analysis of chromosome aberrations in the circulating lymphocytes is widely used to assess the dose. Even in cases of partial-body exposure, the chromosome changes are excellent indicators of the absorbed dose. The evidence to justify the technique is well founded and covers various irradiated populations (in nuclear medicine, radiotherapy and accidents) and very wide ranges of dose [B42, K13, L15, L16]. The technique provides a reliable indication of the acute dose, since lymphocytes are widely dispersed in the various tissues and organs, have a reproducible radiosensitivity and a long life, and circulate rapidly in the body [D13].

267. Many types of radiation-induced chromosomal aberrations may appear in irradiated lymphocytes, but the dicentric aberration is currently taken as providing the most valuable information on dose. This is because the dicentric aberration is almost unique to ionizing radiation and occurs rarely in persons exposed only to normal background radiation. Centric rings occur only 5-10% as frequently as dicentrics in control or irradiated lymphocytes and are thus too infrequent to be used as a sole measure of dose. Some researchers combine the centric and dicentric yields. Acentric fragments, by contrast, have a higher background frequency, which probably reflects their induction by a large number of chemical mutagens. The confounding effect of many environmental non-radiological insults reduces the acentric's value as a measure of dose, although elevated acentric yields may qualitatively support dose estimates derived from the dicentric incidence..

268. Human T-lymphocytes have a long lifetime; a small proportion of them survives for decades. The rate of replacement is quite slow, so that in the few weeks after exposure the dicentric yield remains fairly constant. After a partial or inhomogeneous acute exposure, the lymphocytes that were in the irradiated volume of the body in both the vascular and extravascular pools are rapidly mixed with unirradiated cells. An equilibrium is reached by about 20 hours [T25], and thereafter the dicentric yield in cells from a sample of peripheral blood will provide an estimate of the average whole-body dose.

269. The dose-response for dicentric aberrations in the irradiated lymphocytes of normal individuals is little affected by factors such as the donor's age or sex. The dose-response obtained for irradiation in vivo does not differ significantly from that for irradiation in vitro [C46], so that the aberration yield observed in cells taken from an irradiated subject may be interpreted by reference to the appropriate calibration curve in vitro. In vitro curves have been established for a large range of radiation qualities, including all those likely to be encountered in accidents [L25]. Within any one laboratory, the calibration curves for dicentrics have proved to be very reproducible, provided that the cells are examined at their first post-irradiation mitosis. This is now reliably achieved by including bromodeoxyuridine in the culture medium and staining the chromosomes by fluorescence plus Giemsa [S37].

270. For low-LET radiations, the yield of aberrations, Y, conforms well to the quadratic relationship $Y = c + \alpha D + \beta D^2$ where c is the background incidence (about one dicentric in 10^3 cells), D is the dose, and α and β are fitted coefficients. A dicentric aberration requires the interaction of two breaks, each induced in separate G_0 or G_1 chromosomes. An explanation of the quadratic relationship may be that when both breaks are produced by the passage of a single ionizing track, the yield is represented by the linear term αD. The βD^2 term thus represents those dicentrics that are produced when the two breaks are caused by separate ionizing tracks. The latter term becomes more important when the dose increases.

271. In general, high-LET radiation, such as fission spectrum neutrons and alpha particles, give a linear dose response relationship, $Y = c + \alpha D$ (Figure XXVII). For these types of radiations the ionizing events are so densely distributed along the track that there is a high probability that one track will deposit energy in both chromosomes. RBE values at low doses, calculated as the ratios of the alpha coefficients of two radiations of different quality, may represent the relative hazards of the two radiations at low routine occupational levels [I21]. At higher doses, such as are likely to cause overt symptoms of sickness, the values of RBE decrease markedly [L36]. With neutrons, as their energy increases the average LET decreases and the linear model requires a second (quadratic) term, e.g., with 7.6 MeV and 14.7 MeV neutrons. RBE values for specific energies of neutrons have been proposed [B44, P18].

272. Another feature of the dose-response curves for high- and low-LET radiation is the relative importance of the dose rate. For high-LET radiation with a linear dose response, this is unimportant. For low-LET radiation, the equation $Y = c + \alpha D + \beta G(x)D^2$ can be used, where the number of initial chromosome breaks falls exponentially with time according to the factor $G(x)$. In practice, the dose-squared term reduces until the response can be considered to be linear for x- or gamma-radiation doses of a few Gy, if delivered at a more or less uniform rate over 24 or more hours.

273. An important problem in assessing the dose to an appropriate degree of precision is the number of metaphase cells that have to be examined. As a rule, evaluation of 100-500 metaphases is sufficient to estimate a dose at irradiation levels of medical

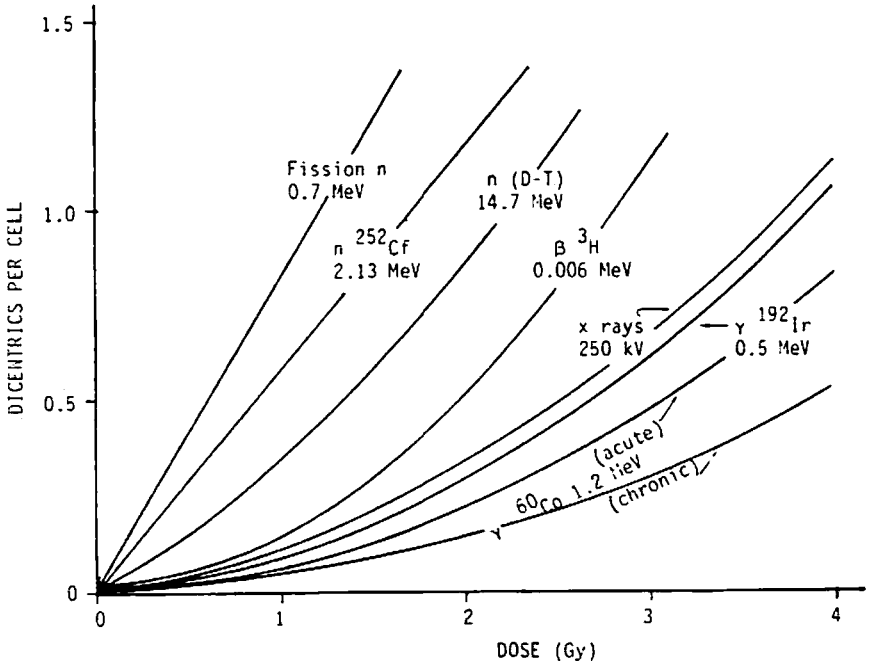


Figure XXVII. A series of generalized dose-response curves for dicentric chromosome induction for human lymphocytes irradiated in vitro.
[D13]

significance [D14, J15, L15]. With a few hundred cells scored from an irradiated subject, the statistical uncertainty on the dicentric yield is the main component of the 95% confidence limits on the dose estimate. It is much greater than the uncertainty attached to the in vitro calibration curve [L17], so that for practical purposes the latter may be ignored when calculating confidence limits. An example of an in vitro curve and the confidence limits for an exposure to gamma-radiation is given in Figure XXVIII. Examples of the use of such curves in cases of accidents have been described [D15, L18].

274. For a uniform exposure to low-LET radiation, dicentrics in the scored cells follow the Poisson distribution [D13]. However, in accidental irradiation, the exposure is almost always non-uniform, often involving just part of the body. This results in an overdispersed distribution of aberrations. The degree of departure from the Poisson distribution may be used to estimate the volume of blood exposed and its average dose [D14]. This has recently been tested in an international collaborative experiment in which partial-body exposures were simulated in vitro. The resultant estimates of dose and volume irradiated were acceptably close to the true values [L28]. The calculations require a number of simplifying assumptions [I22], but they produce values that are probably more meaningful than the average whole-body dose for accidents in which clearly only part of the body has been irradiated. However, estimates of blood volume exposed may not reflect closely the proportion of body mass exposed [L39].

275. Many chromosome aberrations, including breaks and various exchanges of the chromosome or chromatid

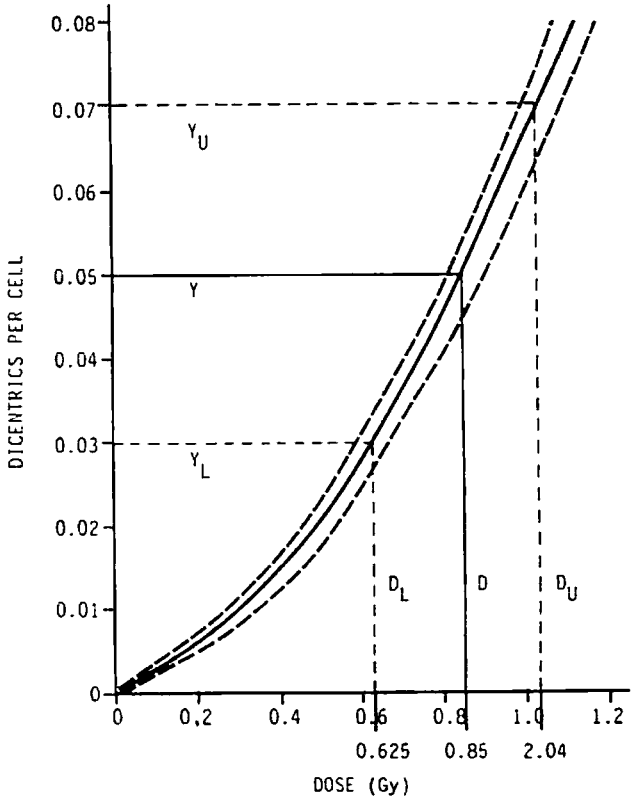


Figure XXVIII. Estimation of a dose of 0.85 Gy from a yield of 0.05 dicentrics per cell (25 in 500) by reference to an in vitro calibration curve for gamma-radiation using human lymphocytes. Statistical uncertainties on the curve are shown by the dashed curves. The upper (Y_U) and lower (Y_L) Poisson standard errors on the yield give 95% confidence limits of 1.03 and 0.625 Gy (D_U and D_L) on the dose estimate.
[I22]

type, involve acentric fragments. The absence of a centromere in these fragments prevents the correct distribution of genetic material during cell division, and they are lost or incorporated in only one of two daughter cells. In the daughter cell the fragment may either join with the main nucleus or remain in the cytoplasm and form a micronucleus [K14, R19]. Recent data indicate that 20-30% of acentric fragments may become micronuclei at mitosis [B54, W23]. Micronuclei may also occur as a consequence of completely aberrant configurations with more than one centromere because such structures frequently cause difficulties in chromosome separation during anaphase. They may also result from normal chromosomes which are left over because of a defect in the mitotic spindle.

276. Counting of micronuclei has been suggested as a dosimetric method for situations which include the evaluation of damage of chemical origin and the identification of particularly sensitive individuals with higher than average potential risks of developing cancer or genetic disorders. In principle, counting of micronuclei appears easier, faster and less expensive than the scoring of chromosome aberrations [I16, H33].

277. There is a higher background incidence of micronuclei than of dicentric aberrations and this may in part reflect the higher background incidence of acentric fragments due to environmental chemical mutagens. This means that the lower limit of dose detection by micronuclei is perhaps 0.25 Gy, thus making the technique less sensitive than that of scoring the dicentric yield. The frequency of micronuclei after x-irradiation of human lymphocytes in vitro reaches a peak after 96 hours of culture [F6, C29]. However, by this time lymphocyte cultures have become asynchronous and individual variability in cell cycling kinetics is likely to impose considerable uncertainty in the quantification of the dose response.

278. The method can be better standardized by scoring cells which have undergone a known number of mitoses. This can be achieved by differential staining of nuclei in cells which have incorporated bromodeoxyuridine [P17], and scoring cells which have previously incorporated tritiated thymidine in S-phase or by scoring cells which have been blocked at the end of mitosis using cytochalasin B [F6].

279. Currently, the cytochalasin-B blocking method is gaining considerable popularity. This technique ensures that micronuclei are scored only in those cells that have just completed their first post-irradiation mitosis. The dose-effect relationship appears to be similar to that observed with aberrations, in that the response for low-LET radiation is quadratic and at low doses it is linear [F6, F16]. Data for exposure to high-LET radiation are not yet available. The micronucleus technique is far easier and faster than scoring for dicentrics and is more amenable to automated methods of analysis using pattern recognition systems, e.g., in polychromatic erythrocytes (PCE). Individual variability, particularly at lower doses, poses some limitations. Factors such as dose protraction, fractionation and partial-body exposure have yet to be investigated. The test can be envisaged as being particularly useful after a serious accident when many people may need to be tested quickly.

4. Dosimetry based on neurophysiological data

280. Whole-body gamma-irradiation is accompanied by immediate functional changes in the central nervous system, particularly with respect to spontaneous and evoked cerebral electrical activity. These changes have been shown in animals [B45, C30, M33] and in man [C31, C32]. They appear immediately after exposure and are an important indicator of the direct effect of radiation on the optic nerve [C31]. Radiation may also affect (a) the function of peripheral receptors; (b) nerve conduction; and (c) synaptic transmission. High doses are required to modify the function of the retina, since more than 6 Gy must be administered to cause changes in the electroretinogram. The administration of large doses (~ 10 Gy) of x or gamma rays triggers a process that lowers the excitability threshold and increases the action potential and, more irregularly, the rate of conduction of the nerve impulse. Examination of synaptic transmission produces results that are harder to interpret.

281. Irradiation, even in small doses, may cause changes in the acetylcholine-cholinesterase balance or in other chemical mediators, such as aspartic acid, adrenergic amines, and gamma-aminobutyric acid (GABA). The examination of vascular lesions makes it possible to study the role of disturbances in membrane permeability. Cell metabolism is probably disturbed, as shown by reversible changes in the nuclear chromatin. Furthermore, whole-body irradiation is accompanied by changes in the acid-base balance, essentially hypocapnia and acidosis, which can be restored from the seventh hour onwards for doses of about 1.5 Gy. It seems likely that, even if the development of functional disorders of the autonomic nervous system is not superimposable on the trend of cerebral electrical activity, changes in the acid-base balance of the blood play an important role in the genesis of the disturbances observed.

282. It has been shown by one group of investigators that whole- or partial-body gamma-irradiation of the organism can act as a stimulant or as an agent of injury, depending on the level of dose, the dose rate, the irradiated volume and, above all, the percentage of the body irradiated [C31]. There is direct stimulation of the brain and particularly of the structures in the bulbar protuberance and the hypothalamus, as well as of all the synapses in the organism. This direct stimulation is followed by an indirect stimulation of the brain by the convergence of impulses originating in the spinal cord and the bulbus in the direction of the brain. Depending on the strength of these direct or indirect stimuli and the number of impulses arising in the subcortical structures, there is an immediate defence response; the intensity and nature of this response from the central nervous system, the modifications to the autonomic nervous system and the changes in cerebral activity and behaviour will differ

with respect to both their general expression and their development. These various effects combine to create an acute functional metabolic encephalopathy.

283. Disturbances in the neurophysiological equilibrium are indicated by (a) changes in excitability, consisting of successive phases of inhibition and excitation; (b) an increase in irritability in the form of paroxysmal abnormalities, ranging from a burst of slow activity through an isolated spike to a deformed spike-wave to grouped bursts of spike-waves, with rare convulsive spasms (in the case of high doses); and (c) the impossibility, at LD_{50} doses, of structures such as the hippocampus maintaining basic rhythms. On the electroencephalogram, changes in the excitation waves are noted; there is a slow-down in cerebral activity (appearance of slow, regular and broad waves), recurring spasms, slow activity or groups of slow waves, and isolated and then grouped spikes. All these phenomena are characteristic of radiation-induced effects.

284. After analysing cerebral electrical activity in the monopolar, conventional and harmonic modes, it is possible to quantify the energy changes in the power density spectrum and thus to describe objectively the slow-down in cerebral electrical activity. This is achieved by calculating the extent of drift and the percentage of the recording time during which the modifications in the EEG are observed [C32]. The dose-effect relationships obtained in animals show a response above 0.25 Gy. This response appears at 15 minutes after exposure. A comparison of whole-body and head exposures makes it possible to separate the effects of direct and indirect stimulation, the latter being under the influence of the convergence of ascending impulses from the whole body. The persistence of the effect observed above 0.25 Gy during the hours following irradiation tends to indicate changes in protein synthesis and the coding of information of the neurons. This dosimetric method is a valuable tool, especially if the assessment is done long after irradiation and in cases where it has not been possible to undertake chromosome analysis immediately after transfusions of blood components. The changes are persistent, particularly in cases of high doses. In survivors of doses of near the $LD_{50/60}$, the normal electroencephalographic patterns seem to take several years to reappear [C30].

5. Other dosimetric findings

285. In cases of exposure to mixed gamma-neutron fields, the dose, its neutron component and its spatial distribution can be estimated by determining the presence of ^{24}Na and ^{32}P [I9]. This radiation-induced activity can be measured in the body, blood, urine and biological or other specimens, such as hair, teeth, fingernails, clothes, metallic objects, jewellery, etc. These measurements are well standardized and form part of the physical dosimetry, in the same way as does the reconstruction of the accident. Techniques based on electron spin resonance [N7], applied to bone, hair, teeth and skin after low-LET irradiation, have shown that the signals obtained are quantifiable at lethal or sublethal doses down to about 0.3 Gy [B46, I17]. The electron spin resonance signal is stable at more than two hours after irradiation [T16, S28]. The intensity of the signal is linearly related to dose [B46, O7, I17, T16, S28]; it is greater for incident radiations of low photon energies [T16, O7], but was not detected after doses of 14 MeV neutrons [I17]. The method has been used to assess doses in accidents [S28] and in survivors from the atomic bombs and cumulative doses in occupationally exposed persons [T16].

286. Another assay described recently measures the frequency of variant erythrocytes produced by erythroid precursor cells with mutations that result in a loss of gene expression at the polymorphic glycophorin A (GPA) locus. A linear relationship was observed between variant frequency and dose received 40 years previously [L37].

287. Other techniques have also been suggested for use in biological dosimetry but have not yet been developed for man. One example is cell death in hair follicles (dose-dependent from about 0.1 to 1.0 Gy) and consequent changes in hair width (dose-dependent from 1 to 10 Gy) [P10, P20]. Another is spermatogenesis, which is very sensitive to irradiation and could be used as a biological indicator of dose [H37]. DNA-synthesizing cells (spermatogonia and preleptotene spermatocytes) can be measured rapidly using flow cytometry, and their concentration in mice shows marked dose- and time-dependent changes [H37].

IV. CONCLUSIONS

288. The Committee has reviewed a large body of data on the effects occurring in man within two to three months of whole-body doses above approximately 1 Gy of low-LET radiation. These data were gathered from three main sources: radiotherapy treatments, radiation accidents and the Japanese exposed to the atomic bombs in the Second World War. Homogeneous doses to the body are usually received only in the case of deliberate exposures in the course of radiotherapy, while non-homogeneous doses are usually received in accidental or warfare situations, and the effect of irradiating organs to different doses must be considered. A patient's response to whole-body radiation may also be confounded by the use of other cytotoxic agents, by disease and by medical treatment after irradiation. Data collected on patients exposed to external or internal irradiation during the nuclear accident at the Chernobyl power plant in April 1986, supplied by the delegation of the USSR, were also examined. These findings are presented in the Appendix. Finally, information on experimental work with animals was used to help interpret the responses.

289. Many of the acute effects of radiation in early-responding tissues are mediated through the death of cells when they attempt to divide. The incidence of cell death is dose-dependent, and cells that have retained their capacity to divide after irradiation can be studied, using precise techniques, in vitro and in vivo.

These surviving cells contribute to the post-irradiation recovery of the tissues. Cell death can also occur independently of cell division, as, for example, the interphase death of lymphocytes.

290. Tissues that are most sensitive to irradiation are usually hierarchical in organization, in the sense that they are structured into different compartments, each of which feeds new cells into the next compartment. One can describe, therefore, the compartment of undifferentiated stem cells, that of the differentiating and dividing precursor cells and that of the maturing and mature cells. The stem cells and the proliferative cells undergoing many divisions are the most radiosensitive. After high doses, the loss of mature cells from the last compartment is not compensated by the decreased production of cells from sterilized precursor cells in the first compartment, and this causes tissue function to fail. After high doses, the proportion of individuals showing failure of a given tissue increases as a function of dose. The relationship between dose and the proportion of individuals affected is called the dose-response curve. It can be characterized by the dose at which 50% of the individuals are affected and by the slope of the curve, which reflects the inhomogeneity in response among different individuals. The inhomogeneity results from the random nature of the radiation action, from the variability in response of the individuals in the population or from inhomogeneity in the dose.

291. Loss of tissue function produces clinical symptoms in the irradiated subject, and these symptoms will differ according to which tissue fails. Also, owing to differences in the sensitivity of cells and in the structure and function of each tissue, organs will fail at characteristic times and doses, so that certain symptoms will appear together at certain times after exposure to typical dose ranges, giving rise to the so-called radiation syndromes. Ideally, for uniform whole-body exposure above some threshold of dose, one should be able to observe a prodromal phase (common to all syndromes but of differing probability, severity and duration, according to the dose received) and three syndromes: the neurological, the gastrointestinal and the haematopoietic. In practice, depending on the level of dose, its distribution in space and time and other variables, the syndromes may often merge into each other, and it becomes difficult to recognize them as such.

292. The prodromal phase of responses after whole-body irradiation comprises the symptoms appearing during the first 48 hours. The reactions are mediated through the autonomic nervous system and are expressed as gastrointestinal (anorexia, nausea, vomiting, diarrhoea, intestinal cramps, salivation, dehydration) and neuromuscular (fatigue, apathy, sweating, headache, hypotension) symptoms and signs. The incidence and latency periods for the effects are dose-dependent. The dose inducing vomiting in 50% of individuals is about 2 Gy, and the latency period is about three hours.

293. Doses higher than 100 Gy result in death from cerebrovascular injury in the neurological syndrome within two days. This syndrome is characterized by severe symptoms and signs of the prodromal phase, followed by transient periods of depressed or enhanced motor activity, leading to cerebral coma and death. Doses between about 10 and 50 Gy result in the gastrointestinal syndrome, with most deaths occurring between days 6 and 9 after irradiation. The symptoms in man follow those of the prodromal phase and include anorexia, increased lethargy, diarrhoea, infection and dehydration. There is also weight loss, decreased food and water intake and decreased intestinal absorption. Other superimposed symptoms due to bone marrow failure include a profound drop in the leukocyte count, haemorrhage and bacteremia, which aggravate the injury and contribute to death. The time to death is influenced by the mucosal turnover time in the gut and by other, secondary factors such as infection, haemorrhage and loss of fluid, protein and electrolytes.

294. Lower doses, of a few gray, result in the bone marrow syndrome. The haemopoietic and lymphoid tissues, megakaryocytes, lymphocytes and precursor cells are radiosensitive, and leucopenia is the most important injury. The lymphocyte count is the earliest sensitive index of injury in blood and doses of 1-2 Gy reduce the concentration to about 50% of normal by 48 hours after irradiation. Neutrophils show an initial abortive increase over the first few days. A second abortive rise is seen at about day 10 after 2-5 Gy. This may be followed by a further decline if the stem-cell population fails to recover. The neutrophil count is correlated with the onset of fever. Thrombocytopenia and associated haemorrhages are increasingly important after higher doses. The time course of thrombocytopenia is broadly similar to that of granulocytopenia, but there is no second abortive rise. Thrombocytopenia below 30,000-50,000 per μl is associated with bleeding.

295. Persons exposed accidentally or therapeutically in the low- or mid-lethal dose range show an increased susceptibility to infection. Reported changes in the immune system of persons irradiated regionally include a persistent reduction in T cells of the helper/inducer and suppressor/cytotoxic phenotypes.

296. In addition to the systemic effects described, irradiation can also cause damage to many other tissues and organs. The resulting effects and clinical symptoms vary in their appearance time and severity. They may or may not be part of the syndromes described, depending upon the dose level, the tissue irradiated, the modalities of irradiation, and on other physical and biological variables.

297. Effects in irradiated skin are correlated with the dose and the area irradiated. The effects include erythema, abnormal hair growth, epilation, desquamation (dry or moist) and vascular and dermal injury. The doses that produce an incidence of 50% of abnormal hairs, erythema within four weeks and moist desquamation are, respectively, about 1.5 Gy (3 cm diameter field), 5.7 Gy (10 cm × 10 cm field) and 20 Gy (35-80 cm^2). The dose in the basal layer of the epidermis determines the amount of stem-cell killing and, hence, the degree of desquamation.

Desquamation is maximal at about three weeks after irradiation. With larger areas, smaller doses elicit the same level of damage. The 50% erythema dose is about 3 Gy when the whole skin is irradiated, which is about half the dose for areas of 100 cm^2. High doses to the dermis induce dermal erythema, necrosis, ulceration and sloughing. Vascular lesions are associated with pain in irradiated extremities.

298. Injuries in the mucosa of the mouth and throat include inflammation and swelling, with ulceration and necrosis after high doses. Mucosal injury is greatest in the cheeks, soft palate and hypoglossal area. Mucosal recovery begins by 2-3 weeks after 5-10 Gy, and it is assisted by the administration of antibiotics. Injury of the salivary glands occurs at about eight hours after 6-10 Gy, persisting to about 48 hours.

299. Acute effects on the eye include acute erythema of the sensitive conjunctiva (after 2 Gy), hyperemia of eyelid skin and hyperpigmentation (4-6 Gy), keratitis (4-10 Gy), epilation of the eyebrows and eyelashes, telangiectasia and necrosis (above 10 Gy). After 15-20 Gy local irradiation, there is lacrymation and pain in the eyes, with irritation of the cornea and iris. Even in the absence of infection, these symptoms may last for a few months.

300. Pneumonitis is the earliest sign of radiation injury in the lung, appearing at 1-3 months after doses greater than 8 Gy. The doses to lung tissue giving pneumonitis in 5% and 50% of patients irradiated over the whole body prior to marrow transplantation are, respectively, 8.2 Gy and 9.5 Gy. The time of onset is not significantly dose-dependent between 6.5 and 12.5 Gy.

301. Acute doses of up to 4 Gy cause temporary sterility in some irradiated male individuals, and the dose inducing permanent sterility in all men is more than 6 Gy. The sperm count begins to drop after 46 days. Some of the early differentiating forms of spermatogonia are very radiosensitive, and the progression of cells into these forms explains the higher sensitivity of the testis to fractionated irradiation, as opposed to acute irradiation, dose for dose. Changes in testicular hormone levels and in Leydig cell numbers are also induced. In women, temporary sterility is induced by doses up to 4 Gy, and permanent sterility by 3-10 Gy. Older women are more susceptible, probably because the number of follicles decreases with advancing age.

302. In many cases, particularly when planning radiation protection for accidental or other types of acute exposure, it is useful to think in terms of the dose at which the probability of survival 60 days after homogeneous whole-body irradiation is 50% ($LD_{50/60}$). The data available for deriving the value of the $LD_{50/60}$ in man come from different sources, each of which poses difficulties: radiotherapy patients, accident cases and the Japanese exposed to the bombs in the Second World War.

303. In Hiroshima and Nagasaki, 50% of the deaths after day 20 in a small documented sample occurred between days 20 and 29; in a group 1,000 metres from the hypocentre at Hiroshima, 58.5% died between days 20 and 38. This peak in the death rate reflects marrow failure. The most recent estimates of the $LD_{50/60}$ from the Japanese data after revision of the dosimetry have yielded values of around 3.0 Gy. This is thought to be applicable to the very special conditions prevailing before and after the bombings and to human beings receiving no medical treatment or only minimal treatment.

304. Until Chernobyl, the two accidents involving the largest number of individuals irradiated solely with acute doses were those at Vinca and at Oak Ridge. Only one out of seven individuals in both accidents receiving doses estimated to be between 2.7 and 4.5 Gy died, not primarily from marrow failure.

305. In the Chernobyl accident (see Appendix), 115 individuals were measured to have received acute marrow doses above approximately 1 Gy of gamma rays, as assessed by dicentric aberrations in their lymphocytes. There was also beta-irradiation of extensive areas of skin in many cases, in particular in individuals also receiving high marrow doses, to accumulated skin doses of the order of 10-20 times the marrow dose. The victims received immediate and comprehensive medical treatment in specialist centres. This included barrier nursing, antibiotic treatments, and blood-cell infusions. Of 43 persons receiving marrow doses between 2 and 4 Gy, none died before 60 days (only one died, at 96 days). Of 21 individuals receiving between 4 and 6 Gy marrow doses, seven died between 16 and 48 days. Of 20 individuals receiving between about 6 and 16 Gy, two rejected a transplant but survived more than 60 days after about 8 or 9 Gy. Nineteen from the last two groups, i.e. 4 to 16 Gy, were given either allogeneic bone marrow transplants (in 13 cases) or embryonic liver cell transplants (in six cases) between one and two weeks after exposure. Fifteen of the 19 died before 60 days (and two others at 86 and 91 days), including seven from skin and intestinal injury, the others from infections and other causes. Extensive information was obtained concerning the effects on different organs of acute high-dose irradiation from a nuclear reactor accident, including bone marrow, intestine, oral mucosa, and the eye.

306. Three groups of radiotherapy patients are useful for assessment. None of 20 children and adolescents given 3 Gy to the whole body died within one year of marrow failure. However, the $LD_{50/60}$ for various groups of adults with disseminated cancers was 2.9 Gy in one series and 3.4 Gy in another. These data indicate that for ill cancer patients, the $LD_{50/60}$ is probably about 3 Gy, while for healthy individuals receiving conventional supportive treatment after irradiation it may be substantially higher, approaching or equal to about 5 Gy. The response of Japanese irradiated during wartime and receiving minimal post-irradiation medical care was more like that of the ill cancer patients than that of the healthy groups of individuals irradiated in accidents and receiving medical care.

307. Data on $LD_{50/60}$ for various species of large animals have been used to estimate the probable slope

of the dose-mortality curve for man. The average coefficient of variation among species is about 0.24, and the ratio of LD_{90}/LD_{10} is about 2. This suggests that the dose that would kill few healthy humans is about 3.0 Gy and the dose that would kill most is about 6.0 Gy.

308. Based on experiments with animals, the $LD_{50/60}$ would be expected to be greater for unilateral than for bilateral irradiation, by about 20%. This depends on the penetration of the radiation used. Doses decrease faster with depth for low energy photon or electron beams and for fission neutrons than for higher energy beams. In and near bone, there is a higher dose from low energy photon irradiation and a lower dose from neutrons.

309. In large animals, the $LD_{50/60}$ may be increased by up to about 1 Gy through conventional supportive medications and transfusions of blood elements. However, such a small dose increment can increase markedly the survival rate because of the steepness of the dose-response curve. Bone marrow grafts also increase survival. After a lethal dose in man, $2\ 10^7$ bone marrow cells per kilogram is needed to rescue 50% of individuals, based on experiments with different animal species, and $4\ 10^7$ cells per kilogram for 100% rescue. More allogeneic than isogeneic marrow cells are required for rescue. The shielding of perhaps as little as 10% of active marrow in man may reduce the mortality to zero after doses near the $LD_{50/60}$.

310. It is concluded from the various groups of individuals discussed in this Annex that the $LD_{50/60}$ for humans receiving no or little medical treatment after exposure is likely to be around 2.5 Gy marrow dose and possibly higher. A similar value may pertain to some groups of ill cancer patients receiving good medical care. For healthy humans receiving good supportive medical treatment after irradiation, the $LD_{50/60}$ is likely to be approaching or equal to about 5 Gy. The $LD_{50/60}$ can be further increased by successful marrow transplantation, probably up to around 9 Gy. After these higher doses, there may be some cases of pneumonitis occurring in the second month, unless the lungs were shielded. After even higher doses (> 10 Gy) acute gastrointestinal injury will become more prevalent.

311. Neutrons are more efficient in causing acute injury than low-LET radiations, by a factor of 2-3, using single doses. However, because of the low penetration of neutrons, values of LD_{50} for large animals can be apparently smaller for neutrons than for low-LET radiation. There is little experience in man of mortality after neutrons, except in a few isolated accidents. The neutron component of the doses to the Japanese survivors from the bombs is now considered to be much smaller than had previously been thought, probably less than 3% of the total dose at distances where acute early effects were seen.

312. In radiobiology, a protracted dose or a fractionated dose is known to have less effect than the same total dose given singly. The early effects of high doses in man also follow this general rule. Thus, prodromal responses are somewhat alleviated by dose protraction or fractionation; for example, small doses of 0.2 Gy can be delivered daily for several weeks without inducing nausea. Low-dose-rate or fractionated irradiation markedly reduces injury to the intestine in all species, including man, but dose-mortality relationships for man due to protracted intestinal irradiation are unknown.

313. The relationship between the lowest concentration of leukocytes and the total dose and exposure time has been measured. There is less effect with protraction of the dose. It has also been found that marrow recovery during irradiation is less in leukaemic patients than in other patients with non-haematological malignancies. The greater radiosensitivity of lymphocytes, as compared to granulocytes, applies to fractionated treatments as well as to single doses. Various types of quantitative formulae have been proposed to estimate changes in the $LD_{50/60}$ as a function of protracted irradiation. As the data base is sparse, these are to be taken as very rough guidelines for assessing the effects of changes in dose-time relationships.

314. The tissue responses are also markedly dependent on the mode of delivery of the dose with respect to time. The responses of the bone marrow and the skin to protracted and fractionated doses are fairly well known from radiotherapeutic experience. The lung, too, is spared by protraction. In contrast with all other tissues, protracted doses are more injurious to the testis, owing to the progression of cells into sensitive phases. In women, a larger dose is generally required to cause infertility when fractionated doses are used, but an accurate assessment is not available.

315. Large amounts of internal emitters are required to produce early effects in man. Bone-marrow depression is observed after large, single doses of iodine-131; 5 Gy is the maximum total dose that can safely be delivered to the blood. Radiocolloids have produced mild radiation sickness and haematological complications, as have radiophosphorus and sulphur-35. Severe acute intestinal injury in man from internal emitters has not been reported, and lung injury has been rare. Treatments for intake of radionuclides by ingestion are based on reduced retention, enhanced excretion or diminished translocation. Emetics, lavage and precipitating agents may help prevent gut toxicity. Decalcification therapy and chelating agents continue to be studied.

316. The radiation response of tissues can be modified by physical or chemical conditions or treatments, such as the removal of oxygen, the use of protective or sensitizing chemicals, drug adjuvants or previous treatments with cytotoxic drugs that produce residual tissue injury.

317. A small section of the population may be particularly radiosensitive because of inherited genetic disorders, such as ataxia telangiectasia (AT). Children with AT are more radiosensitive, and cultured skin fibroblasts taken from them are similarly sensitive.

Estimates of the frequency of hereditary conditions that are likely to render individuals particularly radiosensitive are of the order of one per cent in the general population.

318. It is difficult to establish a prognosis for individuals irradiated above the threshold doses for acute effects solely from an estimate of the dose, because of the steepness and uncertainty in the dose-response curves, including uncertainty of the value of the $LD_{50/60}$ for man. Also, there are many confounding factors, such as the presence of intercurrent disease, the effect of shielding and protraction and the quality of the radiation. The type, severity and duration of the prodromal symptoms, including the presence and extent of erythema, may assist in the prognosis. Haematological signs, particularly the lymphocyte count, are good prognostic indicators. The lowest concentrations of the various blood cell types and the time at which such concentrations are reached following irradiation are important inputs for the prognosis, as is the duration of marrow aplasia after high doses. The appearance and persistence of immature cells in the blood is a sign of marrow regeneration and is a favourable sign. Marrow scanning can give an indication of erythropoiesis in different regions, but for estimating the likelihood of long-term recovery, it is necessary to culture very immature cells in the marrow. Urine and bacteriological analysis may assist in prognosis; sperm analysis is important for assessing the dose and subsequent likelihood of fertility. However, to be valid, a prognosis must be founded on many different data and constantly updated.

319. Biological dosimetry relies on many prognostic indicators, as well as on laboratory tests, for which correlations between effect and dose have been reasonably well established. Changes in lymphocytes that are clearly related to dose include the appearance of nuclear abnormalities, pycnosis, tritiated thymidine uptake and electrophoretic mobility. These measurements should be regarded, for dosimetric purposes, as only semi-quantitative. Leucocyte malformations, the level of serum glycoproteins, the presence in the blood of immature granulocytes and erythroblasts and the appearance of reticulocytes are also indicative of irradiation but are not suitable for accurate dose assessments.

320. Tests of the proliferative ability of the marrow may also be regarded as useful dosimetric indicators. A drop in the mitotic index, for example, is a sign of doses higher than 1 Gy. The migration of granulocytes into the bloodstream after the injection of ethiocholanolon suggests the active production of granulocytes by the bone marrow. Cultures of mixed-cell colonies and granulocyte/macrophage colonies give some indication of the concentration of precursor cells in the marrow, as a function of the dose. By contrast, erythrocytes are relatively radioresistant and long-lived, and hence their concentration in the blood is a poor indicator of dose; however, marrow scans for erythropoiesis may be used to estimate marrow doses. Biochemical analyses of the urine are more indicative of dose than similar analyses of the blood, but no test provides a better estimate of the dose than haematological and cytogenetic measurements.

321. Cytogenetic measurements of chromosome dicentrics, rings, fragments and micronuclei provide the most accurate assessment of the average dose, of importance for acute effects, over the body. The linear or linear-quadratic relationships are well established for irradiation of lymphocytes in vitro, for many radiation qualities and dose rates. With neutrons, linear relationships apply, and the relative efficiencies of different energies of neutrons have been measured. Protracted doses are, however, more difficult to estimate.

322. Changes in a number of neurophysiological parameters have been observed after irradiation, and these have good potential for development as dosimeters. The radiation-induced activation of biological and other materials, as well as electron spin resonance measurements, are quantifiable signals at lethal or sublethal doses, down to about 0.3 Gy, that could also be used as dosimetric techniques.

Table 1

Survival parameters for human clonogenic cells assayed in primary culture after single doses of low-LET radiation

Cell type	D_0 (Gy)	Extra-polation number	Ref.
Haemopoietic progenitor cells producing			
Mixed-cell colonies	0.91	1.0	[N2]
Colonies of granulocytes	1.4	1.0	[S18]
and macrophages	1.6	1.0	[B28]
	1.3	1.1	[R21]
	1.2	1.0	[B47]
	1.4	1.0	[G21]
	1.0-1.1	1.4-1.6	[F13]
Colonies of granulocytes and macrophages in diffusion chambers	0.85	0.96	[G12]
Erythroid colonies	0.93-1.3	1.0	[G21]
Colonies of stromal cells	1.0	1.0	[F13]
T-lymphocyte precursor cells	1.2	0.9	[K6]
Skin keratinocytes	0.7-0.9	10-16	[D10]
	0.97	3.1	[P21]
Skin fibroblasts	1.32	0.95	[W11]
	1.40-1.52	2	[W12]
	0.97-1.80	1.0	[A17]
Skin and lung fibroblasts	0.75-1.30	1.0	[C25]
	1.22	1.0	[C26]
Mammary fibroblasts	1.1	2.0	[C24]
Mammary epithelium	1.3	1.0	[Y4]
	1.2	2.4	[C24]
Thyroid epithelium	0.93	2.0	[H31]
	0.7-1.1	1.0-3.5	[M41]

Table 2

ED_{50} estimates for prodromal symptoms of gastrointestinal injury for irradiated patients a/ [L8]

Response	Previous estimates b/ N = 163 c/	Oak Ridge Associated Universities N = 104	Other hospitals N = 400	All hospitals N = 504	All, six nursing notes required d/
Anorexia	0.97 (+0.31) (-0.26)	0.63 (+0.13) (-0.12)	1.29 (+0.56) (-0.31)	0.92 (+0.33) (-0.20)	0.59 (+0.14) (-0.10)
Nausea	1.39 (+0.72) (-0.33)	1.28	1.72 (+0.83) (-0.43)	1.54 (+0.47) (-0.29)	1.18 (+1.54) (-0.50)
Vomiting	1.83 (+1.78) (-0.53)	1.65	2.76 (+2.17) (-0.87)	2.30 (+1.04) (-0.53)	1.76 (+0.76) (-0.41)
Diarrhoea	2.38 (+1.22) (-0.55)	3.70 (+ ?) (-1.72)	2.94 (+ ?) (-1.66)	3.02 (+1.62) (-0.76)	2.86 (+2.30) (-0.86)

a/ In this Table, doses are given in Gy ± 1 SE; they are the average doses to a 26 cm diameter sphere in the epigastric region. Irradiation was to the whole body, and in 84 of the 163 patients (column 2) the dose rate was about 0.01 Gy per minute. The calculations assume a log-normal distribution of incidence versus dose, with an allowance made for the incidence in non-irradiated patients. The responses refer to anorexia, nausea and vomiting within two days and diarrhoea within six weeks.

b/ Space Radiation Study Panel Report [L4].

c/ N = number of patients.

d/ Clinical histories not having this minimum number of consecutive post-irradiation notes were discarded.

Table 3

Cellularity and kinetics in human intestinal mucosa
[P9, W24]

Small intestine	
Cells per villus	~ 4000-8000
Cells per crypt	~ 300- 500
Crypt cells in cycle	< 300
Cell cycle time	36-60 h
Total number of crypts	~ $6\ 10^8$
Total cells produced per day	~ 10^{11}
Transit time (crypt to villus tip)	3- 4 d
Cell cycle times (hours)	
Stomach	12-30
Ileum	30-70
Colon	16-96
Rectum	13-96
Mucosal turnover times (days)	
Ileum	3-4
Colon	3-6
Rectum	6-8

Table 4

Distribution of deaths among a small sample of documented individuals who died after the bombings in Hiroshima and Nagasaki
[04]

Days after bombing to death	Number of individuals	
	Hiroshima	Nagasaki
0- 1	0	29
2- 3	1	8
4- 5	4	28
6- 7	17	40
8- 9	25	44
10-11	4	21
12-13	7	14
14-15	8	18
16-17	7	19
18-19	17	15
20-29	137	87
30-39	80	43
40-49	13	13
50-59	6	11
60-69	11	6
70-79	2	6
> 80	5	8
Unknown	1	2
Total	345	412

Table 5

Cell kinetic data for human epidermis
[P11, P30]

Number of cell layers		
Nucleated (including basal)		~ 5.5
Corneocytes		> 10
Transit time		
Basal to granular	(d)	14 ± 6 (SD)
Granular to surface	(d)	18 ± 6 (SD)
Lifetime surface cells	(d)	~ 2
Basal cells per mm^2		(20-30) 10^3
Labelling index (18-h average)	(%)	~ 4.7
Mitotic index (18-h average)	(%)	~ 0.63
Length of S phase	(h)	9 ± 2 (SD)
Cell cycle duration	(h)	213 ± 84 (SD)
Cells produced per hour per 100 basal cells a/		~ 0.47

a/ Assuming growth fraction = 1.0.

Table 6

Skin "tolerance" doses (Gy) and field sizes
[H19]

Treatment	Field size (cm x cm)			
	6 x 4 (Small)	8 x 10	15 x 20 (Large)	L/S (%)
[P2]				
Single dose	20.0	14.5	11.00	55
3 weeks	50.0	37.5	29.0	58
5 weeks	58.0	43.5	33.5	58

Treatment	Field size (cm x cm)			
	7 x 5 (Small)	8 x 10	15 x 20 (Large)	L/S (%)
[V8]				
Single dose	25.0	17.0	-	-
3 weeks	52.5	45.0	30.0	57
5 weeks	60.0	50.0	35.0	58

Table 7

Doses to a 1 cm circle of pig skin causing dry desquamation
(modified from [M21])

Isotope	Average energy (MeV)	Threshold surface dose for dry desquamation (Gy)	Dose at 90 µm (Gy)
Sulphur-35	0.17	200	12
Cobalt-60	0.31	40	16
Caesium-137	0.55	20	17
Yttrium-91	1.53	15	12
Strontium-90 / Yttrium-90	0.61 / 2.20	15	14

Table 8

Early effects of radiation on the human eye
[M15]

Tissue	Effect	Latent period	Dose (Gy)	
			Single	Fractionated
Lid skin	Erythema, second wave	2-4 weeks	6	-
	Pigmentation	2-3 weeks	4-6	-
	Moist desquamation	2-8 weeks	-	50-60/5-6 weeks
Lid margin	Epilation (incomplete)	1-2 weeks	10	-
	Epilation (complete)	2-5 weeks		20-30/2-3 weeks
Conjunctiva	Hyperemia	Immediate	>5	-
	Conjunctivitis	1-3 weeks	-	=50/4-5 weeks
Cornea	Punctate keratitis	Several weeks	10	30-50/4-5 weeks
	Edema	1-3 weeks	-	40-50/2-3 weeks
	Mild ulceration	Several (3-6) weeks	-	30-40/2-3 weeks
Iris	Iritis	Several days	20	>60/5-6 weeks
Retina	Edema	Several weeks	-	20-35/3-4 weeks

Table 9

Kinetics of spermatogenesis in man
[B11]

Spermatogonial types	Stages from acrosome development	Duration of		Spermatids	
		Cell-cycle (h)	Spermatogenesis (d)	Number of stages	Number of types
A-dark	I-VI	<384			
A-pale	VI-V	384	64	6	6
B	VI-I	209			

Table 10

Effects of single-dose irradiation (low-LET) on spermatogenesis and fertility
[I9, L4, U4]

Dose (Gy)	Effect on	
	Spermatogenesis	Fertility
0.15	Moderate oligospermia	Temporary sterility (?)
0.20	Moderate oligospermia	Temporary sterility a/
0.50	Pronounced oligospermia	Temporary sterility
1.0	Severe oligospermia	Prolonged sterility
2.0	Azoospermia	Prolonged sterility
> 6	Azoospermia	Prolonged sterility

a/ Type B spermatogonia are exceptionally sensitive, with $D_0 \sim 0.2$ Gy.

Table 11

Previous estimates of $LD_{50/60}$ in man (acute doses of low-LET irradiation)

Data source	Midline or marrow dose (Gy)	Year	Reference
All groups	3.0	1950	[W13]
	3.0	1950,1957	[L14, G15]
	2.6-4.0	1960	[N3]
	2.5-2.9	1967	[L4]
	3.15	1974	[N6]
	3.0	1979	[K9]
	3-6	1984	[M13]
Japanese bomb casualties	5.0	1956	[O5]
	2.6	1969	[L8]
	1.54	1986	[R20]
	2.1-2.5	1987	[F15]
	2.4 a/	1987	[F15]
	2.7-3.1	1987	[F15]
Radiotherapy patients	4.0	1964	[M31]
	2.4	1966	[L11]
Accidents, with supportive treatment	3.6	1960,1962	[C13,U1]
	3.4	1975	[R22]
	5.1	1975	[R22]
	3.5	1979	[K9]
	5.25	1979	[K9]
	5.0	1979	[D21,T28]
	4.5-5.0	1983,1984,1985	[M2,M27,M28]
	4.5	1985	[U5]
Accidents, with successful marrow transplantation	11.0	1985	[U5]

a/ Revision of above value of 1.54 Gy.

FACTORS WHICH MIGHT CAUSE THE $LD_{50/60}$ TO BE:

		Section
LOWER		
	Pre-1986 dosimetry for A-bomb data	II.A.1
	Contribution of extensive burns	II.A.1
	Pre-existing illness	II.A.1
	Chronic nutritional deprivation	II.A.1
	Concurrent infections	II.A.1
	Contribution of high-LET radiation	II.A.5
HIGHER		
	Young, female	II.A.1
	Radiation poorly penetrating	II.A.1
	Unilateral irradiation	II.A.3
	Partial marrow shielding	II.A.4
	Good medical support	II.A.6
	Protracted irradiation	II.B.4

Table 12

Marrow doses (Gy) for selected accident cases
[M28, B7]

Subject	1	2	3	4	5	6	7	8
Y-12								
A	2.69	a/	0.96	3.65	0.14	2.60-4.40	3.06	3.30
C	2.50		0.89	3.39	0.13	2.50-4.10	2.84	3.01
D	2.41		0.86	3.27	0.13	2.40-3.90	2.75	3.11
B	1.99		0.71	2.70	0.11	2.00-3.30	2.27	2.72
Vinca								
V (died)	2.14	1.33	0.89	4.36	0.68	(2.30-3.10) 2.73	3.28	4.53
M	2.09	1.30	0.87	4.26	0.66	(2.30-3.10) 2.67	3.20	4.32
D	1.92	1.36	0.91	4.19	0.69	(1.80-2.50) 2.17	3.14	4.05
G	1.89	1.35	0.90	4.14	0.68	(1.80-2.50) 2.16	3.09	3.99
H	1.58	0.99	0.66	3.24	0.50	(1.70-2.30) 2.01	2.42	3.27

a/ Not stated.

Columns:
1 Gamma-ray emission by source = leakage dose = first-collision dose.
Y-12: [H23]; Vinca: [H22].
2 Gamma-ray dose for neutron capture in the surface of the body.
Y-12: [H23]; Vinca: [H22].
3 First-collision charged-particle dose. Y-12: [H23]; Vinca: [H22].
4 Total dose as published (columns 1 + 2 + 3). Y-12: [H23]; Vinca: [H22].
5 Gamma-ray dose from neutron capture in 6-cm annulus of 30-cm cylinder.
6 Marrow dose [B7].
Y-12, first figure: marrow dose if exposed from the front;
Y-12, second figure: if exposed from the side;
Vinca: uncertainty range is ± 15% of the mean.
7 Marrow dose [M28].
Values are for Y-12: 0.8 (column 1) + 0.8 (column 3) + column 5;
for Vinca: 0.8 (column 1) + column 3 + column 5.
8 A further revision of the dosimetry on the basis of lower body sodium levels, results in increased estimates of dose using sodium activation by factors of 1.06-1.20 at Y-12, and 1.29-1.41 at Vinca [M26, M27].

Table 13

Total-body irradiation in man: schematic classification of dose ranges: symptoms, therapy and outcome

	Prodromal symptoms		Clinical characteristics			Therapy, clinical course and outcome				
									If injury is fatal	
Acute dose (Gy)	Incidence (%)	Latency	Syndrome or organ involved	Characteristic symptoms	Critical period after exposure	Therapy	Prognosis	Lethality (%)	Death within	Usual cause of death
> 50	100	Minutes	Neurological syndrome	Cramps, tremor, ataxia, lethargy, impaired vision, coma	1-48 h	Symptomatic	Hopeless	100	1-48 h	Cerebral oedema
10-15	100	0.5 h	Intestinal syndrome	Diarrhoea, fever, electrolytic imbalance	3-14 d	Palliative	Very poor	90-100	2 weeks	Enterocolitis shock
5-10	100	0.5-1 h	Bone marrow syndrome	Thrombopenia, leucopenia, haemorrhage, infections, epilation	2-6 weeks	Bone marrow transplantation, transfusions of leukocytes and platelets, optimal care (isolation, antibiotics, fluids)	Uncertain depending on success of therapy	0-90	Weeks	Infections and/or haemorrhage
2-5	50-90	1-2 h	Bone marrow syndrome	Thrombopenia, leucopenia, haemorrhage, infections, epilation	2-6 weeks	Transfusions of leukocytes and platelets, optimal care (isolation, antibiotics, fluids), bone marrow transplantation	Uncertain depending on success of therapy	0-90	Weeks	Infections and/or haemorrhage
1-2	0-50	> 3 h	Bone marrow	Mild leucopenia and thrombopenia	2-6 weeks	Symptomatic	Excellent	0-10	Months	Infections and/or haemorrhage

Table 14

Symptoms for midline dose range 1.0-2.0 (Gy)
[Y7]

Symptom	Postexposure time: Hours 0	4	8	12	16	20	24	Days 1	2	3	4	5	6	7	Weeks 1	2	3	4	5	6
Nausea[a]	—30-70% mild to moderate—																			
Vomiting (retching)	—20-50% mild to moderate—																			
Anorexia	—50-90%—																			
Diarrhea (cramps)[b]																				
Fatigue[c]	—30-60% mild to moderate—														Mild					
Weakness	—30-60% mild to moderate—														Mild					
Hypotension																				
Dizziness																				
Disorientation																				
Bleeding[d]														—(e)—				—10% mild—		
Fever														—(f)—						
Infection								—(g)—										10-50% mild to moderate		
Ulceration																				
Fluid loss/electrolyte imbalance																				
Headache																				
Fainting																				
Prostration																				
Death[h]																				≤5%—

a/ References for this group of symptoms: A25, A26, A27, B17, B29, C10, C14, C15, C39, C42, C43, C44, E11, G26, G28, G29, H39, H40, J2, L20, L24, M18, M42, M44, N10, N11, N13, O5, O6, P24, R23, S33, S34, S35, S36, T5, V18, W19, W21, Z4.

b/ 10% of the Marshallese victims exposed to 1.75 Gy experienced diarrhea during the first day after irradiation, according to [A25].

c/ References for this group of symptoms: A27, H39, K21, N13, O6, P24, S33, U8, U9.

d/ References for this group of symptoms: A26, A27, B16, B29, C10, C14, C15, C43, C44, C45, C47, D19, K21, L20, L22, M10, M18, M42, N10, O5, O6, P24, R12, U9, V18, W19, W21, Z3.

e/ Slight to moderate drop in platelets: from 3 10^5/µl to 1.8-0.8 10^5/µl.

f/ Slight to moderate drop in granulocytes: from 6 10^3/µl to 4.5-2.0 10^3/µl.

g/ Slight to moderate drop in lymphocytes: from 3 10^3/µl to 2.0-1.0 10^3/µl.

h/ References for this event: A25, B16, L4, O6.

Table 15

Symptoms for midline dose range 2.0-3.5 (Gy)
[Y7]

Symptom	Postexposure time: Hours 0	4	8	12	16	20	Days 1 (24)	2	3	4	5	6	7	Weeks 1	2	3	4	5	6
Nausea[a]	——70-90% moderate——																		
Vomiting (retching)	——50-80% moderate——																		
Anorexia	——90-100%——																—60%—-		
Diarrhea (cramps)	— ← ~10% moderate													Moderate →			—40—-		
																	60%		
Fatigue[b]	——60-90% moderate——																		
Weakness	——60-90% moderate——																		
Hypotension																			
Dizziness																			
Disorientation																			
Bleeding[c]														—(d)—			—10-50%—-		
																	moderate		
Fever														—(e)—					
																	10-80%		
Infection														—(f)—			moderate		
Ulceration																	—30%—-		
							(g)										moderate		
Fluid loss/electrolyte imbalance																			
Headache																			
Fainting																			
Prostration																			
Death[h]																≤5-50% →			

a/ References for this group of symptoms: A25, A26, A27, B17, B29, B52, C14, C39, C40, C47, D19, E11, G2, G25, G26, G27, G28, H39, H40, I20, J2, K21, L20, M10, M42, N11, N13, O5, O6, P24, R6, R23, S33, S34, S35, T5, T23, W19, W20, W21, W22, Y6, Z3, Z4.

b/ References for this group of symptoms: A24, A27, B16, G2, L10, M42, O6, U9, V18, Y6, Z3.

c/ References for this group of symptoms: A26, A27, A28, B16, B17, C14, D19, F14, I11, J2, K21, L10, L20, L22, M10, M42, N10, O5, O6, P24, R6, S34, T23, U9, W19, W20, Z3.

d/ Moderate drop in platelets: from 3 $10^5/\mu l$ to 0.8-0.1 $10^5/\mu l$.

e/ Moderate drop in granulocytes: from 6 $10^3/\mu l$ to 2.0-0.5 $10^3/\mu l$.

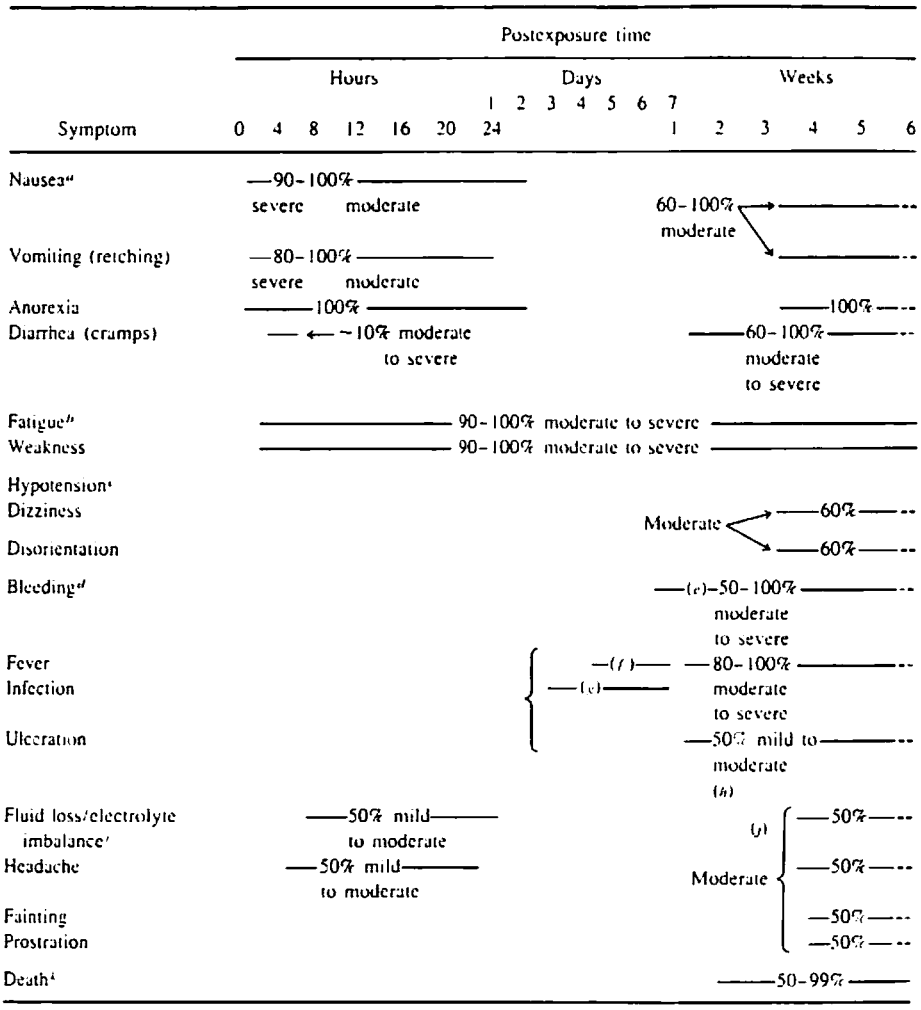
f/ Moderate to severe drop in granulocytes: from 3 $10^3/\mu l$ to 1.0-0.4 $10^3/\mu l$.

g/ Epilation.

h/ References for this event: A25, A27, B16, L4, O6.

Table 16

Symptoms for midline dose range 3.5-5.5 (Gy)
[Y7]



a/ References for this group of symptoms: A24, A25, A26, A27, B16, B17, B29, C14, C39, C40, E11, G25, G26, H39, H40, I20, J2, J19, L20, M42, M43, N10, N11, O5, O6, P24, R12, R23, S33, S34, T5, T23, W19, W20, U9, Z3, Z4.

b/ References for this group of symptoms: A24, A26, A27, B16, G27, H40, I20, M18, O6, P24, S33, U9.

c/ References for this group of symptoms: M43, R12.

d/ References for this group of symptoms: A24, A26, A27, B16, B17, C14, C41, C47, D19, I11, J19, L8, L10, L20, L22, M10, M42, M43, O6, P24, R23, S33, S34, U9, W19, W20, W21, Z3, Z4.

e/ Severe drop in platelets: from 3 10^5/µl to 0.1 10^5-0/µl.

f/ Severe drop in granulocytes: from 6 10^3/µl to 0.5 10^3-0/µl.

g/ Severe drop in lymphocytes: from 3 10^3/µl to 0.4-0.1 10^3/µl.

h/ Epilation.

i/ References for this group of symptoms: A27, B16, L10, O6, R12, U9.

j/ Mild intestinal damage.

k/ References for this event: A25, B16, L4, O6.

Table 17

Modification of $LD_{50/30}$ for single doses, according to direction of the beam
[M28]

	Dog	Sheep	Pig	Goat
Body mass, kg	7-13	32-57	av. 62	60-95
Radiation	1 MV x rays, point source	1 MV x rays, point source	2 MV x rays, point source	2.5 Mev Gamma rays, planar source
Source to midplane of animal (m)	2.1	2.0	2.14	0.25
Diameter of trunk (cm)	14	20-25	28	30
Irradiation conditions	Conscious	Conscious	Conscious	Sedated
Mortality period (days)	0-30	0-60	0-30	0-60
LD_{50} mean ± SE (Gy) a/				
Unilateral exposure	3.37 ± 0.09	2.65 ± 0.11	3.79 ± 0.11	3.94 ± 0.21
Bilateral exposure	2.80 ± 0.08	2.20 ± 0.15	3.16 ± 0.17	3.35 ± 0.26
Difference	0.57	0.45	0.63	0.59
Ratio	1.20	1.20	1.20	1.17
Coefficient of variation				
Unilateral exposure	0.15	0.17	0.11	0.20
Bilateral exposure	0.25	0.28	0.16	0.32

a/ Air kerma at midplane of exposure volume in absence of animal.

Table 18

Examples of adult erythropoietic bone-marrow distributions in several mammalian species
(per cent)

Site	Mice [C22]	Rats [V17]	Dogs [G8]	Monkeys [T15]	Humans: Men [W8]	Humans: Women [W8]	Humans [M30]
Skull)	19.1	4.1	1.0	8.7	8.3	9.4	7.3
Mandible)		2.6	0.1	2.2	1.0	0.7	0.5
Two clavicles	-	0.21	-	0.7	1.0	0.9	0.7
Two scapulae	-	1.5	5.1	3.9	3.8	2.8	2.2
Upper limbs	(5.7)	(8.6)	(11.1)	(12.2)	-	-	(3.7)
Two humeri	4.1 a/	7.0	10.8	9.2	-	-	3.7
Two radii	-	0.4	0.1	1.5	-	-	0
Two ulnae	-	1.0	0.1	1.3	-	-	0
Two wrists (hands)	1.6	0.2	0.1	0.2	-	-	-
Ribs	16.1	6.2	20.5	4.8	18.4	17.3	18.7
Sternum		4.1	2.8	1.5	3.9	3.6	2.6
Vertebrae	(38.1)	(29.7)	(42.6)	(33.1)	(35.9)	(36.6)	(24.4)
Cervical	-	2.4	6.7	2.2	4.1	5.1	4.0
Thoracic	-	9.9	17.6	12.3	17.9	18.0	9.9
Lumbar	-	7.6	15.0	17.0	13.9	13.5	10.5
Sacrococcygeal	8.2 b/	15.5 c/	3.3	1.6	7.7	7.4	7.9
Two hip bones			8.9	12.9	19.7	21.3	20.7
Lower limbs	(12.8)	(39.4)	(7.9)	(20.0)	-	-	(10.6)
Two femurs	6.0	16.9	7.2	13.3	-	-	10.6
Two patellae	-	-	0.0	0.1	-	-	-
Two tibiae		13.5	0.6	5.9	-	-	0
Two fibulae	4.2	8.6	0.0	0.5	-	-	0
Two ankles (feet)	2.6	0.4	0.1	0.2	-	-	-

a/ Includes clavicles and scapulae.
b/ Pelvis.
c/ Includes caudal vertebrae.

Table 19

Accidental human total-body protracted exposures giving marrow doses higher than 1 Gy

Accident	Person(s)	Exposure duration (days)	Approximate marrow dose (Gy)	Outcome	Ref.
Rongelap	64	2	1.75	All survived	[C10]
China a/	Male	5-9	80	Died	[Y1]
	Male	5-9	40	Died	
	Female	5-9	8	Survived	
	Male	5-9	6	Survived	
	Female	5-9	4	Survived	
"Lucky Dragon"	23 fishermen	14 b/	2-7	All survived	[K4]
Algeria a/	Female	38	12-14	Survived	[J3]
	Female	38	12.5-14	Survived	
	Female	38	11-13	Survived	
	Female	38	10-12	Survived	
	Grandmother	38	> 40	Died	
Mexico	Son	24	29-52	Died	[M3]
	Wife	115	20-39	Died	
	Daughter	99	14-19	Died	
	Grandmother	90	18-29	Died	
	Husband	106	9.8-17	Survived	
Morocco c/	Grandmother	82	6-7	Survived	[N5]
	Grandfather	17	0.5-1.5	Survived	
	Cousin	17	2-3	Survived	
Brazil d/					

a/ Very inhomogeneous doses.
b/ Two thirds of dose on first day.
c/ Eight other members of the family and their relatives received exposures over 15 and 45 days, and all of them died. However, assessments of their doses are not available [N5].
d/ Ten individuals received high doses and four of them died (see paragraph 158).

Table 20

Incidence of pneumonitis in man after fractionated irradiation [M38]

Lung dose (Gy)/ number of fractions	Number of patients	Incidence of pneumonitis (%)	Primary diagnosis of tumour
30/15	6	33	Lung,
35/20			Hodgkin's disease
32/15	12	42	Lung,
36/20			Hodgkin's disease
30/10	12	67	Lung,
38/20			Hodgkin's disease,
45/30			Hemangiopericytoma, Thymoma
31/10	10	90	Lung, Breast,
42/18			Thymoma
41/16	14	86	Lung, Breast,
53/25			Sarcoma

Table 21

Clinical course after doses resulting in the bone-marrow syndrome

Phase	Approximate duration
Prodromal	1- 7 days
Latent	7-20 days
Critical	Second or third week to 7 weeks
Recovery	8-15 weeks

Table 22

Gastrointestinal prodromal symptoms at 48 hours in 111 cancer patients [LB]

Symptoms	Doses (Gy)	
	D 10%	D 50%
Anorexia	0.3	0.6
Nausea	0.4	1.2
Vomiting	0.5	1.8
Diarrhoea	0.6 a/	3.0

a/ By six weeks.

Table 23

Summary of symptoms, time course and prognosis in the bone marrow syndrome in man (adapted from [112])

Dose range (Gy)	Prognosis	Appropriate time of delay for nausea and vomiting	Time of delay for critical period	Main symptoms	Time of recovery	Time of death
0-1	Excellent	-	-	-	-	-
1-2	Excellent	3 hours	-	Moderate leucopenia	Several weeks	-
2-6	Uncertain	2 hours	4-6 weeks	Leucopenia, haemorrhage infection	6-8 weeks 1-12 months	< 2 months
6-10	Uncertain	1 hour	4-6 weeks	Leucopenia	Prolonged	< 2 months
10-15	Poor	0.5-1 h	5-14 days	Diarrhoea, fever, electrolyte imbalance	-	< 2 weeks
> 60	Hopeless	0.5 hour	1-48 hours	Ataxia, lethargy	-	< 2 days

Table 24

ED 50 (Gy) for prodromal symptoms in ill cancer patients after whole-body acute or protracted exposure [L9]

Symptom	Exposure period	
	1 day (504 patients)	7 days (103 patients)
Anorexia	0.97	2.0
Nausea	1.4	2.6
Vomiting	1.8	4.9
Fatigue	1.5	2.6 (?)
Diarrhoea	2.3	5.3

Table 25

Main signs and symptoms in the critical phase of the bone marrow syndrome in man (adapted from [N5])

1:	Anorexia Nausea Vomiting Weakness, fatigue Prostration
2:	Sweating, fever Purpura Hemorrhage, epistaxis, gingival bleeding, haematemesis, melaena, haemoptysis Infection
3:	Erythema, epilation, scalp pain
4:	Abdominal pain Abdominal distention Diarrhoea
5:	Oliguria Hyperaesthesia, paraesthesia Ataxia Disorientation Shock Coma Death

Table 26

Minimum values of blood cell counts
after three accidents involving eight individuals
[N5]

(Acute doses from mixed fields in cases 1-7.)

Case a/	Dose (Gy) γ+n		Lymphocytes		Neutrophils		Platelets		Outcome
	Dose 1 b/	Dose 2 c/	Cells/ µl	Time (days)	Cells/ µl	Time (days)	Cells/ µl	Time (days)	
1	3.5+0.9	4.5	37	30	15	26	1900	19	Death (day 32)
2	3.4+0.9	4.3	322	15	48	26	28200	26	Favourable transplantation of bone marrow (day 29)
3	3.2+0.9	4.0	396	29	42	29	14000	26	As in case 2
4	3.3+0.9	4.1	80	29	0	33	25400	29	As in case 2
5	2.6+0.65	3.3	550	33	36	33	14200	26	As in case 2
			45	59					
6	1.6+0.45	-	390	9	916	33	53400	26	Favourable
7	5 +0.5	-	130	4	21	22	10000	22	Favourable
8	12	-	55	8	98	10	26000	10	Death (day 12)

a/ Cases 1-6: Vinca, Yugoslavia (1958).
Case 7: Mol, Belgium (1965).
Case 8: Brescia, Italy (1975).
b/ Dose 1: original estimated doses.
c/ Dose 2: Revised equivalent low-LET marrow dose (see Table 12).

Table 27

Minimum values of blood cell counts
after two accidents involving several individuals
where the inhomogeneous irradiation was prolonged over a few weeks
[N5]

Case a/	Dose (Gy) γ	Lymphocytes		Neutrophils		Platelets	
		Cells/ µl	Time (days) b/	Cells/ µl	Time (days) b/	Cells/ µl	Time (days) b/
9	12-14	41	17	0	9	21000	3
10	12-14	250	22	0-3	7-16	20000	9
11	11-13	124	24	10	21	20000	21
12	10-12	109	30	30	27	20000	26
15	6-7	416	18	63	8	35000	2
16	2-3	560	50	858	44	Normal	-
17	Acute 1.9, chronic 4.0	486	c/	1200	c/	100000	c/

a/ Cases 9-12: Algeria, 1978.
Cases 15-17: Morocco, 1984.
b/ After the end of exposure.
c/ During the first week of hospitalization.

Table 28

Representative effects and related acute doses after whole-body irradiation in man
(adapted from [N5])

Threshold for detection of the effect	Dose (Gy) a/
Chromosome aberrations and sperm-count depression	0.05-0.25
Electroencephalography modifications	0.25-0.5
Vomiting in 10% of exposed individuals	0.5 -1.5
Transient disability and easily detectable haematological changes	1.5 -2

a/ Whole-body dose, which may vary by as much as ± 50%; expressed as midline doses.

back to first page

APPENDIX

Acute radiation effects in victims of the Chernobyl nuclear power plant accident

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Introduction

1. This Appendix sets out the essential findings of the clinical observation of a group of patients suffering from acute radiation sickness following the accident at the Chernobyl nuclear power plant on 26 April 1986. The observations were conducted at the specialized treatment centre in Moscow over a period of two years.

2. An initial report on the accident was submitted by the Soviet representatives to the Post-Accident Review Meeting held at the International Atomic Energy Agency in August 1986 and was summarized in IAEA Safety Series Technical Report No. 75 [I18] and in [G33]. The proposal to present this information in its present form was endorsed at the thirty-sixth session of UNSCEAR in March 1987.

3. The basic information on the radionuclide releases and the types of exposures of the irradiated persons coincided with the expected pattern for an accident at a nuclear power plant of similar type: as much as 100% of gaseous fraction of the noble gases and nuclides may have escaped from the plant; caesium, iodine and tellurium isotopes accounted for up to 10-20% of the nuclide inventory, and other radionuclides for up to 30% [I18].

4. The plant personnel and auxiliary staff present at the industrial site in the immediate vicinity of the accident zone were subjected to the combined effect of radiation from several sources: (a) short-term external gamma/beta radiation from the gas emission cloud (in the case of persons in the immediate area of the accident zone at the time of the explosion); (b) external gamma/beta radiation of decreasing intensity, from fragments of the damaged reactor core scattered over the industrial site; (c) inhalation of gases and aerosol dust particles containing a mixture of radionuclides; and (d) deposition of these particles on the skin and mucous membranes at the time of the intensive generation of steam or dust and the wetting of clothing (as a result of them being blown or washed off contaminated objects).

5. However, the most significant factor was the general, external and relatively uniform whole-body gamma-irradiation and the beta-irradiation of extensive body surfaces, coupled (except in two cases) with a very small intake of nuclides through inhalation, predominantly of radioiodine and caesium isotopes. Thus, the basic clinical picture was that of a distinctive acute radiation sickness caused by gamma-irradiation of the whole body and by beta-irradiation of extensive areas of the skin surface.

6. Direct and indirect dosimetry methods were used to determine the nuclide content in the body. A great many tests were carried out, both while the victims were alive and (in 28 cases) after they had died, so that it was possible to estimate the nuclide content in the body and the resultant dose levels. An example of these types of analyses is shown in Figure A.I., giving the distribution of various radionuclides in the lungs.

7. The iodine isotope content in the thyroid gland was determined repeatedly (as many as four to six

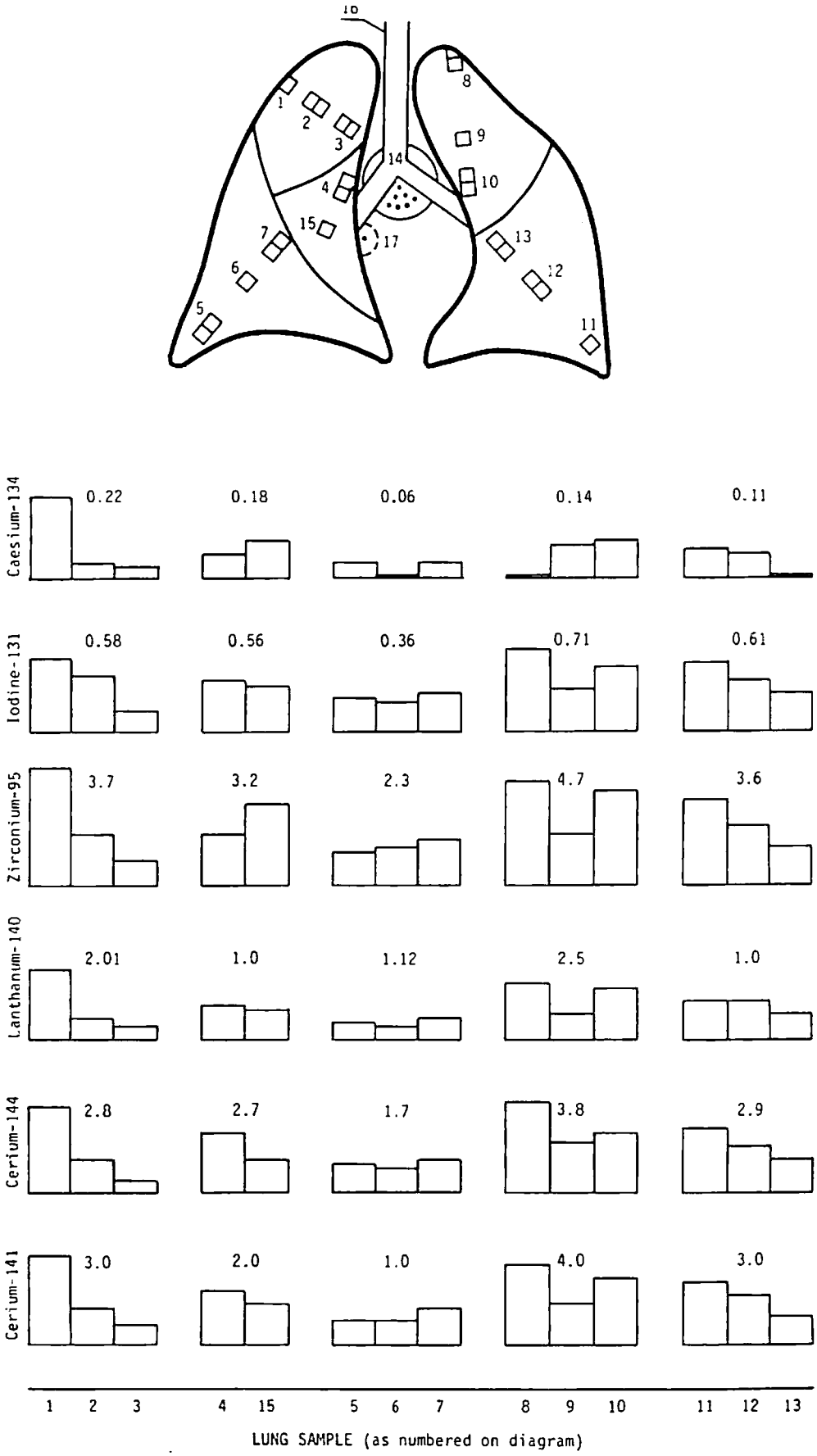

Figure A.I. Location of lung samples taken at the time of autopsy and distribution of the main radionuclides in lung samples. The number above each sample group indicates the approximate relative average value.

times) from the second day after the accident. These measurements showed that ^{131}I accounted for 80 ± 20% of the total activity of all iodine isotopes, ^{133}I for 15 ± 10%, and the remaining isotopes (^{123}I, ^{124}I, ^{126}I, and ^{130}I) for not more than 2%.

8. The calculations for estimating intake quantities from the thyroid measurements were performed according to the recommendations of the International Commission on Radiological Protection [I19]. On the basis of the distribution of thyroid doses in exposed individuals (Table A.1), it may be stated that in the overwhelming majority of cases the thyroid doses were below the levels likely to cause direct injury to that organ (< 3.7 Sv) or of significantly influencing the clinical picture during the onset of acute radiation sickness. Low radioiodine dose levels were also suggested by the post-mortem nuclide measurements of the 28 persons who subsequently died.

9. Internal dose values according to post-mortem measurements for 6 patients are shown in Table A.2. The maximum amount of ^{137}Cs and ^{134}Cs incorporated activity was 7.4 MBq, except for two patients with extensive steam burns, which allowed intake of nuclides through the wound. The post-mortem dosimetry gave 40 and 80 MBq of ^{137}Cs plus ^{134}Cs, and 450 and 1,100 MBq of ^{131}I, for these two patients, respectively. The whole-body internal doses in these two individuals from these nuclides were estimated as approximately 1 Sv and 2 Sv during the two to three weeks before they died, which are commensurable with their external gamma doses. This fact was taken into account during the interpretation of clinical data. Internal doses for other patients did not exceed 1-3% of the external irradiation doses.

10. The transuranic elements (e.g., ^{239}Pu) were studied in urine specimens from 266 persons (635 analyses), including, in some of the cases, analyses conducted before and after the administration of pentacine. The urine activity values and a negative finding after chelation treatment confirmed the absence of a significant plutonium contamination of all the patients observed. Post-mortem tests by alpha spectrometry for transuranic elements showed their presence (74-300 Bq per organ) only in the lungs; curium accounted for as much as 90% of the specimen activity, and plutonium and americium for 10%.

11. Gamma-spectrometric analysis of the first specimens within 36-39 hours of the accident failed to reveal any sign of $^{22,24}Na$ activation, which confirmed that neutron irradiation of the victims was not significant.

12. For most of the victims, the energy peaks of more than 20 radionuclides were detectable in the spectrum of their whole-body gamma measurements; however, apart from the iodine and caesium isotopes previously mentioned, the contribution to the overall dose from the others (^{95}Nb, ^{144}Ce, ^{140}La etc.) was negligible. These measurements, performed while the victims were still alive, were also confirmed through the analysis of autopsy specimens (approximately 35 specimens from each deceased person) (Figure A.I).

13. The dose levels from external irradiation were reconstructed from the indication of several measurements on the basis of previous experience. Subsequently, in three cases with a lethal outcome, these findings were refined using methods earlier proposed by Kraytor for clothing fabrics [B31] and according to the electron spin resonance technique for dental enamel [T16]. These measurements agreed within ± 20% with the dose estimates based on clinical and biological criteria.

14. The total number of affected individuals among the persons present at the reactor site in the early hours of 26 April 1986 was 203, as given in the report presented by Soviet representatives at the Post-Accident Review Meeting in August 1986 [118]. Of these, 115 were treated, beginning on day 2, at the specialized treatment centre in Moscow; it was this group that provided most of the scientific analytical data discussed in this report. At other hospitals in Kiev there were only 12 patients with a clearly defined clinical pattern of second-degree acute radiation sickness and one person with fourth-degree acute radiation sickness, a fact that cannot in any substantial way alter the overall assessment of the data for the entire group of victims.

15. The increase in the total number of affected individuals from 203 to 237, announced in November 1986, was due solely to persons suffering from first-degree acute radiation sickness. There were 31 persons suffering from first-degree acute radiation sickness at the special treatment centre in Moscow and 109 persons in Kiev. The task of establishing a diagnosis distinguishing between first-degree acute radiation sickness and ordinary somatic diseases according to generally accepted criteria is a complex one, and one that continued throughout 1986. On the whole, a critical analysis of the data shows a decrease in the number of persons suffering from first-degree acute radiation sickness in comparison with the number given originally. At the time of writing this report, up to three quarters of these persons are for all practical purposes healthy. Their clinical signs of reaction to the accident during the first three months were neither individually significant nor typical of a reaction to irradiation. Table A.3 shows the distribution of patients with acute radiation sickness according to its degree of severity [B31] in the group selected for scientific analysis.

A. INITIAL DIAGNOSIS OF ACUTE RADIATION SICKNESS

16. The medical unit serving the plant was informed of the accident within 10-15 minutes of its occurrence. First aid to the affected individuals was provided by middle-level medical personnel and emergency teams over a time period from 30-40 minutes to 3-6 hours after the accident. First aid consisted in the evacuation of the victims from the industrial site, the simplest forms of medical attention, the administration of antiemetic and symptomatic (sedative, cardiotonic) drugs, the distribution of potassium iodide and the transportation of persons suffering from a pronounced primary reaction to the medical unit. During the first

12-24 hours after the accident, other persons who were in satisfactory condition were urged to go to the medical unit for examination; a total of 132 persons were hospitalized there during the first 12 hours. One person with severe thermal burns died during the first hour. Another, a reactor operator, could not be found; his working station was located in the collapsed high-activity zone.

17. Within 12 hours, a specialized emergency team arrived at the site and began work. Within 36 hours, this team, together with the on-site medical unit, examined more than 350 persons and carried out approximately 1,000 blood tests, each person undergoing two to three such tests. The treatment with potassium iodide was continued.

18. Within the first three days, 299 persons suspected of suffering from acute radiation sickness were sent to the specialized treatment centre in Moscow and to hospitals in Kiev, and over the subsequent days some 200 additional persons were admitted for examination.

19. The primary diagnostic criteria for assessing the priority for hospitalization were the presence, time of onset and intensity of nausea and vomiting and of primary erythema of the skin and mucosae, and a decrease of the lymphocyte count in the peripheral blood to below $10^9/l$ during the first days following irradiation.

20. The diagnosis of acute radiation sickness was subsequently confirmed in 99 of the 128 persons (firemen, Unit 4 operators, turbine-room duty officer and auxiliary personnel) admitted to the specialized treatment centre in Moscow during the first two days and in six of the 74 victims hospitalized during the following three days. This is an indication of the high specificity of the screening methods used. An additional 10 cases of minor acute radiation sickness were diagnosed among persons present at the site at the time of the accident, who were later admitted to the hospital facility for a variety of reasons. In the reception area the patients were monitored again for contamination and, if necessary, subjected to decontamination measures (washing under a shower with ordinary soap and change of underwear). Blood and urine samples were taken for a quick test of the presence of radionuclides; the patients also underwent measurements (repeated a further 4-6 times during the first 6-10 days) of the radioactive iodine content in the thyroid. Measuring devices consisting of a scintillation detector or a semiconductor detection unit were used for the whole-body counting of radionuclide activity.

B. THE BONE MARROW SYNDROME AND ITS TREATMENT

21. Dosimetric data, together with an analysis of the circumstances of the accident and the presence in a considerable number of the victims of obvious primary reaction symptoms (nausea, vomiting, diarrhoea, hyperaemia of the mucosae and skin, lymphopenia), confirmed that the principal modes of irradiation had been: (a) by external, relatively uniform gamma-radiation; and (b) by deposition of beta/gamma-emitting nuclides on the skin. Radionuclide ingestion was below the level likely to cause acute radiation injury. As already noted, two patients suffered from all three of these irradiation modalities, in combination with extensive steam burns.

22. The important diagnostic task during the first few days after the accident was the assessment of the degree of severity of the bone marrow syndrome resulting from the external gamma-irradiation dose. This was possible through the use of previously devised methods, which are based on the number of lymphocytes and on chromosome aberrations in peripheral-blood lymphocytes or on the incidence of chromosome aberrations in bone marrow cells [B31, B50, G24]. These data were later transformed into a prognosis of the overall dynamics of the blood picture. A subsequent re-assessment of dose levels involving a larger sample of cells scored revealed not more than 5-10% changes in the estimated doses.

23. Dose-effect relationships for these indicators had been derived earlier through the analysis of relatively uniform accidental or therapeutic irradiations of human subjects having normal initial haematological characteristics and exposed to well established doses [P22]. Figure A.II shows the curves (and analytical expressions) for the relationships between the dose and the blood lymphocyte count for each of the first nine days and the average lymphocyte count on days 4-7 and days 1-8 after irradiation. The radiation dose received by each person was estimated according to the number of chromosome aberrations (dicentrics) in a blood-lymphocyte culture, using a dose-effect curve for 100 first-mitosis cells that had been obtained after whole-body gamma-irradiation to treat acute leukaemia patients during a period of full clinical and haematological remission [P23].

24. The formula for calculating of the dose is as follows:

$$D = (-a + \sqrt{a^2 + 4by})/2b$$

This assumes that the yield of dicentrics shows a linear-quadratic dependence on dose:

$$y = (a \pm 2.24)D + (b \pm 0.56)D^2$$

where D is the average gamma-irradiation dose in the body (Gy), y is the dicentric count per 100 cells; $a = 8.36$; $b = 5.70$.

25. Up to day 7 after the accident, the estimates of the average dose of total gamma-irradiation were refined, mostly on the basis of the peripheral-blood lymphocyte counts but also, in the more severe cases and to a lesser degree, on the chromosome aberration count. This made it possible to divide the patients into various prognostic groups [B31], according to the severity of the bone marrow syndrome as follows (see Table A.3):

(I)	slight	(1-2 Gy)
(II)	intermediate	(2-4 Gy)
(III)	severe	(4-6 Gy)
(IV)	extremely severe	(6 Gy and above)

It was also possible to separate those persons who received doses of less than 1 Gy.

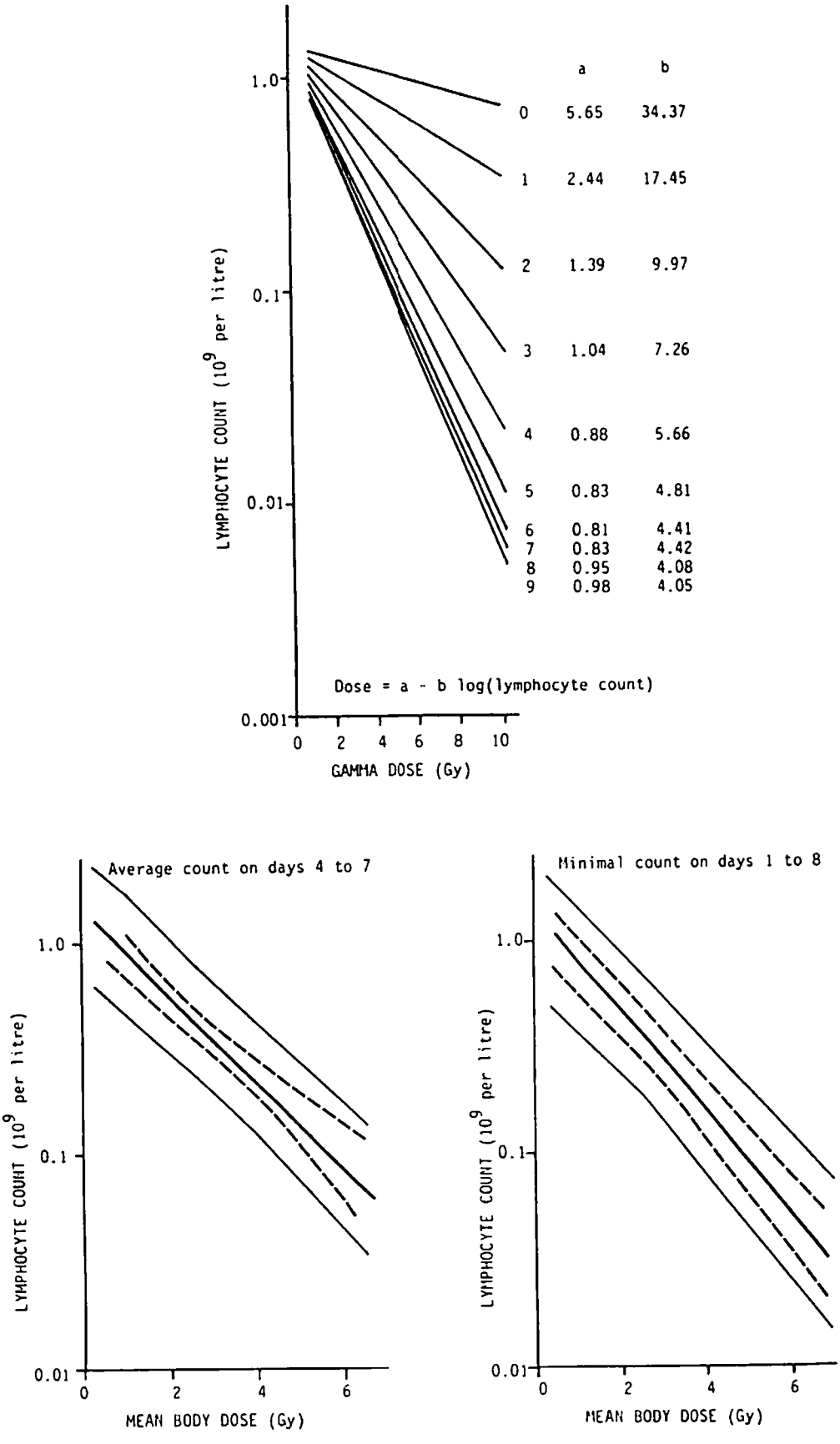

Figure A.II. Estimation of the total gamma dose according to the blood lymphocyte counts. Upper panel: Dose-effect relationships for lymphocyte counts at the days post irradiation shown on the curves; analytical expression and coefficients of these relationships. Lower panels: Curves showing the dependence of average lymphocyte counts on days 4-7 and the minimum lymphocyte count on days 1-8 as a function of the irradiation dose.

26. Particular attention during the first days was directed at identifying persons with an extremely severe and irreversible degree of myelodepression, for whom an urgent decision was required regarding a bone marrow transplant. Additional signs providing further evidence that a patient belonged to this group were (a) vomiting during the first half-hour and of diarrhoea during the first 1-2 hours from the start of irradiation; (b) a swelling of the parotid glands during the first 24-36 hours: and (c) the ascertainment of an irreversible degree of myelodepression using a diagnostic table previously devised (Table A.4).

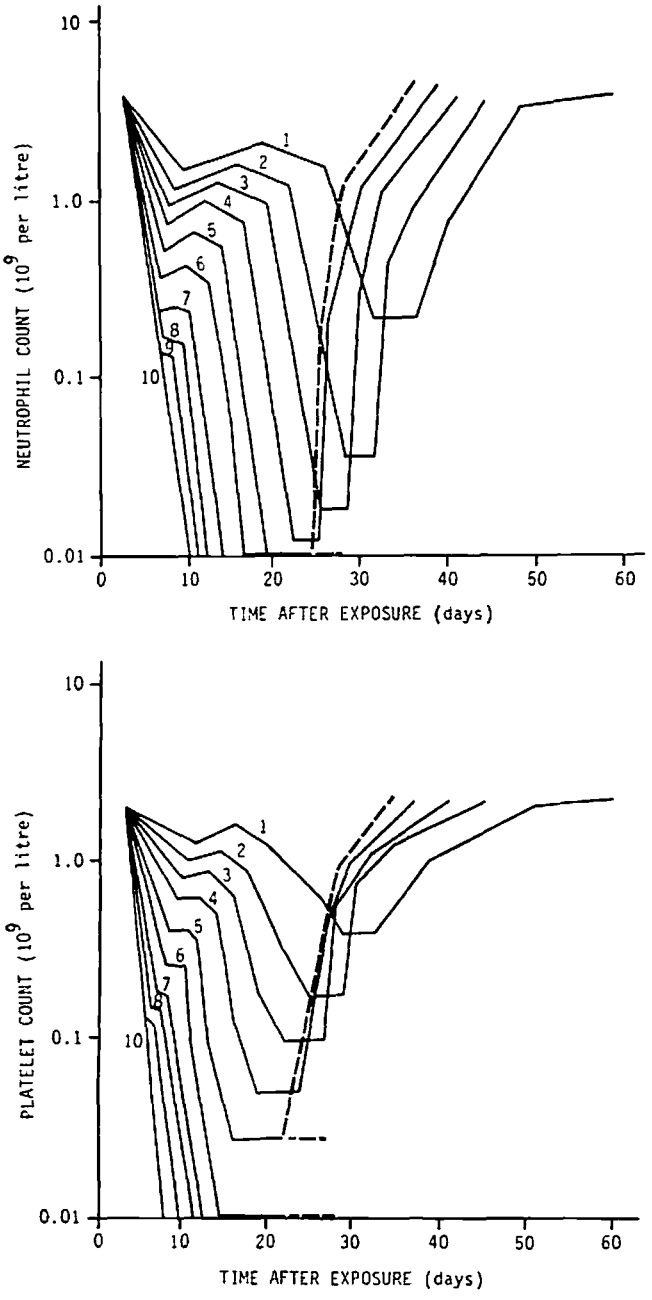

Figure A.III. Standard curves showing the changes of the neutrophil and platelet counts after various doses (numbers on the curves indicate the dose in Gy) in the case of relatively uniform whole-body gamma irradiation of human subjects. [B31]. (The broken segments of the curves at doses of 5-6 Gy indicate that recovery may not occur at these times in all patients.) Upper panel: Neutrophils. Lower panel: Platelets.

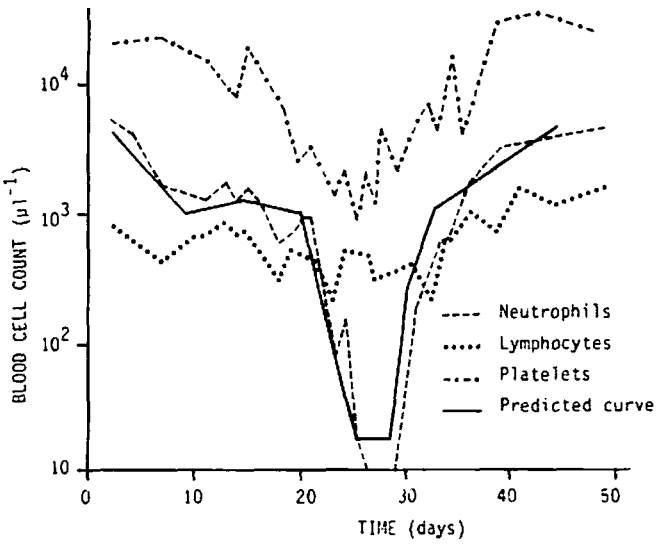

Figure A.IV. Example of the changes in neutrophils, lymphocytes and platelets observed in a patient (case 39) suffering from acute radiation sickness (estimated dose 2.4-3.3 Gy) and the predicted neutrophil curve for a total gamma dose of 3.0 Gy.

27. The results of numerous biochemical, immunological and biophysical indicators are undergoing processing and analysis at the time of writing this report. None of these indicators is as informative as the signs described above. However, it may be noted, for example, that hyperamylasemia was used as a supplementary prognostic test.

28. On the basis of the estimated dose, a prediction was made using standard curves [P22], of the overall trend with time in the neutrophil and platelet counts (Figure A.III). By way of example, Figure A.IV shows the real and predicted neutrophil curves for one patient (case 39). The total gamma-irradiation doses estimated according to the average lymphocyte count from day 4 to day 7 and according to the dicentric yield totalled 2.4 and 3.3 Gy, respectively. The patient's measured neutrophil curve almost coincided with the predicted neutrophil curve for 3.0 Gy of total gamma-irradiation.

29. The calibration curves of neutrophil counts as a function of dose were also used for a final assessment of the magnitude of the total gamma-irradiation dose. The dose calibration curve was chosen that coincided with the measured second depletion phase. Also, use was made of the dose dependency of the second depletion phase, such as the time required for the neutrophil count to decrease to 0.5 10^9/l or the time to reach the minimum of the second depletion phase (Figure A.V).

30. For relatively low doses (1.0-1.5 Gy), the diagnosis was finally established over time periods of up to three months only in those cases that showed typical post-irradiation neutrophil and/or platelet time courses with distinct second depletion and restoration phases. The latter generally developed beginning in the fourth to the fifth week after irradiation. In order to determine these changes, it was necessary to carry out blood analyses not less than two or three times a week

over a period of two to three months. Examples of these curves are shown in Figure A.VI: (a) case 48: doses, estimated according to the lymphocyte count on days 4-7 and the dicentric yield were 1.1 Gy and 1.4 Gy, respectively; and (b) case 97: doses, estimated according to the lymphocyte count on day 9 (the first blood analysis was made at this time because of late arrival) and the dicentric yield were 0.3 Gy and 0.9 Gy, respectively. It should be noted that in the case of low doses, the minimum level for neutrophils occurred later (day 30-50) than for platelets (day 20-40), and the reduction in the number of platelets and their recovery were more clearly pronounced than for neutrophils.

31. On the basis of all these data, a diagnosis of acute radiation sickness with the bone marrow syndrome of the first, second, third and fourth degree of severity was definitively established for 31, 43, 21 and 20 patients, respectively (see Table A.3). The analysis of the observations carried out on these patients is the subject of the exposition that follows.

32. Clinical manifestations of the bone marrow syndrome corresponded to the level and duration of post-irradiation pancytopenia (neutrophils 0.1-0.5 $10^9/l$, platelets 10-20 $10^9/l$). The main signs were fever, infectious complications and petechial haemorrhages in the skin and oral mucosa.

33. Treatment was based on the principles of supportive therapy, including isolation, antimicrobial decontamination of the intestine, administration of systemic antibiotics and replacement transfusions of blood cells. In cases in which there was a prognosis of irreversible myelodepression, transplantations of allogeneic bone marrow and embryonic human liver cells were performed.

34. All patients suffering from a bone marrow syndrome of the second, third or fourth degree were individually accommodated in ordinary hospital rooms. These were adapted to ensure (a) barrier nursing; (b) air sterilization by means of ultraviolet lamps; (c) strict observance by the attending personnel of hand disinfection on entering and leaving the room; (d) mandatory use of individual or disposable gowns, masks, and caps; (e) antiseptic decontamination of footwear; (f) changes of underclothing for patients at least once a day; (g) use of antiseptic agents for washing the walls and floor of the room and the items of use; and (h) individual assignment of antiseptically treated nursing items in the room. This regimen made it possible to maintain the micro-organism population at less than 500 m^{-3} in the room air. Ordinary food was served, with the exclusion of raw vegetables, fruits and canned products.

35. Prophylaxis against endogenous infections was by means of the internal administration of biseptol-480 and nistatin in amounts of six tablets and five million units per day, respectively, for one and 2-3 weeks prior to the development of agranulocytosis (leucocytes 1.0 $10^9/l$, neutrophils 0.1-0.5 $10^9/l$).

36. With the onset of fever, intravenous administration of two or three broad-spectrum antibiotics was prescribed, one of them being from the aminoglycoside

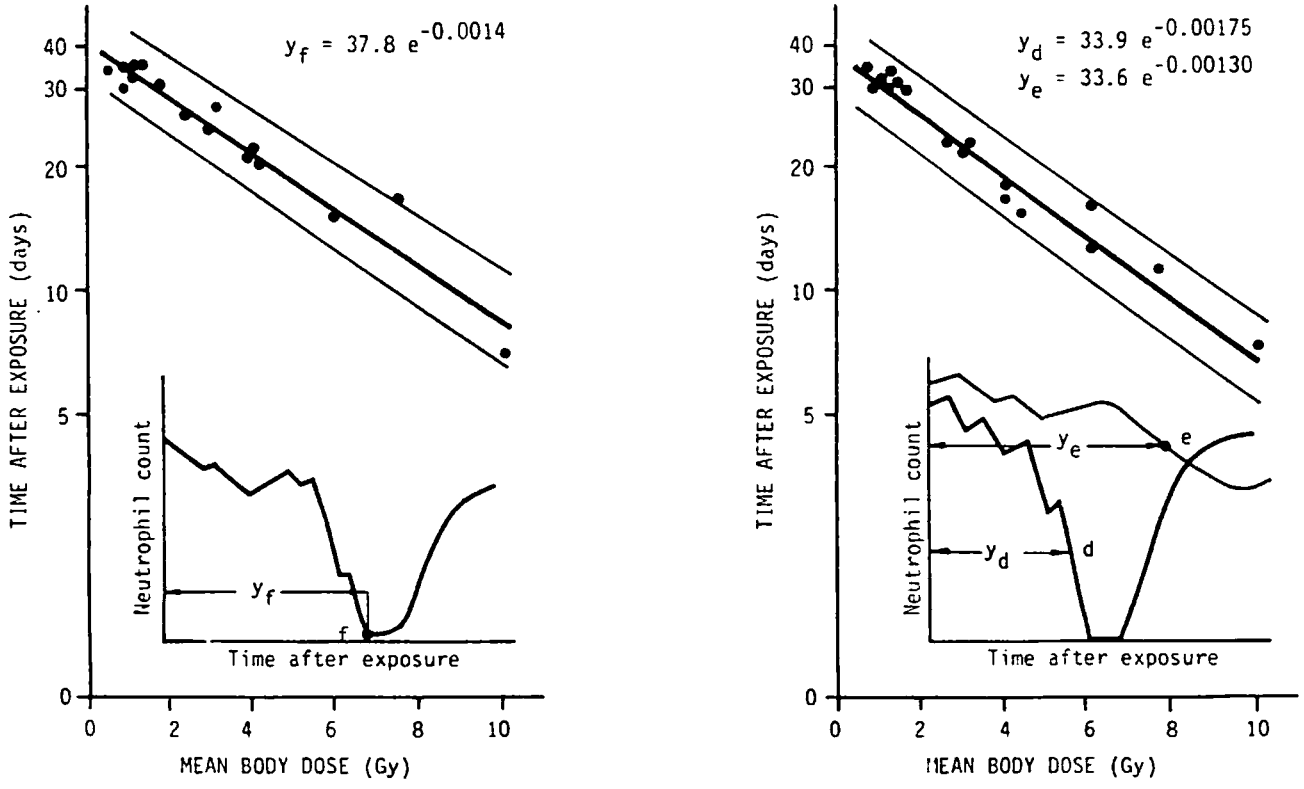

Figure A.V. Estimation of the total gamma dose according to two neutrophil counts: Left panel: time to the minimum of the second phase of depletion; Right panel: time to the "500 neutrophil day" or to the middle of the second depletion.

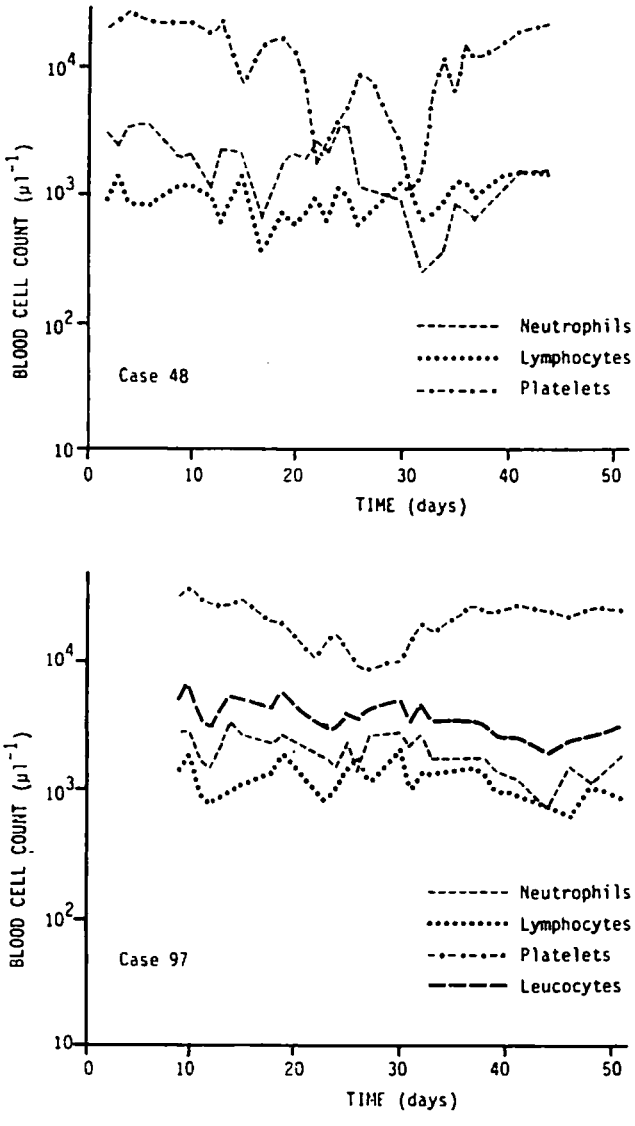

Figure A.VI. Changes in neutrophil, lymphocyte, platelet and leucocyte counts after whole-body gamma irradiation. Case 48, estimated dose 1.1-1.4 Gy. Case 97, estimated dose 0.3-0.9 Gy.

group (gentamicin or amicacin), the cephalosporins (cephzol, cephamecin, cephobide) and the semi-synthetic penicillins with activity against pseudomonas aeruginosa (carbenicillin, pipracil), all in maximum doses. This treatment reduced the fever in more than half of the patients. If within 24-48 hours there was no effect, extensive use was made of gamma globulin (Sandoglobulin), made available by the Sandoz Company (Switzerland). Six grams were administered intravenously every 12 hours, three or four times. The policy adopted was one of early empirical prescription of 1 mg/kg per day amphoterricin-B given intravenously, if the fever had not disappeared within a week of the above mentioned antibiotics, in combination with the intravenous administration of gamma globulin.

37. In this situation, acyclovir was used for the first time, and with good effect, in the treatment of patients with acute radiation sickness suffering from a herpes simplex infection. Not less than one third of the patients with third- and fourth-degree acute radiation sickness were affected by this virus. Acyclovir was not used prophylactically; experience has shown that this should be the case with high-dose whole-body irradiation. An ointment containing acyclovir proved effective in the treatment of skin lesions involving herpes virus.

38. The regimen described above for the empirical treatment to combat infection proved to be highly effective: there was no evidence that deaths were caused by bacterial infection alone in patients suffering from the bone marrow syndrome. This was so even among those with a severe or extremely severe form of acute radiation sickness, provided it was not complicated by burns, radiation-induced enteritis or acute secondary syndromes as a result of a bone marrow transplantation.

39. Blood screenings carried out while the patients were still alive or, in the case of those who died, posthumously, most often revealed epidermal staphylococcus. It will only be possible to evaluate the role of this form of bacteria as an agent of terminal septicaemia when these results have been compared with the histological data from analysis of various organs that have not yet been completed.

40. One of the undoubted successes in the treatment of the bone marrow syndrome in patients with acute radiation sickness was the rational use of fresh donor platelets for the prophylaxis and treatment of bleeding. To make these measures possible, the collection of the thrombocytes was organized on an urgent basis, using the method of fourfold thrombocytopheresis from individual donors at seven blood transfusion centres. For one transfusion, platelets obtained from a single donor (on the average 300 10^9 platelets in 200-250 ml of plasma) were used. Transfusions were carried out when the platelet level in the blood fell to 20 10^9/l or lower, with appearance of the first signs of bleeding. The infusions were repeated every 1-3 days. As a prophylaxis against acute secondary syndrome, the thrombocytes as well as the other blood components were irradiated with 15 Gy before infusion in order to inactivate the immunocompetent cells originating from the donor.

41. Platelet transfusion prevented life-threatening bleeding, even in patients with protracted (more than 2-4 weeks) and severe thrombocytopenia. The majority showed no signs of bleeding at all, although autopsies disclosed microcirculatory failures and porosity of the capillaries in a number of organs. In this situation, successful use was made of cryo-preserved allogeneic and, what is particularly important, autologous thrombocytes. The latter were obtained from patients with second- or third-degree bone marrow syndrome during the first days following irradiation (1-2 sessions) —this had no effect on the post-irradiation behaviour of their platelet counts—and were used with great effectiveness when the patients developed critical thrombocytopenia. No cases of refractoriness to thrombocytes transfusion were observed. On the average, from three to eight transfusions of standard amounts of thrombocytes (300 10^9 cells) were required for the treatment of a single patient with second- and third-degree acute radiation syndrome. Leucocytes

were not used for the prophylaxis or treatment of agranulocytic infections. Requirements for erythrocytes turned out to be considerably greater than expected, even for patients with second- and third-degree acute radiation sickness uncomplicated by severe radiation burns.

42. Figure A.VII illustrates the clinical record of one patient (case 21), showing the typical extent and duration of substitutive and supportive therapy for the bone marrow syndrome. In the case of this patient there were no life-threatening acute radiation injuries to the other tissues.

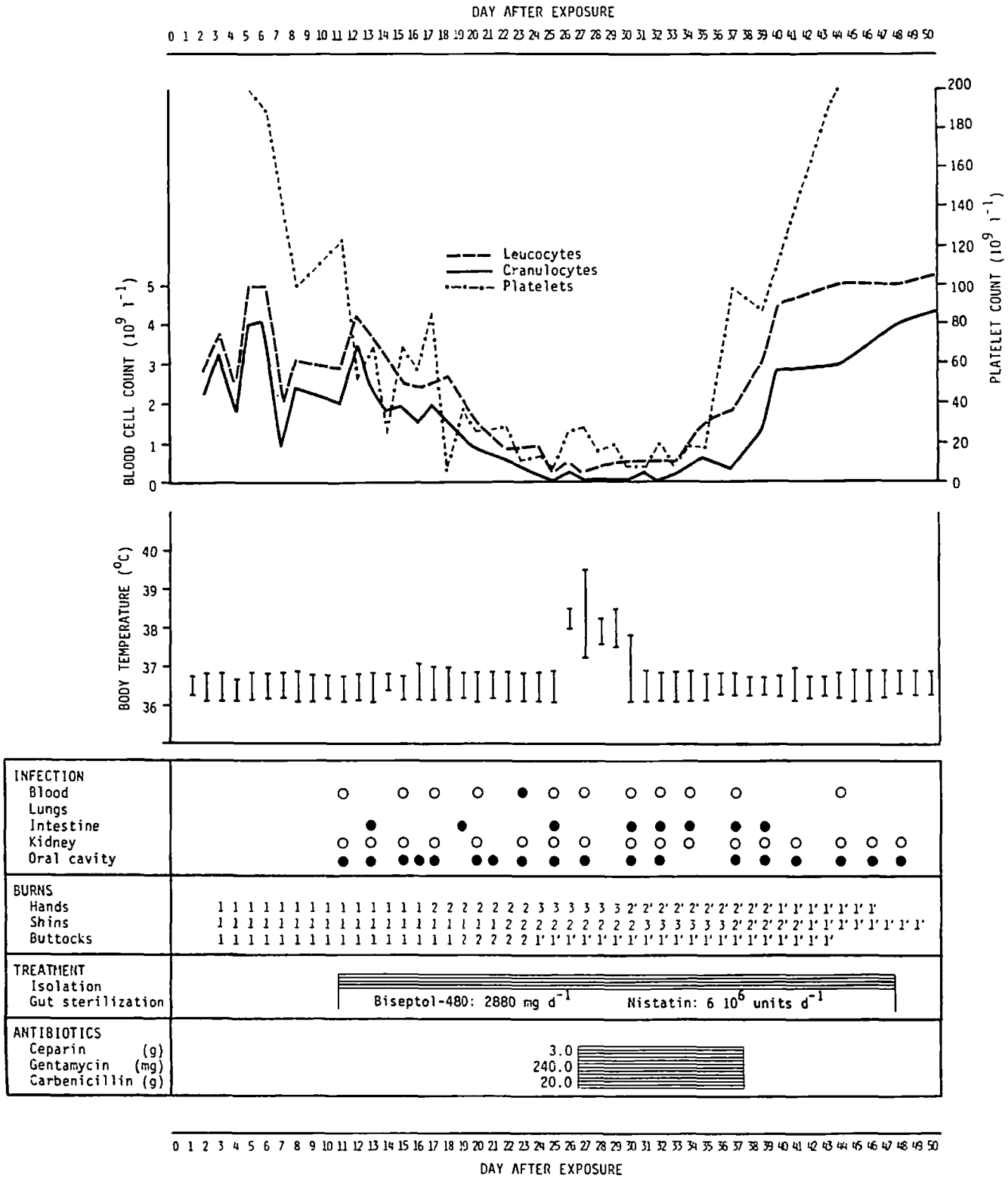

Figure A.VII. Clinical record of a patient (case 21) suffering from acute radiation sickness with bone marrow syndrome of an average degree of severity; whole-body gamma dose of 3.9 Gy, estimated according to the chromosome aberrations in blood lymphocytes. The circles in the infection section indicate the results of bacteriological tests of biological samples (blood, mucus, faeces, etc.); empty circles indicate absence of growth; solid circles indicate the presence of bacteria or fungi. The numbers in the burn segment designate the three degrees of radiation-induced burns.

43. The indication for an allogeneic bone marrow transplantation or an embryonic liver cell transplantation was the whole-body gamma-irradiation dose, estimated according to the peripheral-blood lymphocyte count and the chromosome aberrations at about 6.0 Gy and above. At these dose levels the prognosis expected was for irreversible or extremely protracted severe myelodepression.

44. A total of 13 allogeneic bone marrow transplantations and six embryonic liver cell transplantations were performed. The latter contains haemopoietic stem cells and a minimum of immunocompetent cells, which sharply lowers the risk of an acute secondary syndrome.

45. Seven patients who received allogeneic bone marrow transplant died between two and 19 days (15-25 days after irradiation) from acute radiation injuries to the skin, intestines and lungs.

46. Of six patients who did not suffer fatal skin burns and intestinal injuries and whose total doses had been estimated at between 4.4 and 10.2 Gy, two survived allogeneic bone marrow transplants (gamma-ray doses of 5.6 and 8.7 Gy). Both had haplo-identical female donors (sisters), rejected the partially functioning transplant (at days 32 and 35) and experienced a restoration of their own myelopoiesis, beginning at day 28.

47. Four patients who received allogeneic bone marrow transplantation died between 27 and 79 days (34-91 days after irradiation) from mixed viral-bacterial infections. Two of them had effectively functioning HLA-identical transplants (cases 6 and 28: total gamma-ray doses 5.2 Gy and 6.4 Gy, respectively), and two had early rejection (day 16 and 42) of "haplo + 1" and haplo-identical transplants, but during times when their own myelopoiesis was restored (cases 5 and 16: gamma-ray doses 4.4 and 10.2 Gy).

48. Similarly, all the patients who received embryonic liver cell transplants died from skin and intestinal injuries within a brief period (14-18 days after irradiation), with the exception of one woman of 63 years (case 8, embryonic-liver cell transplant from an 18-week male donor), who lived 30 days, having received a dose of 8-10 Gy. At death (17 days after transplant), numerous mitoses were discovered, against a background of severe marrow pancytopenia, and all the cells had a female karyotype, i.e., regeneration of the host bone marrow had begun.

49. This experience confirms that in an emergency situation like the one described, the group of persons for whom bone marrow transplants would be indicated with a reasonable prospect of success is extremely limited. Seven of 13 patients died as a result of skin and intestinal injuries before bone marrow engraftment could be expected. In Table A.5 a comparison is given of survival or cause of death of patients reiceiving bone marrow transplantations and of patients in a control group.

C. OTHER INJURIES AND THEIR TREATMENT

50. The extensive skin lesions caused by beta-radiation represented a distinctive feature of the injuries suffered in this emergency situation. Radiation-induced skin burns in firemen and personnel from the plant were observed only in combination with radiation injury to haemopoiesis and were therefore an integral part of the general acute radiation sickness.

51. This situation may be regarded as one in which there is an extremely non-uniform distribution of dose as a function of the depth of penetration within the body; the skin doses are estimated to be 10-20 times greater than the bone marrow doses. There was a definite correlation in the severity of the injuries in both tissues.

52. Table A.6 and Figure A.VIII show the distribution of cases involving radiation-induced skin burns of various degrees in patients with acute bone marrow syndrome of different severity. Skin injuries were observed in more than one half of the patients and in virtually every patient suffering from third- or fourth-degree bone marrow syndrome.

53. The aggravating contribution of radiation-induced skin injuries to the overall clinical picture arose not only from their severity, but also from the duration of the injuries, characterized as they are by recurrences of the pathological process. As a rule, the burns occurred at different times on various parts of the body. The most frequent locations during the early period were the wrists, the face, the neck and the feet; later, lesions appeared also on the chest and back, and still later on the knees, hips and buttocks. Exceptions to this sequence were encountered in individual cases.

54. The development of the injury was similar to that described by Cronkite et al. [C16], but in a more severe form. The diffuse hyperaemia in the first few days (primary erythema) was followed after 3-4 days by a period of latency. Secondary erythema in the more severe cases developed after 5-6 days and in the majority of patients from day 8 to day 21. Depending on the degree of the injury, it reached a level of dry (first-degree radiation burn) or moist desquamation with the development of blisters (second-degree burn), or the formation of vesicular-ulcerated and ulcerated-necrotic dermatitis (third- to fourth-degree burn). The re-epithelialization of the desquamated surfaces continued for two or three weeks from the occurrence of the visible injury to the skin. In six patients, the healing of the burns over skin areas involving deep necrosis did not begin until the end of the second month. A characteristic feature in the time course of the burns, and one which could be monitored throughout in this group of victims, was the appearance of recurrent waves of erythema, beginning by the end of the fourth week and continuing up to days 45-60. These changes were characterized by hyperaemia on the previously unaffected skin areas or by the increase in clinical signs of injury at the foci of the primary lesions then in the process of healing. For example, late secondary erythema appeared in the area of the

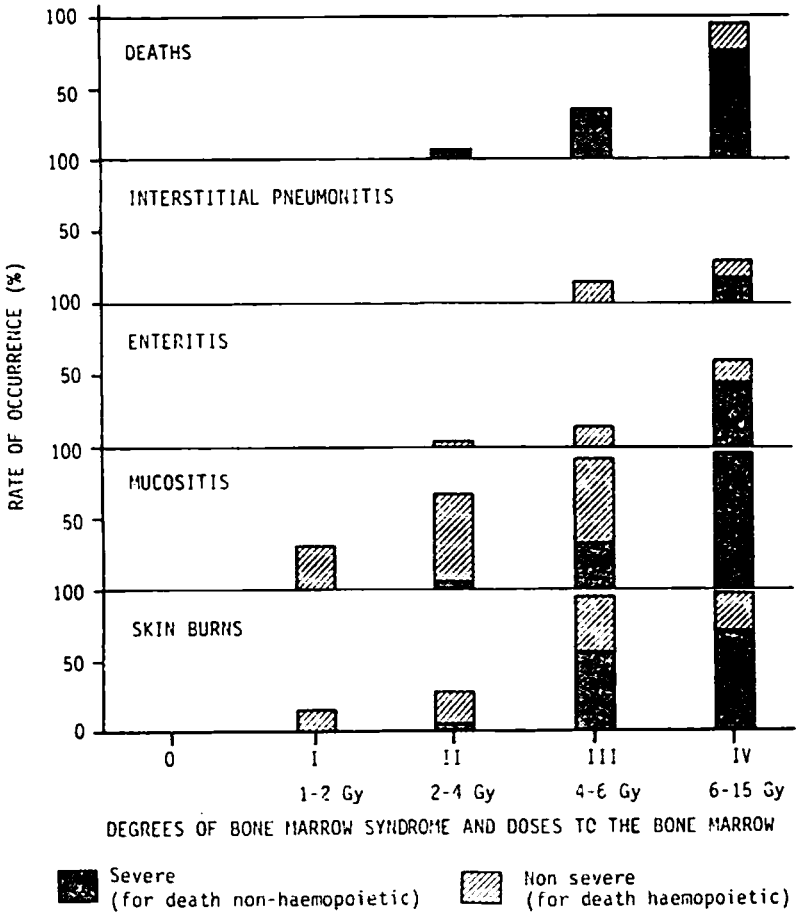

Figure A.VIII. Frequency of deaths and non-bone marrow syndromes for various degrees of bone marrow syndrome in accident victims suffering from acute radiation sickness.

ankles and feet, or on the hips and buttocks, of those patients who during the first three weeks displayed "flowering" burns on their knees. By the time of appearance of this late erythema, the lesions that had occurred earlier in many instances had already been repaired. As a rule, late erythema was accompanied by oedema of the subcutaneous tissues, which was particularly noticeable when located on the knees: pain was experienced in walking; palpation of the skin and underlying tissues (muscles, tendons) caused discomfort. The most severe cases involved fever and a general worsening of the patient's condition. Late secondary erythema was successfully resolved within two weeks by purely topical treatment, although in the more severe cases it was necessary to resort to additional therapeutic means, such as the prescription of glucocorticoids, a form of treatment that fairly rapidly eliminated all manifestations of epidermatitis and subcutaneous oedema, both general and local.

55. As may be seen in Table A.6, the burns suffered by the patients with acute radiation sickness covered from 1% to 100% of the body surface. It may be noted, in this connection, that if there were relatively early (from day 5-6) second- or third-degree burns over an area of even 30-40% of the body, followed by the spread of hyperaemia, these burns were life-threatening. In 19 of the 56 patients suffering from burns, the burns proved fatal (Figure A.VIII). It was found that patients with early secondary erythema over a body area of more than 40% first developed a febrile-toxemic syndrome, followed by renal-hepatic insufficiency and encephalopathic coma with cerebral oedema, resulting in death at 14-48 days after irradiation. A causal link connecting the fatal renal-hepatic insufficiency and the encephalopathic coma to the skin injuries is confirmed by the fact that a similar development of such fatal syndromes was observed in several patients who had neither severe bone marrow syndromes nor intestinal syndromes. However, in the majority of cases, burns were combined with an extremely severe bone marrow syndrome and severe acute enteritis, and in some cases the burns may have been the primary cause of death.

1. Intestinal syndrome

56. The intestinal syndrome was one of the more threatening manifestations of acute radiation sickness. In 10 patients, diarrhoea was observed from day 4 to day 8. This suggested that these persons had received total gamma doses of about 10 Gy or above; all these patients died during the first three weeks following irradiation. The occurrence of diarrhoea after eight days in seven other persons was an indication that they had received lower doses. The presence of radiation-induced enteritis lasting from day 10 to days 18-25 in spite of intensive water-electrolyte-protein supportive treatment suggests that the intestinal syndrome was not the main cause of death.

2. Oropharyngeal reactions

57. Acute radiation-induced inflammation of the oral and pharyngeal mucosa was observed in 82 patients. Its more benign manifestations (first and second degree of severity) were characterized by desquamation and oedema of the mucosa in the area of the cheeks and tongue and by tenderness of the gums. These were observed in 42 persons (dose range 1.7-4.0 Gy) from days 8-9 to days 20-25. The basic signs of a more acute oropharyngeal reaction were observed in 40 patients with third and fourth degree acute radiation sickness (dose range 4.5-16.0 Gy), and these were erosions and ulcers of the oral mucosa, sharp pain, and a large production of rubber-like mucus occasionally blocking the throat and causing breathing problems. The first signs appeared as early as days 3-4, attained their maximum intensity by day 10 and then subsided after days 18-20, when there was also granulocytopenia. The process involved no selective localization, as is characteristic of the ulcerated lesions in the area of the tonsils and gums when there are infectious complications. However, in a significant number of cases, the radiation-induced inflammation of the mucous membranes was complicated by secondary microbial and viral infection, which prolonged its course.

58. Another typical finding was the early (days 3-4) appearance of herpes-like rashes forming massive crusts on the lips and facial skin; this was observed in nearly 30% of the patients with severe bone marrow syndrome. Within this group of patients, primarily those suffering from fourth-degree acute radiation sickness, a pronounced radiation-induced parotitis was observed, coupled with an inability to salivate and a high level of amylase in the blood from days 1-4. The swelling of the parotid glands disappeared without special treatment, whereas recovery of salivary gland secretion was slower.

3. Lung reactions

59. Lung reactions were observed in seven patients suffering from third- and fourth-degree acute radiation sickness. Its characteristic signs were a rapidly intensifying dyspnoea together with respiratory insufficiency progressing over a period of two to three days culminating in death. Autopsies revealed large, blue lungs with pronounced interstitial oedema, without destruction of the mucous membranes of the trachea and bronchi. As a rule, interstitial pneumonitis developed several days before death, generally in combination with extremely severe lesions of the skin and the intestine. The times to death were 14-30 days after irradiation.

4. Causes of death

60. The frequency of non-haemopoietic injury increased as a function of total dose (Figure A.VIII). Clinical observations indicated the essential role of skin injuries in pathological processes prior to death. Among the patients that died, in two thirds of them there was extensive and severe radiation and thermal skin burns, which were considered life-threatening. In five cases skin injuries were the sole cause of death, because there was neither radiation enteritis or irreversible myelodepression. Deaths were observed between 10 and 96 days after exposure. The clinical picture in all fatal cases was characterized as following a difficult course, because in every case two or three radiation syndromes had occurred with complex toxicity, infection and circulation disorders. A summary listing of patient identification and causes of death is given in Table A.7.

61. A detailed clinico-morphological analysis made it possible to identify the predominance, within specified time periods, of particular lethal syndromes. Up to day 24, a total of 19 patients (65%) died. In one half of these patients the competing causes of death were skin and intestinal reactions (cases 3, 4, 10, 14, 15, 17, 20, 23, 26, 2097; in all the cases the gamma-radiation dose in the bone marrow was estimated to be greater than 10 Gy). Four patients showed acute radiation injury in the lung (cases 2, 9, 12 and 27; the doses were, respectively, 9.2, 9.7, 9.3 and 8.3 Gy), and, of these, two (cases 2 and 9) suffered also severe injuries to the skin. Two patients died of combined thermo-radiation burns (cases 24 and 25; the gamma-radiation doses in the bone marrow were estimated to be 3.7 and 5.7 Gy, respectively, in combination with internal irradiation doses. Within this time-frame, one patient (case 62, dose about 6 Gy) died almost exclusively from severe radiation burns at a time when haemopoiesis had begun to be restored. Three cases (cases 17, 26, 62) had involvement of mycobacterial sepsis. One patient (case 30, dose about 5.5 Gy) died of bleeding caused by mechanical injury to the subclavicular vein during catheterization, and another (case 7, dose about 4.7 Gy), suffering from severe radiation injury to the skin, died of post-transfusion shock. Characteristics of the deaths on days 11-24 were marked circulatory problems during the terminal period. This was shown by the relatively high frequency of signs indicating cerebral oedema and focal haemorrhaging into the brain and spinal cord.

62. Six patients died during the period from days 25-48. All six cases were characterized by extremely severe complications of a toxic or infectious nature. Two patients (cases 31 and 34) involved subtotal skin injuries (bone marrow doses about 6.7 and 5.8 Gy, respectively, with death occurring on days 32 and 48, respectively), coupled with practically restored haemopoiesis. The immediate cause of death in these cases was severe respiratory insufficiency and cerebral oedema. In one patient (case 28, dose about 6.4 Gy; death on day 48) the cause of death was severe, graft-versus-host disease and fungal and viral infections. One additional patient (case 5, dose about 4.4 Gy) died on day 34 of severe pulmonary and renal insufficiency, caused, most likely, by the transplantation of HLA-non-identical bone marrow and by post-transplant immunosuppression using cyclosporin and methotrexate. Two patients (cases 1 and 8, doses about 6.6 and 8.3 Gy) died on days 25 and 30, respectively, with symptoms of severe toxicity and pulmonary insufficiency. In nearly all six cases there were marked circulatory disorders in the lungs, intestines, brain and myocardium.

63. At a relatively late stage, days 86-96, three patients died. One patient (case 6, dose about 7.5 Gy) died on day 86 of graft-versus-host disease complicated by cytomegalovirus (CMV) infection. Cytomegalovirus infection was also the cause of death for another patient (case 16, dose about 10.1 Gy) on day 91. A female patient (case 33, dose about 4.1 Gy) died on day 96 displaying marked disruptions of cerebral blood circulation against a background of renal-hepatic insufficiency and foci of mycococcal infection (pneumonia). This patient suffered also skin injuries from beta-radiation which extended over one third of her skin surface and underwent a severe recurrent wave of erythema with oedema of the subcutaneous tissue.

5. Eye damage

64. Eye injuries were characterized by the early and subsequent involvement of all eye tissues in the pathological process (Table A.8). In this group of patients, damage to the skin and eyelid conjunctiva was caused, to a considerable degree, by beta-radiation.

65. At doses not exceeding 1 Gy there were no visible alterations in the structure of the eyes. In the case of patients suffering from first-degree acute radiation sickness, changes were noted only in the front segment of the eye: there was in individual cases a slight erythema in eyelid skin during the first two to four days and an intensification of the vascular pattern in the lid and conjunctiva of the eyeball. In 40% and 100% of the patients suffering from second- and third-degree acute radiation sickness, respectively, the eyelid skin showed a first wave of of erythema within 6-12 hours of irradiation, and within 2-3 weeks there was a second wave. These cutaneous alterations disappeared without trace, leaving hyperpigmentation and scaling. In all patients suffering from fourth-degree acute radiation sickness, the times to the appearance of the first and second wave of erythema were 1-2 hours and 8-10 days, respectively.

66. Microscopy of the bulbar conjunctiva revealed a number of alterations in the microcirculation: there was a dilation of the venules and capillaries (more rarely the arterioles), and an increase in the number of functioning vessels coupled with a reduced blood flow.

67. Two patients suffering from combined radiation and thermal second-degree lesions on the lid skin and conjunctiva experienced ulcerations on the skin around the eye that did not re-epithelialize for a long time. Epilation of the eyebrows was noted at days 15-17 in 16% of the persons with second-degree acute radiation sickness, and in 67% and 100% of those with third- and fourth-degree acute radiation sickness, respectively. The epilation was partial and transient. Hair growth on the head was fully restored. All patients retained their eyelashes.

68. Corneal damage was manifested in an early reduction in corneal sensitivity coinciding with the first wave of erythema, although first-degree patients did not show such an effect. At later times (days 35-55), superficial radiation-induced keratitis was observed in patients suffering from second-, third- and fourth-degree radiation sickness in 5%, 52% and 100% of the cases, respectively. Also noted were focal defects on the superficial epithelium of the cornea; these defects, which often merged, stained with fluorescein. The radiation keratitis regressed over a period of 1-1.5 months, leaving no opacification of the cornea.

69. Signs of disturbances in the haemodynamics of the retina were related to the dose and the degree of severity of radiation sickness. From a few days after irradiation, a reduction was observed in the level of diastolic pressure in the central retinal artery, followed later by signs of hypotonic angiopathy of the retina. Coinciding in time with the peak of the sickness, other injuries appeared, e.g., retinal oedema along the vessels and increased permeability of the retinal vessels (plasma discharge and haemorrhaging). The low diastolic pressure in the central retinal artery persisted over the entire acute phase.

70. In one severely ill patient (case 29, dose about 8.7 Gy) with fourth-degree acute radiation sickness, who survived the acute phase, the symptoms of angioretinopathy with haemorrhaging and plasma discharge recurred within 4.5 months, accompanied by a persistently low diastolic pressure in the central retinal artery (up to 5-10 mm Hg).

71. In the acute period, the treatment consisted in the topical application of ointments to the scaling surface of the eyelid skin and the instillation of 20% albucid, sophradex and vitamin solutions as eyedrops into the conjunctival cavity.

72. Within observation periods of up to one year, no obvious radiation-induced alterations of the lens were noted.

6. Treatment of radiation burns and other injuries

73. The treatment of radiation burns and other non-bone-marrow syndromes and their complications posed complex and multifaceted problems [J18]. From day 2 through day 8, 15 haemosorption sessions (purification using activated charcoal) were conducted for 13 patients suffering from the most severe skin lesions. Three patients who had been exposed to a total dose range of 2.0-4.6 Gy survived; they underwent haemosorption on a single occasion at days 5-8, i.e., considerably later than the time at which this might have affected the treatment of the bone marrow syndrome. This method of treatment did not change the outcome of the illness by modifying the haemocytopenia.

74. During the haemosorption process, and particularly towards the end of the session, many patients experienced a short-term improvement (lasting from a few hours to a single day), a reduction or disappearance of the pain in the extremities, and also a decrease of the oedema in their tissues. In this connection, contributory effects from the medication accompanying the procedure cannot be totally excluded.

75. A more widely used technique to combat the development of renal-hepatic insufficiency and fatal encephalopathic coma was plasmapheresis. Lesions induced by beta-irradiation over 30-40% and more of the body surface served as an indication for the application of this procedure. Plasmapheresis sessions were conducted for 17 patients from days 18-37. For a number of patients, daily sessions were conducted, up to six times.

76. The positive effect of repeated plasmapheresis was shown by a reduction of bilirubinemia and transaminasemia and a lowering of the nitrate level in patients suffering from renal-hepatic insufficiency caused by skin burns. On occasion, the plasmapheresis sessions were accompanied by reactions of minor severity such as chills and fever; there were no fatal complications. Another method used to treat toxicosis due to skin injuries was the injection of 1,000 ml of freshly-frozen plasma, accompanied by round-the-clock administration of heparin (1,000 active units/hour) with a liquid load (2-6 litres/day) and forced diuresis adequate to the intake volume. A pre-condition for this treatment was the presumption of disseminated intravascular clotting (DIC) syndrome (no typical anomalies in respect of coagulation were present) as a possible cause of encephalopathy and renal-hepatic syndrome. In its most strictly applied form, the heparin treatment method was used with two patients over a period of 7-15 days. The impression was that these patients survived longer than did patients whose condition was similar in terms of severity and extent of their burns. Their renal-hepatic insufficiency was less pronounced; however, a death due to encephalopathic coma was not averted.

77. The topical treatment of the burns required the involvement of a group of surgeons and nurses. A broad range of preparations and agents having an anti-inflammatory, bacteriostatic and regeneration-stimulating effect was used. Good results were achieved with lioxanol aerosol, an anti-burn ointment based on hydrocortisone with locally acting antibiotics, as well as BALIZ-2 solution and collagenous coatings. In each individual case the treatment varied in accordance with the stage of the lesions. Experience gained in the use of bactericidal fabric, both as a dressing material and for supplementary bedding, for patients with extensive burns deserves a particularly favourable comment in this connection [Z2].

78. Treatment of pain, as is typical of radiation injuries, was rather ineffective. At present, there are clearly no suitably effective local anaesthetics.

79. In patients suffering from severe radiation-induced inflammation of the oral mucosa, and enteritis, total parenteral nutrition had a positive effect; this was based on alvesin hydrolysate or an aminoacid mixture, aminone and a 40% glucose solution as the energy material. The treatment was carried out according to the principles and rules described by Dudrick et al. [D18]. This method was tested over a number of years with good results in patients receiving whole-body therapeutic gamma-irradiation at a dose level of 10 Gy for allogeneic bone marrow transplantation. The danger, which has possibly not been fully evaluated, is the probability that certain severely injured, comatose patients may enter a state of hyperosmolarity. Data on plasma osmolarity that would appear to be necessary in a programme of total parenteral nutrition were not provided for all patients.

80. For the majority of patients suffering from first- and second-degree bone marrow syndrome, the period of clinical convalescence was completed by the third or fourth month. A longer period of treatment was required by persons suffering from severe radiation burns and the sequelae of third- and fourth-degree bone marrow syndrome. At the present time, the bulk of the patients have resumed work with the exclusion of any contact with radiation sources.

81. Over the period from the fourth month to one year after the accident, the specialized treatment centre was periodically visited by patients with skin lesions (dystrophic and ulcerated areas and also oedema of the subcutaneous tissues, mainly on the knees and feet). These patients are being treated with agents designed to improve local blood circulation and tissue trophism. Five patients with deep and extensive ulcers on their arms and other areas of the body underwent repeated plastic surgery, and a number of them will require more extended treatment.

82. Immunological examination data, acquired 0.5-1.5 years after the accident, have shown that in the peripheral blood of the patient groups with a history of acute radiation sickness of the second, third and fourth degrees a decline was observed in the number of T-lymphocytes with helper activity along with an increase in the number of T-lymphocytes with suppressor activity. This led to a considerable reduction in the normal ratio between these immunoregulatory lymphocyte sub-populations. At the same time, there was no reduction in the general lymphocyte level or in their T- and B-sub-populations. As an average for the groups, the level of class A, M and G immunoglobulins in the patients' blood serum corresponded to the physiological norm. Similar changes were not observed in the case of patients with a history of acute radiation sickness of the first degree. During this time they experienced no severe or life-threatening infections. In a number of cases an effort was made at immuno-corrective therapy using T- and B-activin.

83. Within these same patient groups, an estimate of the number of respiratory illnesses over the same period of time was conducted retrospectively. It was found that the incidence of illness in the group of 19 patients with a history of first-degree acute radiation sickness did not differ from the incidence of illness for the group of persons for whom no acute radiation sickness diagnosis had been established, and that it averaged 0.3 cases per person per year. During the same period, this indicator approached 1 for 22 patients who had experienced second-degree acute radiation sickness, and 3 for 8 persons with a history of third- to fourth-degree acute radiation sickness.

84. This comparison underlines the importance of the immune system in maintaining anti-infection

resistance in radiation convalescents and raises the question as to the usefulness of conducting supportive immunomodulating therapy courses, long after the incident, for persons who have undergone severe forms of radiation sickness.

85. The experience of the specialized treatment centres in Moscow and Kiev in the organization of medical care of persons exposed in this nuclear reactor accident has been described [N16]. For the survivors, a plan of scheduled follow-up observation is in effect, and decisions as how best to arrange their living and working conditions are being taken.

D. CONCLUSIONS

86. The analytical data presented in this Appendix and derived from clinical observations of the victims of the accident at the Chernobyl nuclear power plant are in agreement with the data in Annex G.

87. However, the fact that such a large group of 115 patients, who had all received uniform whole-body irradiation, was treated simultaneously for acute radiation sickness of varying degrees of severity, represents a unique event that makes it possible to clarify numerous aspects of early effects in man. A complicating factor was the presence of severe and extensive beta-radiation skin injuries in 58 patients which aggravated the course of the sickness in 19 of the 28 who died. Two more patients died during the first days as a result of severe combined injuries (trauma plus thermal burns plus irradiation).

88. The analysis provides a basis for describing the principal clinical syndrome, the bone marrow syndrome, with various degrees of severity in all 115 patients. In the case of some of them the bone marrow syndrome was combined with intestinal and oropharyngeal injuries and radiation damage to the skin, the foreward segment of the eye (keratitis), and the lungs.

89. The treatment provided was in accordance with international practice and proved highly effective for the patient group exposed to doses of 2-4 Gy and for two thirds of the patients who received doses of 4-6 Gy. In the group of patients receiving 6-16 Gy, two patients who received doses of 8-9 Gy survived past 60 days.

90. The average bone marrow dose and the prognosis regarding the further course of the illness were determined on the basis of biological criteria. During the early period, most information was obtained from the karyological analyses, the lymphocyte counts and the primary reaction periods; later, from the granulocyte counts. The remaining indications were of an auxiliary nature. In three cases, the dose value coincided with the electron spin resonance study of dental enamel after death.

91. There is a need for further analysis of the time course of the early effects for a more accurate understanding of the nature of lung and neurological injuries, and for more detailed data on the relevance of biological dose indicators and the reasons for disparities between them. It is hoped that these data will be of use in the preparedness to respond in the event of an accident of a similar type in the provision of medical treatment.

Table A.1

Thyroid doses received by exposed persons

Range of thyroid doses (Sv)	Number of persons
0 - 1.2	173
1.2- 3.7	18
3.7- 6.1	4
6.1- 8.6	4
8.6-11.0	2
11.0-13.4	2
13.4-15.9	0
15.9-18.3	2
18.3-20.8	0
20.8-23.2	1

Table A.2

Doses of victims receiving higher internal exposures

Case number	Thyroid dose a/ (Gy)	Lung dose a/ (Gy)	Whole-body dose (Sv)	
			Internal	External
24	30	2.5	2.0	1.7
25	6	2.0	1.0	4.7
17	1	0.4	0.2	10.0
3	0.3	0.3	0.2	12.0
4	1.2	0.4	0.1	11.0
26	0.5	0.3	0.1	12.0

a/ Doses accumulated until time of death.

Table A.3

Distribution of patients with acute radiation sickness treated at the specialized treatment centre

Degree of severity	Number of patients	Bone marrow dose range (Gy)	Number of deaths	Time to death (days)
I	31	0.8-2.1	-	-
II	43	2.2-4.1	1	96
III	21	4.2-6.4	7	16,18,21,23,34,48,48
IV	20	6.1- 16	20	10,14,14,15,15,17,17, 18,18,18,20,21,23,24, 24,25,30,32,86,91
	115		28 a/	

a/ In addition to the patients who died of acute radiation sickness, one person died at the plant site and another within the first 12 hours following the accident, as a result of thermal burns, at the in-patient clinic in Pripyat where he had been given first aid.

Table A.4

Assessment of irreversible myelodepression according to diagnostic scores in cases of acute radiation sickness

Sign			Diagnostic score a/
Time to the onset of vomiting	(hours)	0.00- 0.4	+ 8
		0.41- 0.8	+ 4
		0.81- 1.2	+ 2
		1.21- 1.6	- 2
		1.61- 2.0	- 6
		> 2.01	-10
Lymphocyte count on the second day	(10^9/l)	0.00- 0.2	+ 6
		0.21- 0.4	+ 2
		0.41- 0.6	- 2
		0.61- 0.8	- 8
		> 0.81	-15
Lymphocyte count on the third day	(10^9/l)	0.00- 0.1	+ 8
		0.11- 0.2	+ 2
		0.21- 0.3	- 2
		0.31- 0.4	- 9
		> 0.41	-10
Lymphocyte count on the fourth day	(10^9/l)	0.00- 0.1	+ 4
		0.11- 0.2	+ 2
		0.21- 0.3	0
		0.31- 0.7	- 2
		0.71- 0.8	- 3
		0.81- 0.9	- 8
Lymphocyte count from day 4 to day 7	(10^9/l)	0.00- 0.1	+ 5
		0.11- 0.2	+ 2
		0.21- 0.3	- 1
		0.31- 0.4	- 5
		0.41- 0.5	-13
		> 0.51	-15
Average reticulocyte count from day 3 to day 5	(10^9/l)	0.0 - 8.0	+ 2
		0.1 -10.0	0
		10.1 -14.0	- 4
		14.1 -18.0	- 6
		18.1 -20.0	-10
Minimum neutrophil count for day 6 to day 7	(10^9/l)	0.00- 0.3	+12
		0.31- 0.6	+ 5
		0.61- 0.9	0
		0.91- 1.2	- 3
		1.21- 2.4	- 6
		2.41- 3.0	- 8

a/ The diagnostic signs are used to determine the diagnostic scores, which are then added together. A sum of +10 is the basis for a prognosis of irreversible myelodepression; a sum of -10 for a prognosis of no irreversible myelodepression. If after the diagnostic coefficients of all the available signs have been added no positive value has been reached, the answer is indeterminate (the available information is insufficient for a differential diagnosis, with an error probability of not more than ± 10%).

Table A.5

Survival or cause of death of patients receiving bone marrow transplantations and of patients in control group

Dose range (Gy)	Bone marrow transplant patients				Control patients		
	Number of patients	Deaths a/	Deaths b/	Number of survivors	Number of patients	Deaths a/	Number of survivors
< 6.5	4	0	3	1	5	0	5
6.5-9	3	2 c/	0	1	4	3	1
> 9	6	5	1	0	5	5	0
Total	13	7	4	2	14	8	6

a/ Skin and intestinal injuries.
b/ Bone marrow rejection (graft-versus-host disease) plus infection.
c/ Positive graft-versus-host disease post-mortem histology.

Table A.6

Distribution of cases of radiation burns of different degree in the presence of acute bone marrow syndrome

Degree of severity of bone marrow syndrome	Total number of patients	Number of patients with radiation burns to various percentages of the body surface		
		0-10%	10-50%	50-100%
I	31	2	1	0
II	43	2	9	1
III	21	3	15	3
IV	20	1	10	9
Total	115		56	

Table A.7

Patient identification, estimated dose, cause and day of death

Degree of severity of ARS	Case number	Bone marrow dose (Gy)	Treatment a/	Day of death b/	Cause of death
II	33	4.1		96	Infection, renal-hepatic insufficiency and skin injuries
III	5	4.4	BMT	34	Infection, post-transplantation immunosuppression
	7	4.7		18	Skin injuries, post-transfusion shock
	24	3.7		23	Thermal and radiation burns
	25	5.7		16	Thermal and radiation burns
	28	6.4	BMT	48	Infection, graft-versus-host disease
	30	5.5	BMT	21	Bleeding from mechanical injury during catheterization
	34	5.8		48	Respiratory insufficiency, cerebral oedema
IV	1	6.6	BMT	25	Toxicity, respiratory insufficiency
	2	9.2	BMT	15	Skin and lung injuries
	3	12	BMT	17	Skin and intestinal injuries
	4	11.8	BMT	18	Skin and intestinal injuries
	6	7.5	BMT	86	Infection, graft-versus-host disease
	8	8.3	LCT	30	Toxicity, respiratory insufficiency
	9	9.7		23	Skin and lung injuries
	10	11.1	LCT	14	Skin and intestinal injuries
	12	9.3		24	Lung injuries
	14	10.9	LCT	18	Skin and intestinal injuries
	15	>10	LCT	14	Skin and intestinal injuries
	16	10.1	BMT	91	Infection, graft-versus-host disease
	17	10	BMT	18	Skin and intestinal injuries
	20	12.4	LCT	17	Skin and intestinal injuries
	23	13.7	LCT	15	Skin and intestinal injuries
	26	12.5		20	Skin and intestinal injuries
	27	8.3	BMT	24	Lung injuries
	31	6.7		32	Respiratory insufficiency, cerebral oedema
	62	6.1		21	Radiation burns (skin injuries)
	2097 (Kiev)	10.2		10	Skin and intestinal injuries

a/ BMT = bone marrow transplantation; LCT = liver cell transplantation.

Table A.8

Type of eye changes and per cent incidence in the victims of the accident

Nature of the changes	Degree of acute radiation sickness			
	I	II	III	IV
First wave of erythema	6.1	39.5	100	100
Second wave of erythema		20.9	80.9	100
Reduction in cornea sensitivity		18.6	100	100
Epilation of the eyebrows		16.3	66.7	100
Keratitis		4.6	52.4	100
Fundus				
Dilation of blood vessels		32.6	74.4	100
Decreased diastolic pressure of the central retinal artery		48.8	95.2	100
Retinal oedema		4.6	-	80
Haemorrhaging		13.9	23.8	80
Plasmorrhaging		4.6	23.8	80

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back to first page