Creating and Presenting Dynamic Scientific Posters

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The objective of a science poster

- Showcase your science
- Demonstrate your abilities as a scientist
- Allow you to share information with the scientific community
- Develop your science communication skills
- Builds networks & contacts
- Help identify and establish collaborations
- Is a great source of feedback
- Help towards transitioning to the next step

Qualities of good posters

- Organized and flows logically
- Visually appealing & readable
- Succinct
- Presented clearly & with enthusiasm
- Provide everybody with something
- Have legs

Making your poster stand out

- Interesting title
- Abstract
- Attractive pictures and figures
- Clean and organized
- “You had me at hello.”

Considerations while creating your poster

- Who is the target audience
- Event guidelines
- Quality over quantity
- The numbers game
  - 10 seconds for person to decide to stay or go
  - 10 minutes max to go through your poster
  - 20 % text, 40% graphics, 40% white space
  - 4 ft to 6 ft – distance away from that poster is readable
  - 2 to 4 major results and/or conclusions
- The results are the starting point
- Create an interesting story

NIH Logo

Succinct descriptive title

Authors & affiliations

IC Logo

Introduction

Goals/Objective

Methods

Result 1

Result 2

Result 3

Result 4

Summary/ Conclusion

Other

- Introduction
  - Provides a starting point and a reason of why the research is important
  - Provide Abstract as supplementary material
- Goals/Objective
  - Clear statement of problem and your hypothesis
- Methods
  - Graphic conveyance of overall approach
- Results
  - 2 to 4 most relevant figures that support your conclusions
  - Best representative data with titles that state what the key finding is
  - Clearly labeled and readable
- Summary (if needed)
  - Quickly summarize major results for supporting your conclusion
- Conclusions
  - Succinct, bulleted information that corroberates your hypothesis
  - Provide an example of the human impact – BIG PICTURE
- Other
  - Indicate future direction if you have an incomplete story
  - References

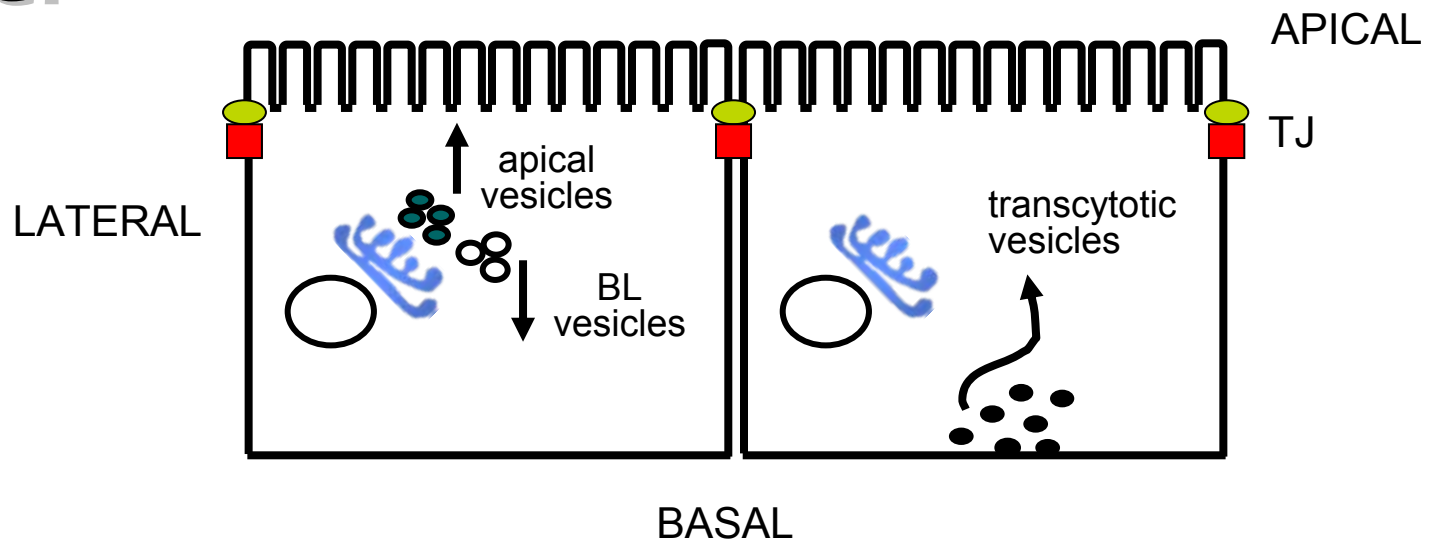
Section Examples

Before

INTRODUCTION

Epithelial cells are highly polarized with apical, basal and lateral membranes. Tight junctions form a barrier between the apical and basolateral surface. Some proteins are targeted directly to one plasma membrane surface, while some are targeted to the apical membrane following transcytosis from the basolateral surface. We still do not understand the molecular mechanisms that underlie the polarized sorting of proteins in epithelial cells.

After



- **Epithelial cells are polarized cells with apical, basal and lateral membranes. Tight junctions (TJ) form a barrier between the apical and basolateral surface.**
- **Some proteins are targeted directly to one plasma membrane surface, while others are targeted to the apical membrane following transcytosis from the basolateral (BL) surface.**
- **We still do not understand the molecular mechanisms that underlie the polarized sorting of proteins in epithelial cells.**

Materials and Methods

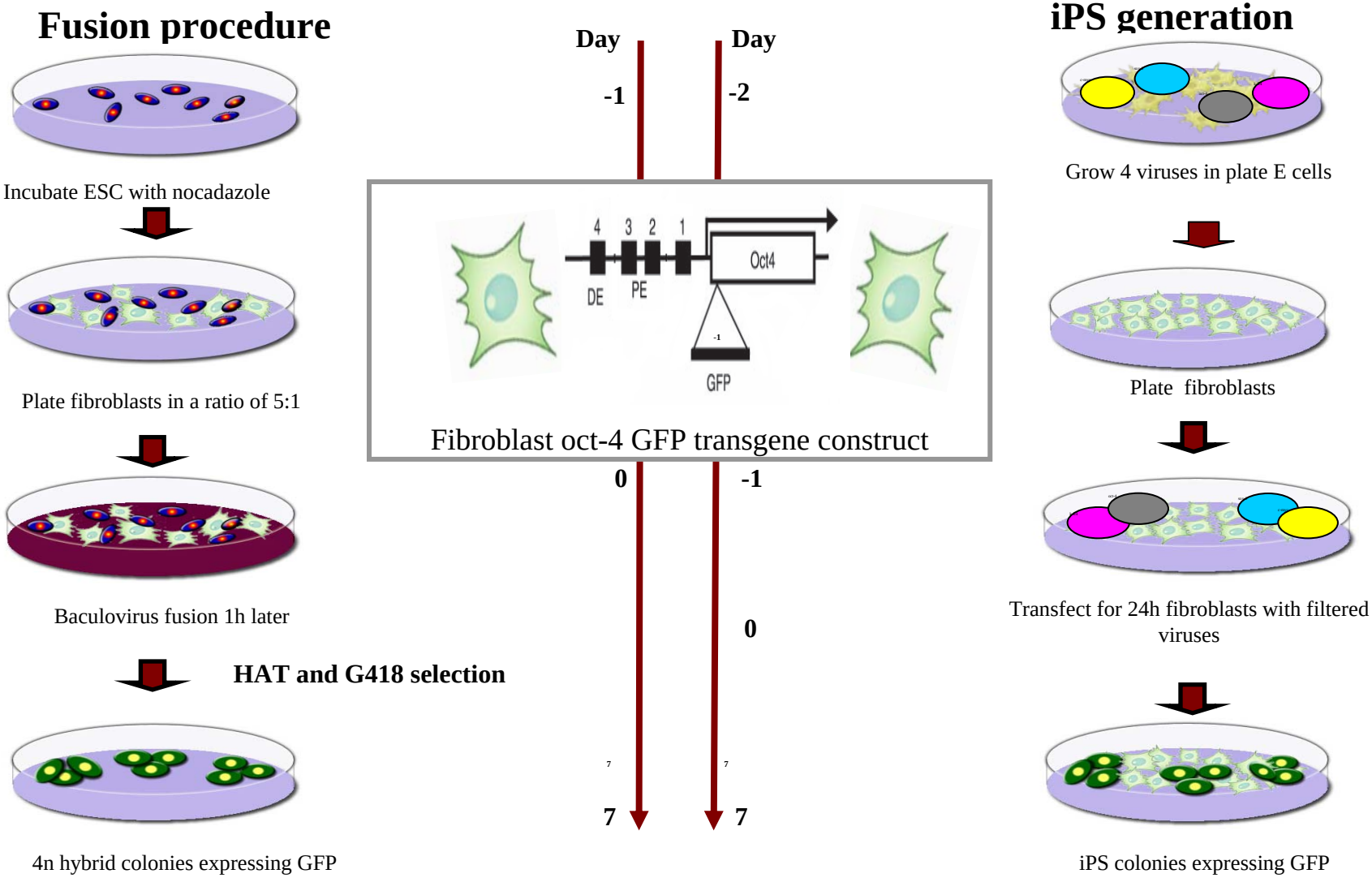
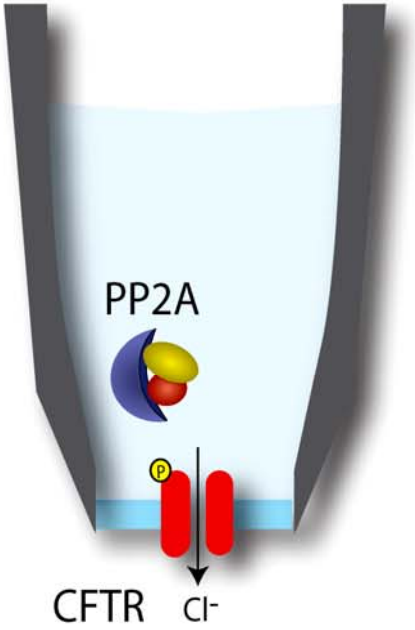
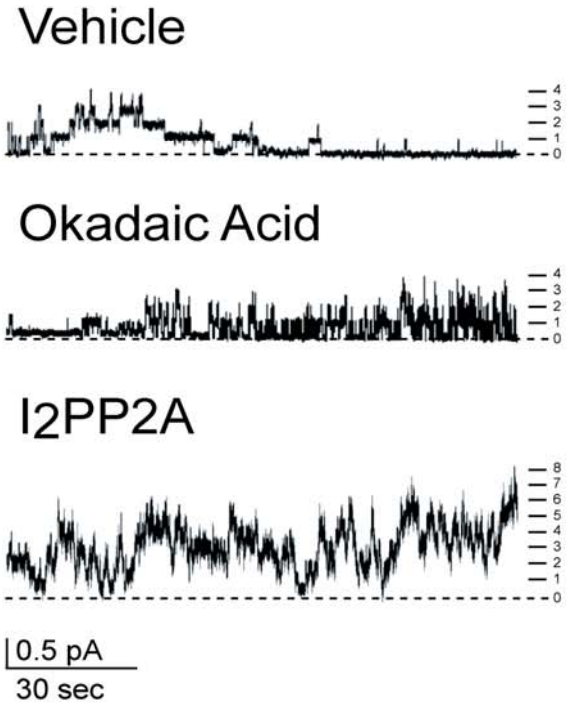


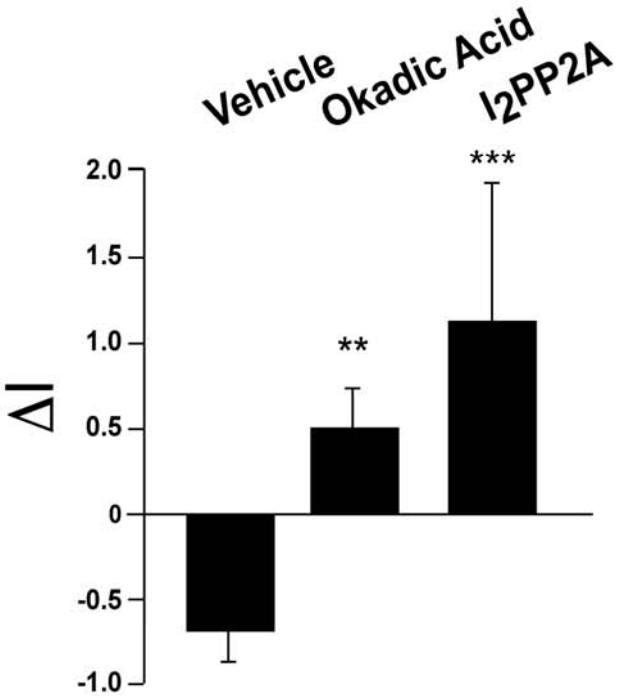
Figure 1. Flow chart of reprogramming by baculovirus mediate cell fusion (left panel) and by retroviral transfection of 4 genes (right panel).

PP2A regulates CFTR channel activity

A. Experimental design	B. Single channel recordings	C. Averaged data N=6

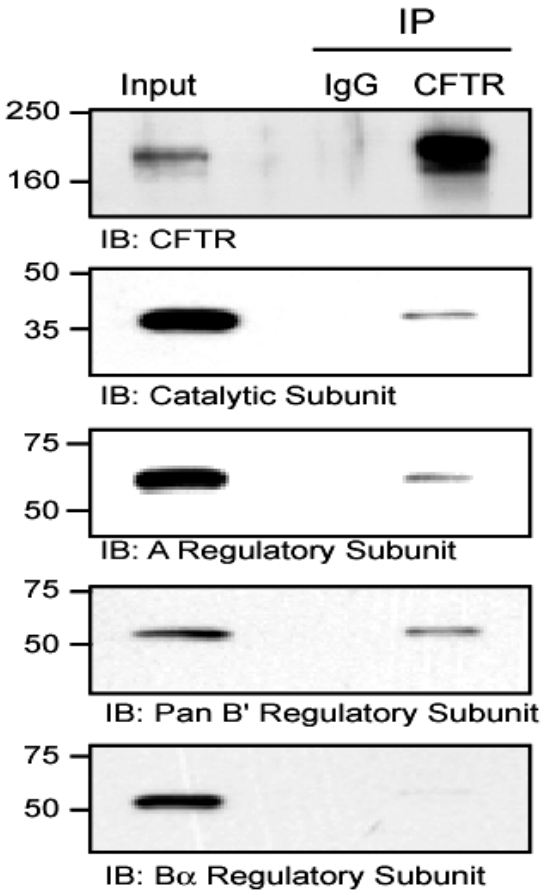




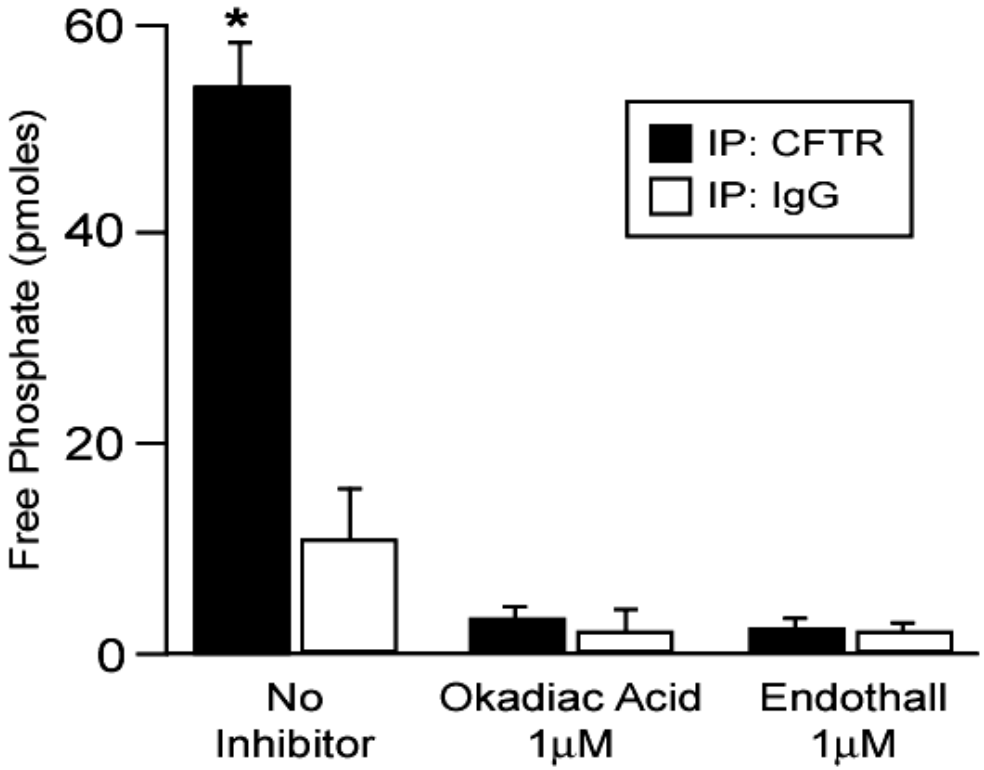


PP2A subunits and activity co-precipitate with CFTR in airway cells

A. Western blot analysis



B. Phosphatase activity assay



Conclusions (Before)

Conclusions

We used affinity purification to identify proteins that associate with CFTR and found that the the B'ε subunit of PP2A directly associates with the CFTR C-terminus. Using western blotting and in-vitro phosphorylation assays, we showed that PP2A protein and activity co-immunoprecipitate with CFTR from airway epithelial cells. The PP2A B'ε is the subunit responsible for targeting the phosphatase to the channel. We further found that PP2A negatively regulates CFTR channel activity in mouse intestinal and human airway epithelial cells. Thus we conclude that inhibitors of PP2A may improve clinical outcomes in cystic fibrosis.

Conclusions (After)

Conclusions

- The B'ε subunit of PP2A directly associates with the COOH-terminus of CFTR
- PP2A protein and activity co-immunoprecipitates with CFTR in cultured airway epithelial cells
- PP2A negatively regulates CFTR channel activity in mouse intestinal and human airway epithelial cells
- Inhibitors of PP2A may improve clinical outcomes in Cystic Fibrosis

Making the delivery work for you

- Radiate enthusiasm and confidence
- Maintain eye contact
- Find out what your audience knows
- Tell a great story
- Use tone and inflection to emphasize key points
- Practice! Practice! Practice!

Example Posters

Somatic journey to pluripotency and back to lineage commitment

Anna Davidhi[1], Marta Wegorzweska[2], Rafael Casellas[2], Lisa Boyette[3], Rocky Tuan[3], Eran Meshorer[4], Itai Tzchori[1], Heiner Westphal[1]

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Background

Somatic cell reprogramming reverts the epigenetic and subsequently the differentiation identity of a cell to a pluripotent embryonic stem cell-like state. Embryonic stem cells (ESC), obtained from the inner cell mass of the blastocyst, are pluripotent: they are unspecialized, possess long term renewal ability and can give rise to the whole embryo excluding the extraembryonic tissue. As such they are highly prized for patient specific tissue replacement. The birth of Dolly in 1997, by somatic cell nuclear transfer, showed that: cellular differentiation is a reversible process when germ line modifications are not involved. Thus, in the presence of the appropriate ***"reprogramming environment"*** the epigenetic memory of a cell is re-established to a pluripotent-like state. A somatic cell becomes pluripotent-like when fused with an ESC either by polyethylene glycol (PEG) or by electrofusion. In 2006, Yamanaka *et al*, showed that this "reprogramming environment" can also consist of four retrovirally encapsulated transcription factor genes, which when transfected into somatic cells give rise to ***induced pluripotent stem (iPS) cells.***

Dolly the sheep

All these three reprogramming methods employ major architectural changes in genome expression patterns including histone post-translational modifications. These biochemical alterations work combinatorially and cumulatively in defining the epigentic state of a cell and thereby its biological function.

Objective

We have employed two strategies to investigate interrelated factors influencing somatic cell reprogramming:

- ***Baculovirus mediated fusion*** of two ESC lines with mouse embryonic fibroblasts (MEFs) investigating:
  1. Is the reprogramming ability of different ESC lines, as measured by the overall number of tetraploid hybrids obtained, "the same"?
  2. Are chromatin remodeling markers involved in modulating this phenotype and if so how?
- ***Viral mediated transfection*** of MEFs addressing the questions:
  1. Is the iPS reprogramming ability any different from that of a standard ESC? If so, is this ability amenable to pharmacological manipulation?
  2. Can iPS *in vitro* differentiate well into the Mesenchymal Stem Cell (MSC) lineage and then into into mesodermal tissue?

Materials and Methods

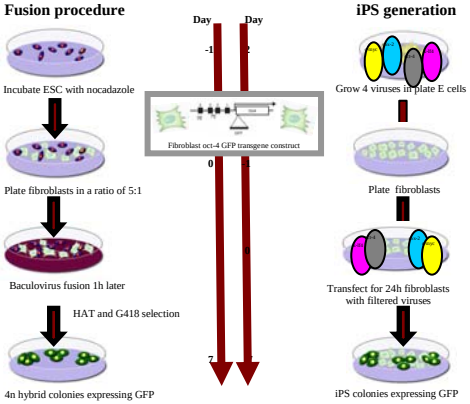

Figure 1. Flow chart of reprogramming by baculovirus mediate cell fusion (left panel) and by retroviral transfection of 4 genes (right panel).

The MEF/ESC hybrid possesses pluripotent-like properties

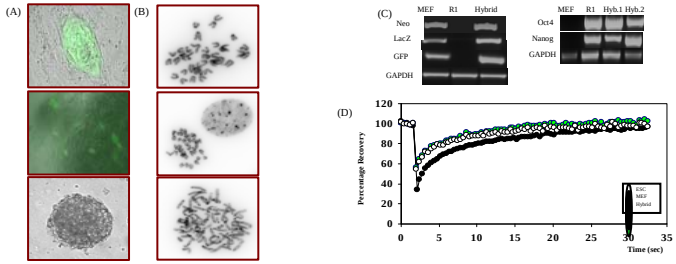

Figure 2. (A) From top to bottom, oct-4 GFP expressing hybrid colony, has the ability to self-renew, as well as form *in vitro* embryoid bodies. (B) From top to bottom, karyotype analysis of 2n ESC nucleus, 2n fibroblast nucleus, and 4n MEF/ESC hybrid nucleus. (C) Left panel: Genotype of MEF, R1 and hybrid for transgene markers. Right panel: Gene expression analysis by reverse transcription-polymerase chain reaction.: Lane 1: MEF; Lane2: R1 ESC; Lane 3 and 4 MEF/ESC hybrid1 and hybrid2 (D) Pluripotent-like properties of MEF/ESC hybrid chromatin. Fluorescence recovery after photobleaching of CFP labeled heterochromatin protein 1 (HP1) in wild type ESC (white circles), MEF (black circles) and MEF/ESC hybrid (green circle).

Increased H3K9 acetylation levels elevate stem cell potency

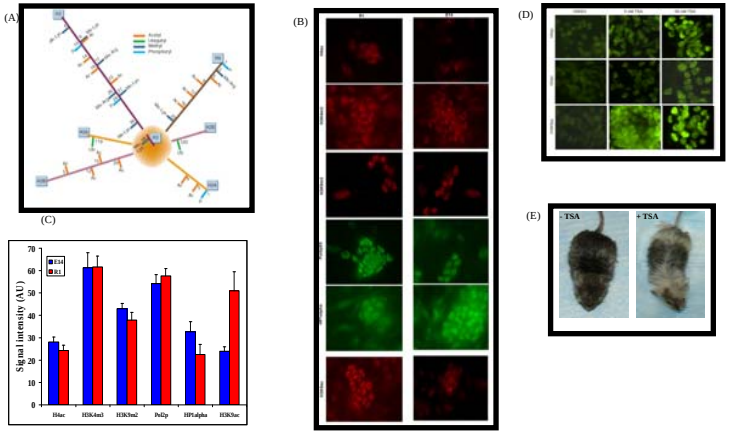

Figure 3. (A) Chromatin histone modifications *Adapted from from Felsenfeld and Groudine (2003).* (B) Immunofluorescent images of pan-acetylated H4 (H4ac), tri-methylated H3 on lysine 4 and 9 (H3K4me3, H3K9me3), RNA polymerase II phosphorylated on serine 5 (Pol2pS5), HP1alpha and H3 acetylated at lysine 9 (H3K9). (C) Quantification of B. The Y axes contains arbitrary fluororescent units. Values represent results from at least 20 cells from 3 independent experiments. (D) TSA treatment increases H3K9ac in the E14 stem cell line. E14 cells are treated with the vehicle (DMSO, left), 5nM (middle), and 25nM (right) of trichostatin A (TSA). Immunofluorescence of histone acetylation levels were done by using antibodies specific for pan-acetylated H4 (H4ac, top), pan-acetylated H3 (H3ac, middle) and H3 acetylated on lysine 9 (H3K9ac, bottom). (E) From left to right E14 chimera mice without TSA treatment and with 24h TSA treatment.

iPS derived from MEF's differentiate into mesodermal lineages

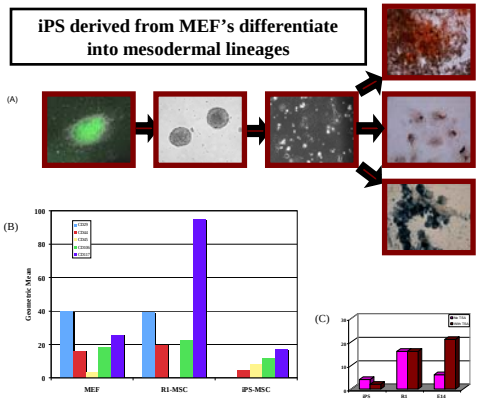

Figure 4. (A) From left to right: Fluorescence microscopy of an iPS GFP expressing colony, phase contrast microscopy of iPS-derived embryoid bodies, phase contrast microscopy of iPS-derived MSC's. From top to bottom: alizarin red staining of MSC derived osteocytes, oil red staining of MSC derived adipocytes and alkaline phosphatase staining of MSC derived osteocytes. (B) Comparison of cellular differentiation marker expression levels for different cell types as measured by flow cytometry. (C) Comparing the reprogramming abilities of iPS, R1 and E14 stem cell lines with and without TSA treatment. The Y axes represents the number of MEF/ESC hybrids obtained for 20 million ESC used.

Conclusions

- Reprogrammed hybrids exhibit pluripotent like characteristics such as morphology, long term renewal ability, embryoid body formation, gene expression profile and chromatin protein hyperdynamic plasticity.
- Different ESC lines display characteristic higher-order chromatin structure. While it is true that no one singular epigentic modification invariantly translates to one single biological output, we have shown that pharmacologically elevated levels of H3K9ac significantly increase the overall reprogramming ability of the E14 ESC line as measured by the most stringent reprogramming criterion: chimera contribution.
- When iPS are fused again with somatic cells from which they themselves originated, they reprogram them, although the efficiencies of this reprogramming merit further investigation.
- iPS differentiate into MSC's but flow cytometry analysis indicates that there are significant differences in the cellular differentiation marker levels as compared to standard *in vitro* MSC's.

Future Direction

- iPS are heterogeneous with respect to pluripotency. In attempts to "quantify" such stemness differences we will investigate iPS chromatin epigenetic remodeling.
- The *in vivo* aspect of our work, will focus on examining the functional potential of iPS derived differentiated cells.
- Present iPS generating methods are such that these "golden cells" are still disqualified for translational use due to their increased oncogenic potential. We are working on finding new strategies to efficiently generate clinically usable iPS.

References

1. Cowan CA, Atienza J, Melton DA, Eggan K. Nuclear reprogramming of somatic cells after fusion with human embryonic stem cells. Science 309, 1369-1373 (2005).
2. Hoshikawa Y, Kwon HJ, Yoshida M, Horinouchi S and Beppu T. Trichostatin A induces morphological changes and gelsolin expression by inhibiting histone deacetylase in human carcinoma cells. Experimental cell research 214, 189-197 (1994)
3. Meshorer E, Yellajoshula D, George E, Scambler PJ, Brown DT and Misteli T. Hyperdynamic plasticity of chromatin proteins in pluripotent embryonic stem cells. Development Cell 10, 105-116 (2006).
4. Takahashi, K. & Yamanaka, S. Induction of pluripotent stem cells from mouse embryonic and adult fibroblast cultures by defined factors. Cell 126, 663–676 (2006).

In Vivo Imaging of Brain Metastasis Development and the Effects of Anti-Angiogenic Treatment

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[1]Cell and Cancer Biology Branch, NCI; [2]Laboratory of Functional and Molecular Imaging, NINDS, NIH

Background

- Brain metastasis occurs in 25-40% of cancer patients and over 50% of brain tumors are metastatic.
- Current treatments are ineffective on brain metastases.
- Anti-angiogenic treatments are of interest due to the important role of angiogenesis in brain metastases.
- A relevant animal model is needed for the development of more effective treatments.

Purpose

To test the effects of anti-angiogenic treatment using non-invasive imaging in a mouse model of brain metastasis.

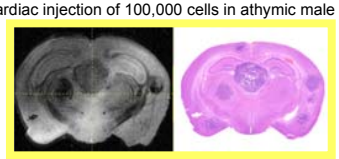
Methods

Cell Line: DU145

- Isolated from the brain metastasis of a prostate cancer patient
- Parent line is lowly metastatic in animal models.
- We have isolated a highly metastatic line from mice after the introduction of a Ras effector mutant.

Animal Model

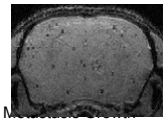
Intracardiac injection of 100,000 cells in athymic male nudes



Within 3-5 weeks, mice develop brain metastases which are detectable by MRI and confirmed by histology.

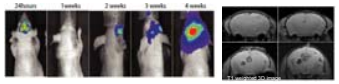
Imaging of Brain Metastasis Development

- Tumor Cell Dissemination



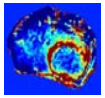
Cells are labelled with 1.63 μm iron oxide particles and extracted through a magnetic field.
Labelled cells are detected by 3D gradient echo images. Presence of cells is indicated by hypointensive spots

- Brain Metastasis Growth



Brain metastasis growth can be monitored through both bioluminescence imaging and MRI

- Angiogenesis in Brain Metastasis

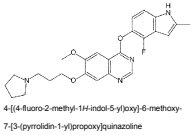


Angiogenesis was imaged through the use of USPIO contrast enhanced blood volume measurement
Mice are injected in the tail vein with 5nm iron oxide particles with a dextran coat (for a total size of 30nm). Images are acquired 5 minutes post injection. Data was then processed using software routines written in Matlab

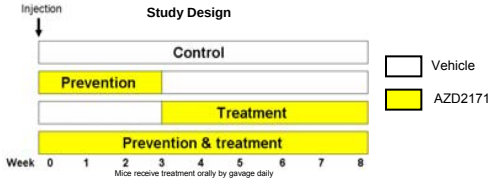
Methods (cont.)

Drug Treatment

- There is evidence that tumor produced VEGF contributes to angiogenesis and metastasis.
- We decided to use AZD2171, a potent inhibitor of vascular endothelial growth factor (VEGF) receptor tyrosin kinases.

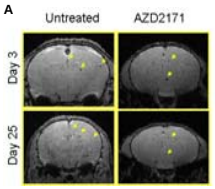


4-[(4-fluoro-2-methyl-1H-indol-5-yl)oxy]-6-methoxy-7-[3-(pyrrolidin-1-yl)propoxy]quinazoline



Results

Tumor Cell Dissemination



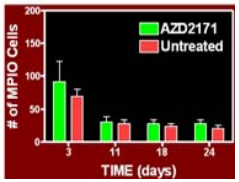
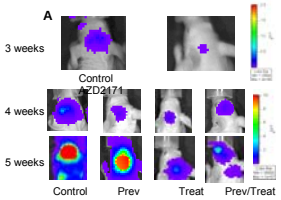
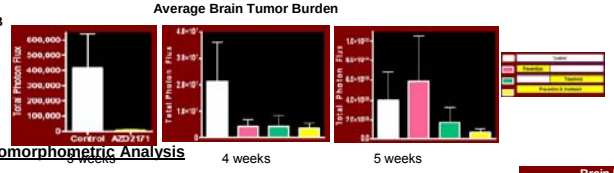


Figure 1. 3D MRI scans show that drug treatment has no effect on tumor cell dissemination. There are similar numbers of hypointensive spots found in the brains of both treated and untreated mice over time.
A) 3D echo images at day 3 and day 25 p.i.. B) Time-course of the number of spots per brain.

Brain Metastasis Growth

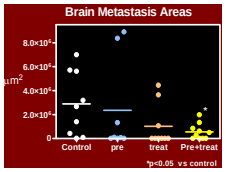
Figure 2. AZD2171 treatment reduced the growth of brain metastases as shown by bioluminescence imaging. The effects of treatment are quick, as shown by the comparable signal of the treatment group to the prevention/treatment group at week 4. The inhibitory effects of treatment don't last after withdrawal as shown by the high signal in the prevention group at week 5. A) Bioluminescence images 3, 4, and 5 weeks post injection. B) Average brain tumor burden per mouse (n=10 per group) at 3, 4, and 5 weeks p.i..





Histomorphometric Analysis

Figure 3. Histomorphometric analysis of endpoint mice shows that treated mice have fewer large tumors when compared to control. The prevention group has large but fewer tumors than control showing that while withdrawal of treatment did not prevent the growth of established metastases, early treatment prevented the establishment of metastases.



Results (cont.)

Survival

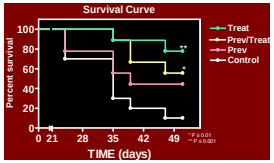


Figure 4. Treatment prolonged the survival of tumor bearing mice.

Angiogenesis

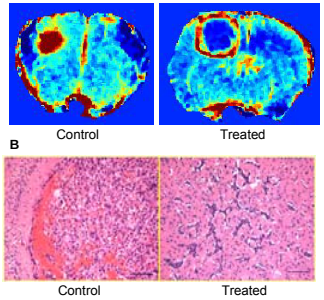


Figure 5. Brain metastases of treated mice have a lower level of angiogenesis when compared to the brain metastases of untreated mice.

A) Relative cerebral blood volume images. B) H&E staining which shows densely packed tumor cells with a high level of angiogenesis in control tumors, and sparse tumor cells with a lower level of angiogenesis in treated tumors.

Summary

- We have developed a relevant mouse model of brain metastasis.
- Non-invasive imaging can be used on our model to monitor the steps of brain metastases and therefore also the effects of anti-angiogenic treatment.
- AZD2171 treatment…
  - o Did not affect tumor cell dissemination.
  - o Inhibited the progression of established metastases.
  - o Inhibited the development of new metastases.
  - o Reduced angiogenesis in brain metastases.
  - o Prolonged the survival.
  - o Removal of treatment results in rapid relapse of brain metastases.

Conclusion

Anti-angiogenic therapy may be useful in treating not only patients with brain metastases, but also patients at risk of developing brain metastases.

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Targeting Human Disease with Virus Mimicry

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[2]Department of Microbiology and Immunology, School of Medicine and Biomedical Sciences, State University of New York (SUNY) at Buffalo, Buffalo, NY

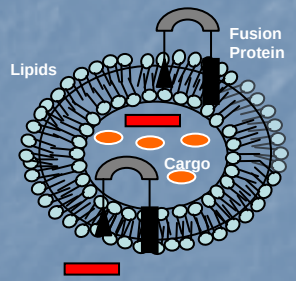
[3]SAIC-Frederick, Inc., NCI-Frederick, Frederick, MD

Abstract

Viruses hijack human cells using a variety of sophisticated mechanisms that range from fusion with the cell membrane to regulation of protein expression and genetic modification. These natural principles are excellent models from which we can design targeted therapies to treat human disease.

We are designing nanoparticles that are based upon virus entry mechanisms. One of our hypotheses is that the efficiency of nanoparticle payload delivery can be dramatically enhanced by the capacity for direct membrane fusion with the plasma membrane. We are utilizing viral membrane fusion proteins incorporated into liposomal nanoparticles to deliver payloads directly into the cytoplasm of targeted cells.

TARGETED FUSOGENIC PROTEOLIPOSOME



Targeting Moieties:

- scFv C10 (targets insulin-like growth factor receptor 1)
- CDCRGDCFC peptide (targets αVβ3 integrins)

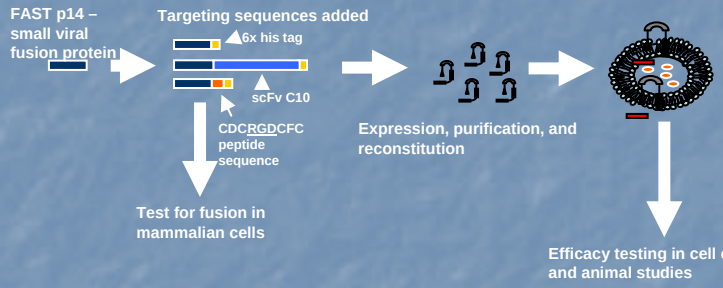
Introduction

The great promise of **nanoparticle delivery** is its ability to salvage drugs or other therapy modalities that have successfully made it far into preclinical or clinical trials, but that have failed near the end of the pipeline because of toxicity or deleterious immunological response.

Liposomes present a promising biomaterial-based method of therapeutic delivery, constituting more than 250 NIH clinical trials.[1] A primary issue that remains unresolved in liposomal delivery, and in nanoparticle delivery in general, is avoidance of the endocytic pathway, which often leads to uncontrolled release, sequestering, and/or degradation of cargo molecules in vesicles in the entry pathway.

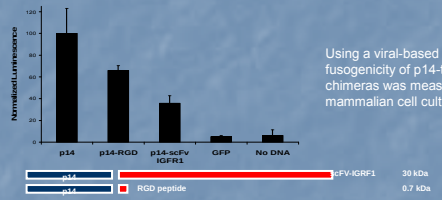
Our goal is to avoid the endocytic pathway by direct fusion with the plasma membrane. The fusogenic protein that we use is a fusion-associated small transmembrane (FAST) protein, p14, from a reptilian reovirus.[2] FAST p14 is promising in engineering fusogenic liposomes because it is much smaller, at 14 kD, and less complex than other fusogenic protein machinery, for instance, the HIV-entry machinery, which is a trimer of heterodimers at ~500 kD.

Methodology



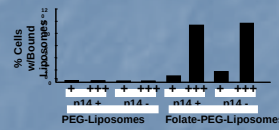
Results

Fig. 1: FAST p14 chimeras containing C-terminal targeting peptides retain fusogenic activity.



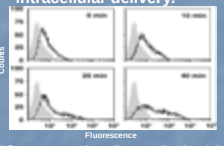
Using a viral-based assay, the fusogenicity of p14-targeting chimeras was measured in mammalian cell culture.

Fig. 2: FAST p14 liposomes can be targeted to specific cell receptors.



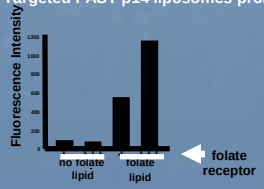
Treatment of cells to up-regulate the folate receptor resulted in a dramatic increase in liposome adherence to target cells.

Fig. 3: FAST p14 liposomes promote fusion and intracellular delivery.



The increased fluorescence in cells seen by the rightward shift in fluorescence, indicates that calcein entrapped in the liposomes, self-quenching at higher concentrations, has been released into the cytoplasm.

Fig. 4: Targeted FAST p14 liposomes promote increased intracellular delivery.



Cell fluorescence increase caused by folate-targeted liposomal delivery was quantified, correcting for the background fluorescence at the non-fusogenic temperature of 4°C.

Conclusions

- FAST p14 remains fusogenic with the addition of targeting moieties to the C-terminus of the protein.
- FAST p14 does not interfere with targeting of liposomes to cells using a folate lipid targeting the folate receptor.
- Targeted-FAST p14 liposomes show increased intracellular delivery.

Ongoing Work

- Test RGD and scFV-chimeras for targeted fusion with cells
- Encapsulate and deliver cytotoxic drugs
- Encapsulate and deliver pro-apoptotic peptides
- Deliver DNA/RNA
- Begin testing in small animal models

Future plan

Pursue detailed studies of virus mechanisms with an eye toward utilization of this knowledge to drive innovation in nanomedicine.

References

1. Information on Clinical Trials. *National Library of Medicine.* www.clincaltrials.gov.
2. Top, D, R de Antueno, J Salsman, J Corcoran, J Mader, D Hoskin, A Touhami, MH Jericho, R Duncan (2005). *EMBO J.* 24: 2980-2988.

Collaborators

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Jacek Capala, Radiation Oncology Branch, National Cancer Institute, Bethesda, MD

Dimiter Dimitrov, Center for Cancer Research Nanobiology Program, NCI-Frederick, Frederick, MD

Targeting Human Disease with Virus Mimicry

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[2]Department of Microbiology and Immunology, School of Medicine and Biomedical Sciences, State University of New York (SUNY) at Buffalo, Buffalo, NY

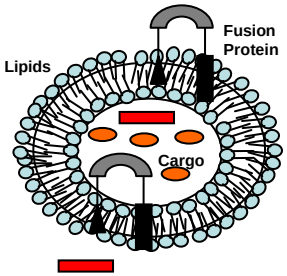
[3]SAIC-Frederick, Inc., NCI-Frederick, Frederick, MD

Abstract

Viruses hijack human cells using a variety of sophisticated mechanisms that range from fusion with the cell membrane to regulation of protein expression and genetic modification. These natural principles are excellent models from which we can design targeted therapies to treat human disease.

We are designing nanoparticles that are based upon virus entry mechanisms. One of our hypotheses is that the efficiency of nanoparticle payload delivery can be dramatically enhanced by the capacity for direct membrane fusion with the plasma membrane. We are utilizing viral membrane fusion proteins incorporated into liposomal nanoparticles to deliver payloads directly into the cytoplasm of targeted cells.

TARGETED FUSOGENIC PROTEOLIPOSOME



Targeting Moieties:

- scFv C10 (targets insulin-like growth factor receptor 1)
- CDCRGDCFC peptide (targets αVβ3 integrins)

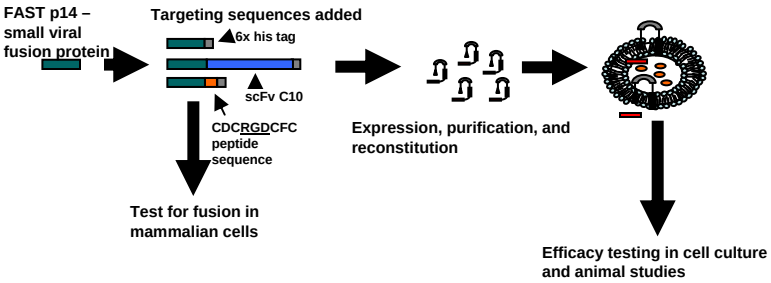
Introduction

The great promise of **nanoparticle delivery** is its ability to salvage drugs or other therapy modalities that have successfully made it far into preclinical or clinical trials, but that have failed near the end of the pipeline because of toxicity or deleterious immunological response.

Liposomes present a promising biomaterial-based method of therapeutic delivery, constituting more than 250 NIH clinical trials.[1] A primary issue that remains unresolved in liposomal delivery, and in nanoparticle delivery in general, is avoidance of the endocytic pathway, which often leads to uncontrolled release, sequestering, and/or degradation of cargo molecules in vesicles in the entry pathway.

Our goal is to avoid the endocytic pathway by direct fusion with the plasma membrane. The fusogenic protein that we use is a fusion-associated small transmembrane (FAST) protein, p14, from a reptilian reovirus.[2] FAST p14 is promising in engineering fusogenic liposomes because it is much smaller, at 14 kD, and less complex than other fusogenic protein machinery, for instance, the HIV-entry machinery, which is a trimer of heterodimers at ~500 kD.

Methodology



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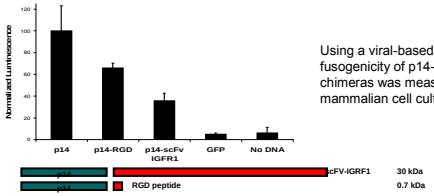

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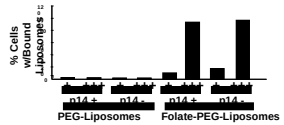

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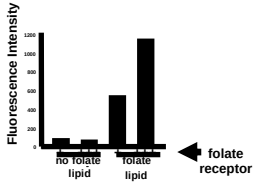

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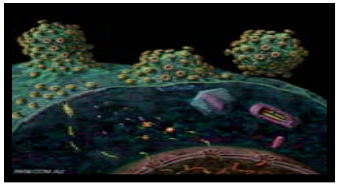

References

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Mistakes to avoid

- Re-inventing the wheel
- Poster is too “busy”, not enough white space
- Too much text, not enough graphics
- Copy and paste issues
- Not following the guidelines set by the organizers
- Not coordinating with the printer early enough
- Taking into account technology issues
- Proofing the material before sending to the printer
- Proofing the poster after printing
- Waiting until the last minute to put together poster
- Not practicing the delivery sufficiently enough

Contact Us

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Email: mullensh@mail.nih.gov