

Creating and Presenting Dynamic Posters



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Poster sessions are a lot like farmers markets



Poster sessions are a lot like farmers markets





A poster is more than presenting data

- Develops your verbal communication skills
- Allows you to share information with the scientific community
- Builds networks & contacts
- Helps identify and establish collaborations
- Is a great source of feedback
- May even help you find a job



Effective poster presentations are:

- organized
- visually appealing & readable
- succinct
- presented clearly & with enthusiasm

And are not...

Manuscripts printed out and tacked up on a board!!!

REMEMBER:

- The average person decides within ~10 seconds whether to stay or go onto the next poster
- Most people stay at a poster less than 10 minutes

Typical parts of a poster

- Title
- Authors and affiliations
- Introduction
- Methods
- Results
- Conclusions
- References
- Acknowledgements

Short descriptive poster title

Authors & affiliations

Introduction	Data 1	Data 4
Goals	Data 2	Conclusions
Methods	Data 3	Other stuff

- Present data from top to bottom, left to right
- Maintain empty space between rows

Somatic journey to pluripotency and back to lineage commitment

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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 Yamaoka *et al.* showed that this reprogramming can be achieved by introducing specific epigenetic changes in genome expression with *comparative and integrative genomics*. These biochemical epigenetic transcription factor genes, which when transcribed into somatic cells, are able to induce pluripotent stem (iPS) cells.

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 reprogramming be translated into the Mouse Embryonic Stem Cell (ESC) lineage and then into mesodermal tissue?



Dolly the sheep

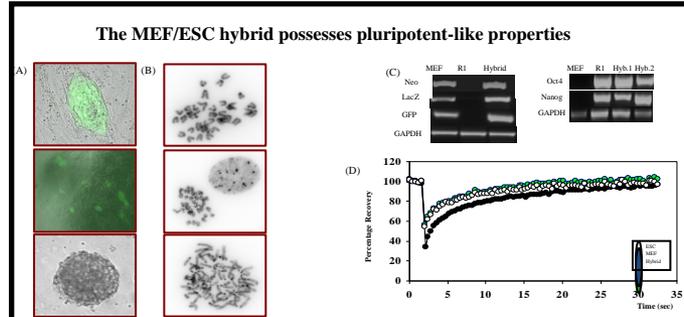


Figure 2. (A) From top to bottom, oct-4 GFP expressing hybrid colony, has the ability to self-renew, as well as form *in vitro* embryoid bodies. (B) From top to bottom, karyotype analysis of 2n ESC nucleus, 2n fibroblast nucleus, and 4n MEF/ESC hybrid nucleus. (C) Left panel: Genotype of MEF, R1 and hybrid for transgene markers. Right panel: Gene expression analysis by reverse transcription-polymerase chain reaction. Lane 1: MEF; Lane 2: R1 ESC; Lane 3 and 4 MEF/ESC hybrid 1 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).

Materials and Methods

Fusion procedure

iPS generation

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

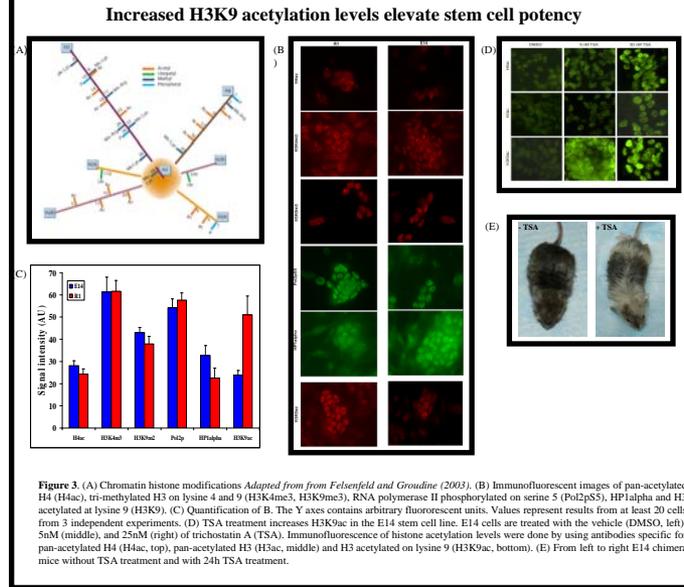


Figure 3. (A) Chromatin histone modifications Adapted from Felsenfeld and Groudine (2003). (B) Immunofluorescent images of pan-acetylated H4 (H4ac), tri-methylated H3 on lysine 4 and 9 (H3K4me3, H3K9me3), RNA polymerase II phosphorylated on serine 5 (Pol2pS5), HP1alpha and H3 acetylated at lysine 9 (H3K9). (C) Quantification of B. The Y axes contains arbitrary fluorescent 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.

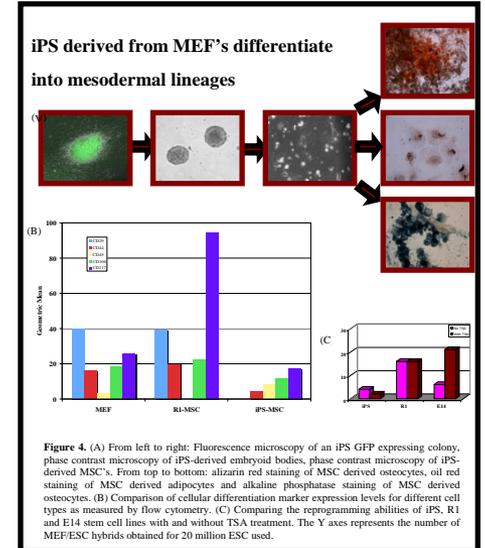


Figure 4. (A) From left to right: Fluorescence microscopy of an iPS GFP expressing colony, phase contrast microscopy of iPS-derived embryoid bodies, phase contrast microscopy of iPS-derived MSC's. From top to bottom: alizarin red staining of MSC derived osteocytes, oil red staining of MSC derived adipocytes 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.

Conclusion

- 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 epigenetic modification invariably 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

- 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. Crane CA, Hovatta M, Miki D, Egger K. Nuclear reprogramming of somatic cells. *Stem Cells* 19: 1161-1171 (2005).
2. Hoshida Y, Kamei M, Yoshida M, Mizuno H, Saito T, Takizawa A. Induced pluripotency and gene expression by linking human pluripotency to human cell lines. *Development* 134: 4047-4056 (2007).
3. Thomson J, Itskovitz E, Shapiro J, Kwiatkowski D, Gokhale P, Yu J, Izpisua-Belmonte J, Suter U, Heintz N, Melnick M. Pluripotent stem cell generation by direct reprogramming. *Development* 134: 303-311 (2007).
4. Thomson J, Itskovitz E, Shapiro J, Kwiatkowski D, Gokhale P, Yu J, Izpisua-Belmonte J, Suter U, Heintz N, Melnick M. Pluripotent stem cell generation by direct reprogramming. *Development* 134: 303-311 (2007).

Making your poster readable - from ~4 feet away!

- Make sure it fits in the space provided; follow the instructions!
- A light background with dark text is often easier to read
- Avoid garish colors and complicated backgrounds
- Use one font and style to integrate text, tables and graphics
- Leave space between each column of your poster
- Use tables and not graphics for small data sets
- Put labels directly on the graphics so complicated legends are not needed

What do I mean by “visible from several feet away”?

- Section heading 48 pt
- Figure heading 30 pt
- General text 28 pt
- Text for labels 20 pt

The Title

- Used by many to decide whether to come to your poster or not
- Should not be too long or contain jargon and abbreviations
- Should state the main focus of your study; if it is too general, few people will come
- Must be visible from 6 feet away

Names and affiliations

- Include first names
- Spell out affiliations that may not be universally recognized
- Street addresses are not necessary
- Smaller than title, but still >72 point font
- Logos and pictures can be nice, but not if they clutter up your poster

Introduction

- Gets the viewer interested and brings them up to speed in the field
- Puts your work into the context of what is known
- Justifies your model system and approach
- Often ends with a clear statement of your specific goals or hypothesis

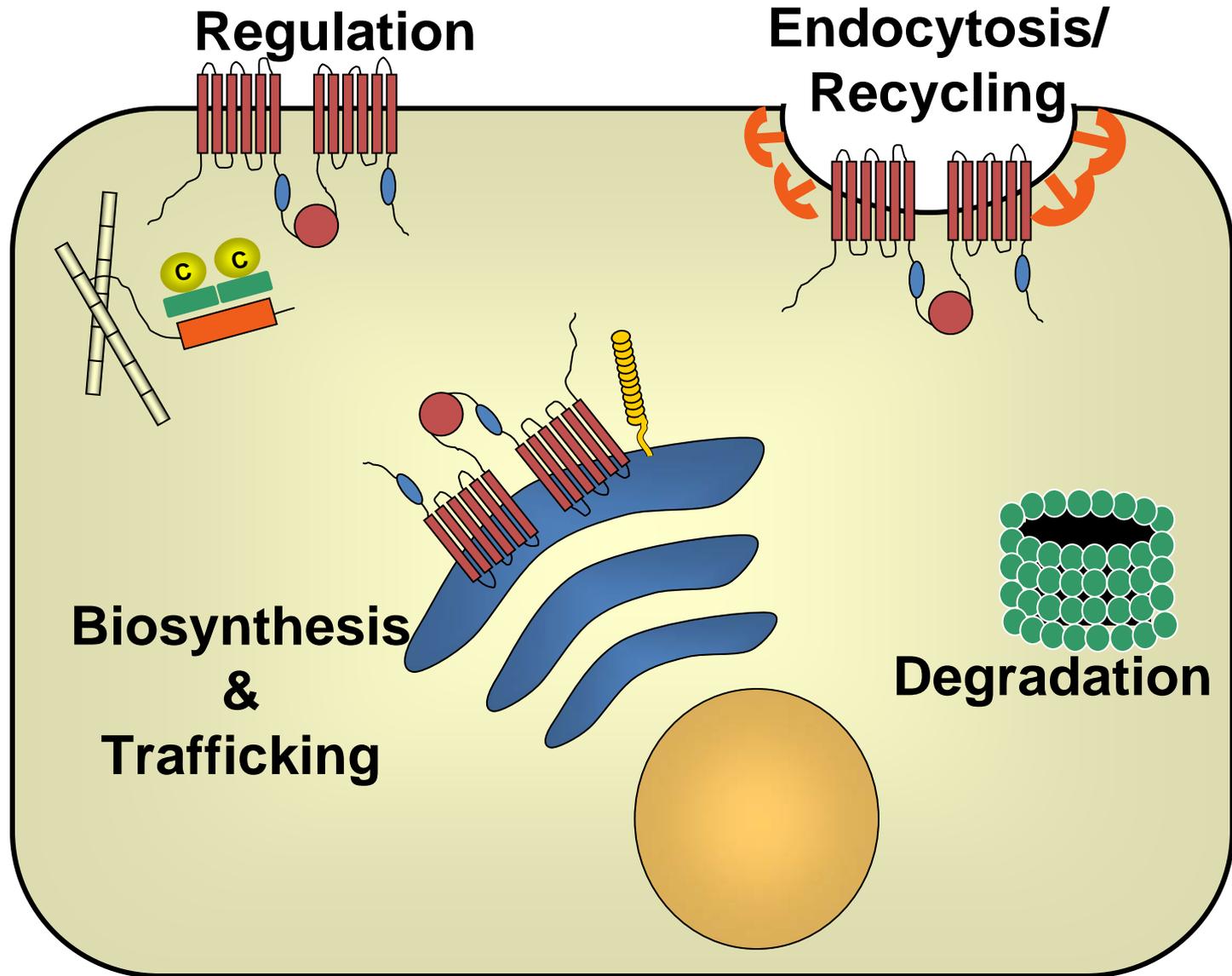
REMEMBER:

- Use figures & flow diagrams, not text
- Use bulleted points, not long paragraphs

INTRODUCTION - an example

The Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) is an apical membrane Cl⁻ channel expressed in a variety of epithelial cells. CFTR is a member of the ABC transporter superfamily with two Nucleotide Binding Domains (NBD) and a large Regulatory (R) domain. Although key to understanding the mechanisms underlying cellular defects in CF, we still know little about cellular factors that regulate CFTR biosynthesis, trafficking, and regulation in polarized cells. Recent data suggests that CFTR exists as part of a multiprotein complex, but few CFTR-interacting proteins have been identified or characterized. Furthermore, we know little about how these interactions are modified in mutant proteins known to cause CF. We used proteomic approaches to identify novel CFTR-interacting proteins and characterized these interactions using a series of biochemical and cellular assays in cells expressing wild-type and mutant CFTR proteins.

Protein interactions regulate all aspects of CFTR biology



Methods

- Details are usually not needed; should be as brief as possible
- Should include graphics and flow charts, and not text, if possible
- Should not be highly referenced (as described in...) or include basic methods

REMEMBER:

Most viewers don't want to read the details; they will ask for details if they want them

Our overall approach:

**USE MS TO IDENTIFY
INTERACTORS**

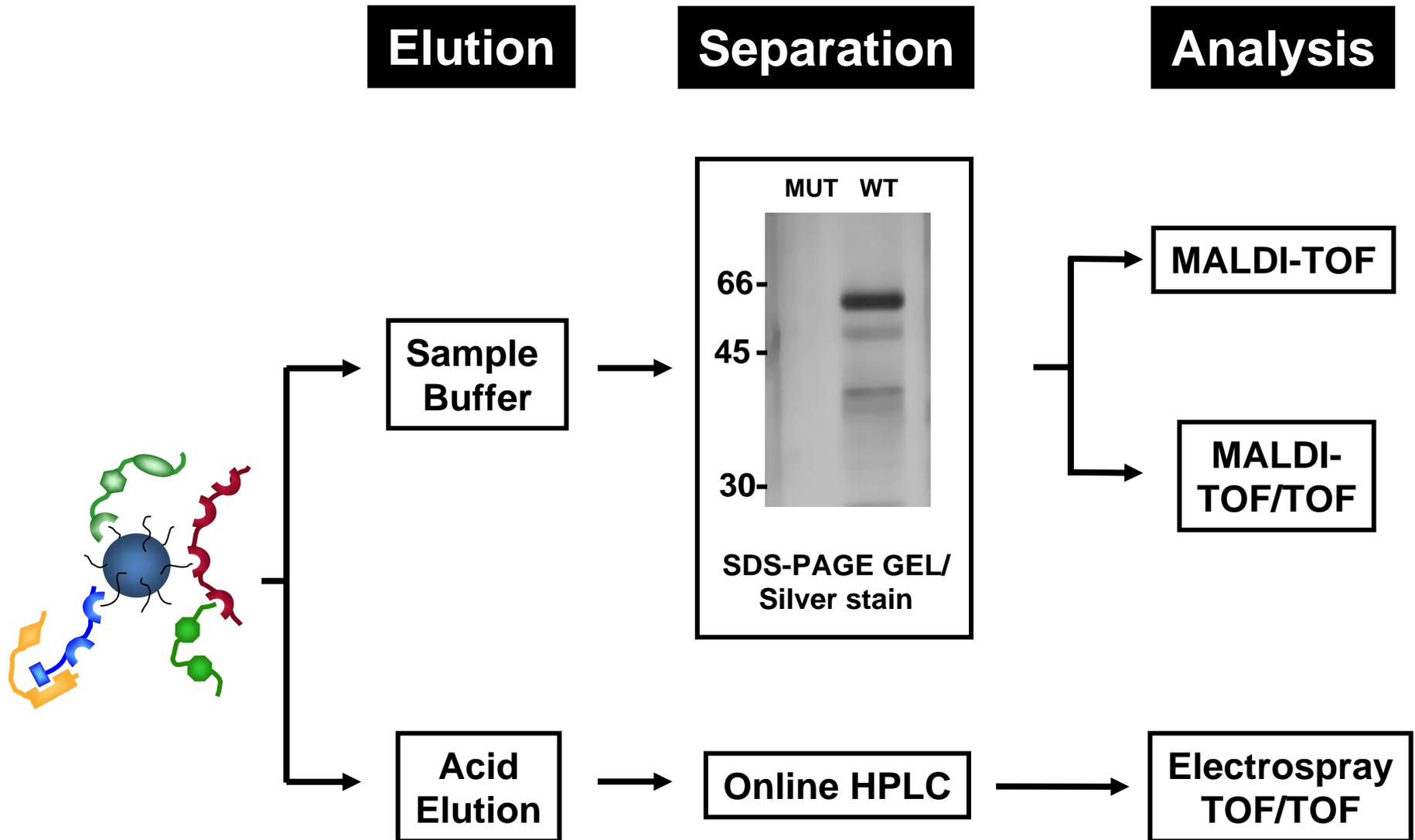


**VALIDATE USING
BIOCHEMICAL APPROACHES**



**CHARACTERIZE USING FUNCTIONAL
ASSAYS**

Sample prep and analysis



Results

- Labels should guide the reader through the data
- Use titles that summarize the results, not ones that focus on the assay or method used
- Figures should be large, labeled clearly and easy to understand without a long legend
- Use tables rather than graphs for small data sets
- All text should be visible from several feet away

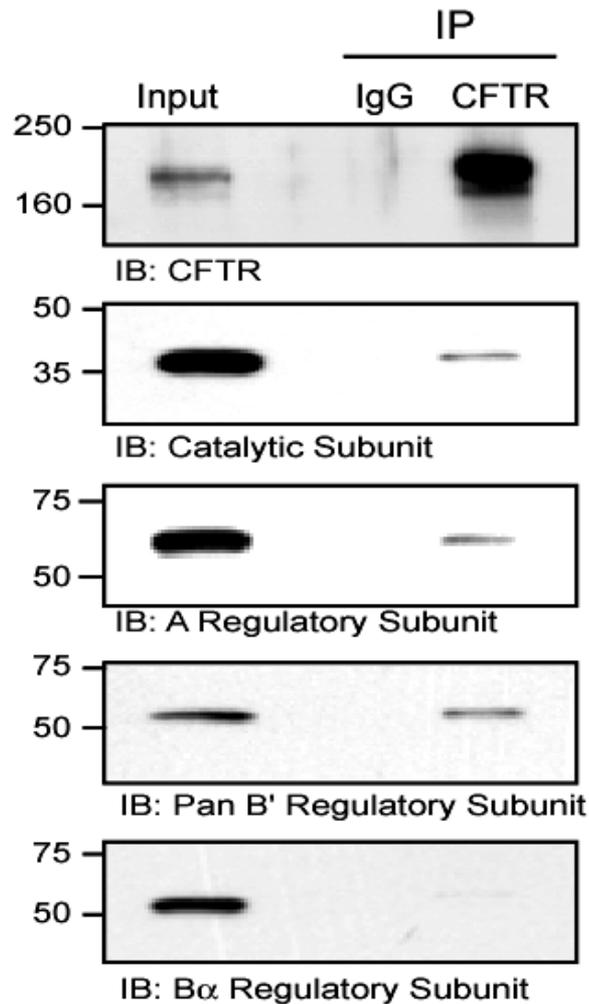
REMEMBER:

You should show the most relevant data,
not **ALL** your data

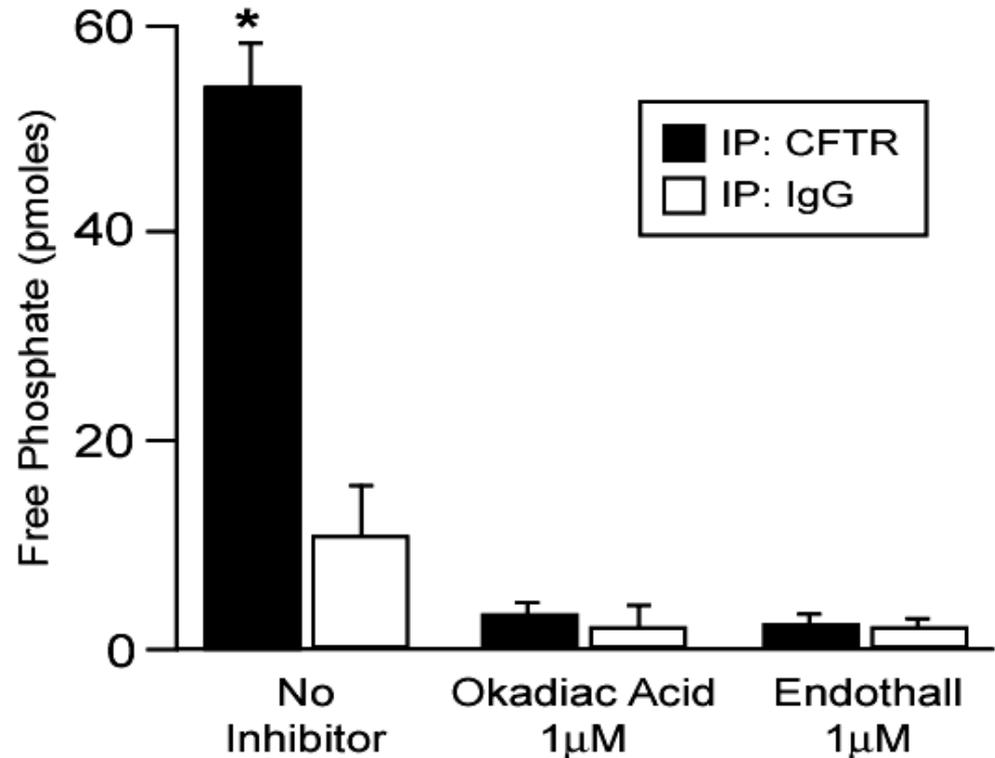
Put your best foot forward!

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

A. Western blot analysis

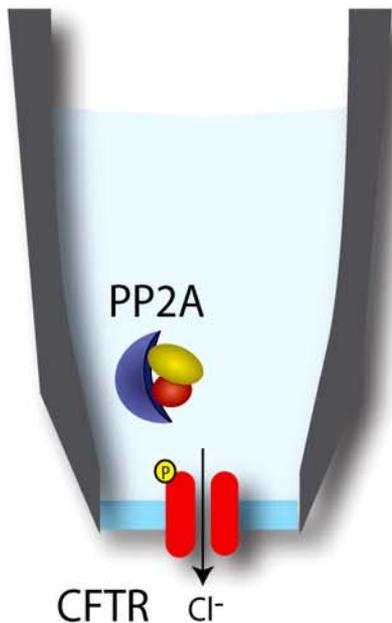


B. Phosphatase activity assay

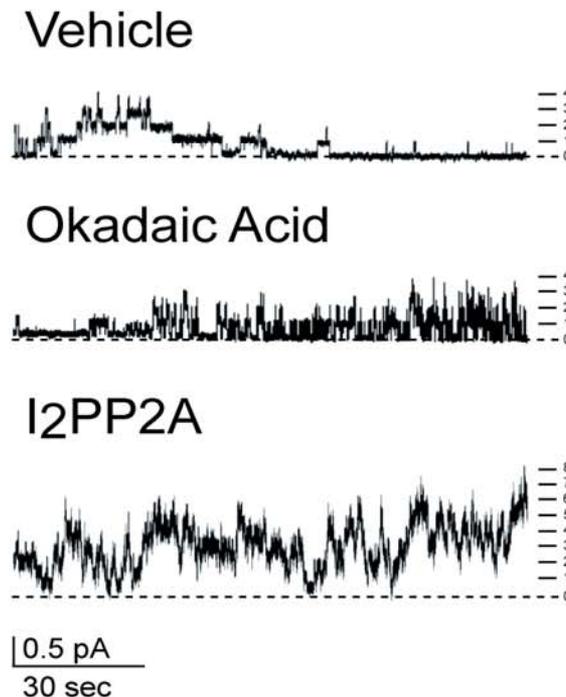


PP2A regulates CFTR channel activity

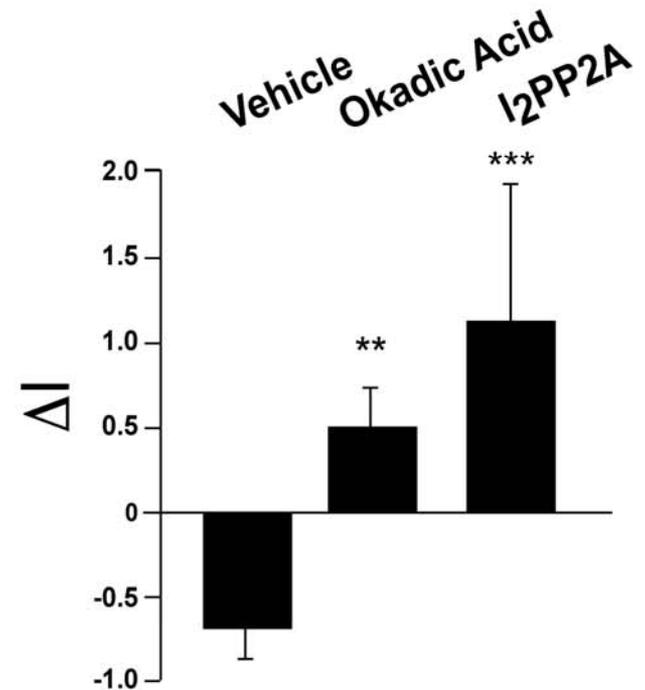
A. Experimental design



B. Single channel recordings



C. Averaged data
N=6





Conclusions

- Use bullet points to highlight major findings
- A model can be very useful
- Consider briefly addressing future plans, but this is not required

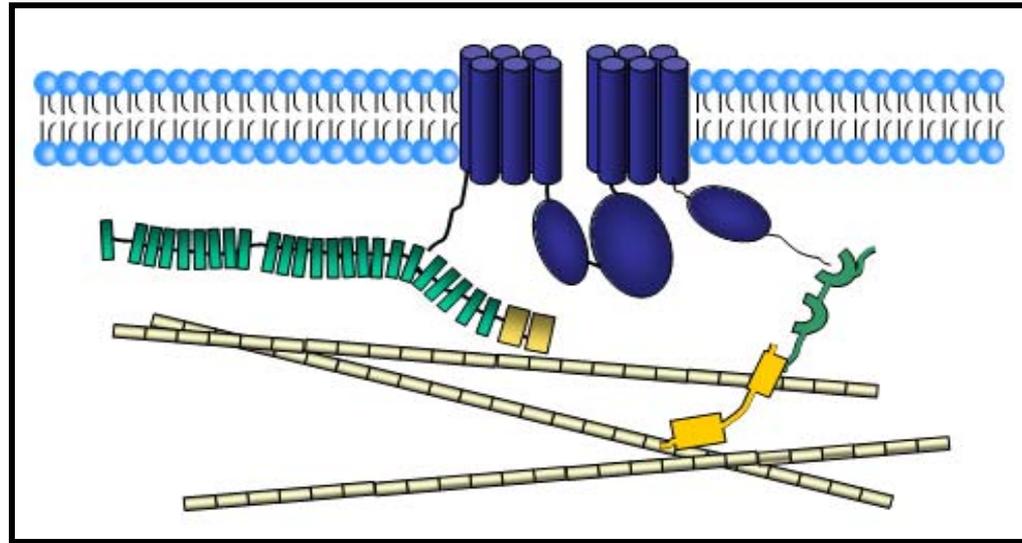
CONCLUSIONS - an example

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

- 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 CF

Filamins may regulate many aspects of CFTR function



Our data suggest three possible functions :

- Stability on the cell surface
- Scaffolding regulatory factors
- Directly altering channel activity

Targeting Human Disease with Virus Mimicry

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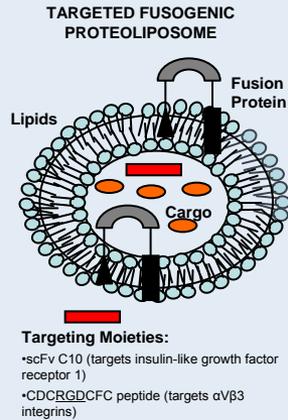
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Abstract

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.



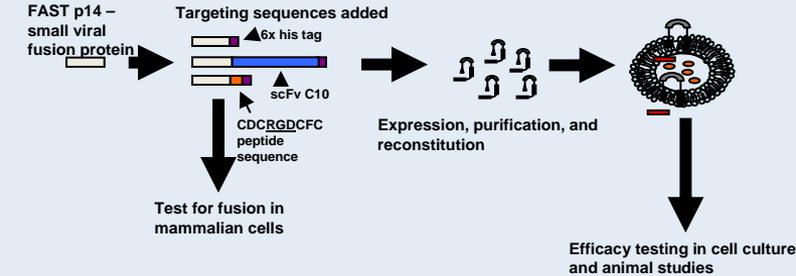
Introduction

The great promise of **nanoparticle delivery** is its ability to salvage drugs or other therapy modalities that have successfully made it far into preclinical 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.

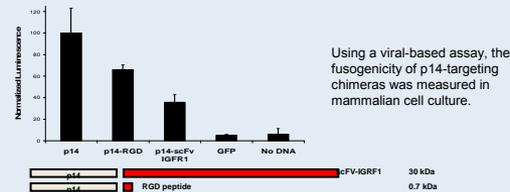


Fig. 2: FAST p14 liposomes can be targeted to specific cell receptors.

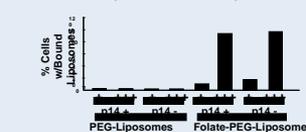


Fig. 3: FAST p14 liposomes promote fusion and intracellular delivery.

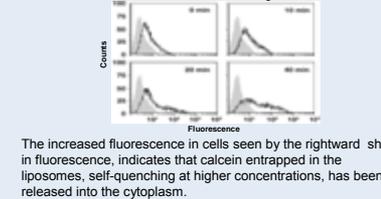
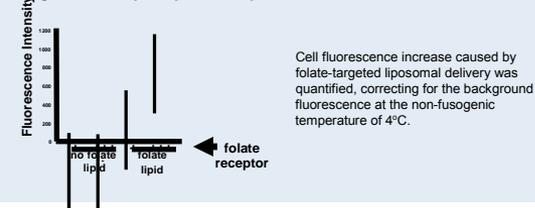


Fig. 4: Targeted FAST p14 liposomes promote increased intracellular delivery.



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



References

1. Information on Clinical Trials. *National Library of Medicine*. www.clinicaltrials.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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- Dimitar Dimitrov**, Center for Cancer Research Nanobiology Program, NCI-Frederick, Frederick, MD
- SAIC** SAIC-Frederick, Inc.
A subsidiary of Science Applications International Corporation

Using digital processes

- Inform your graphics arts service what program and platform you used to prepare your poster
- Beware of using fonts other than System (standard) fonts
- Do not use images and logos directly from the Internet.
- Make sure the service offers electronic approvals
- When checking your proof, make certain to read the text carefully
- Make sure you understand screen vs printing color differences to avoid surprises in the finished poster



Common mistakes

- Too much material
- Too much text
- Poor layout makes reading and digesting the information too difficult
- Blocks of text longer than 10 sentences
- Waiting until last minute to print
- Neglecting to prepare to present your work

USEFUL RULES OF THUMB:

- A poster should be ~40% graphics, 20% text, and 40% empty space
- **It takes 1 - 2 weeks to put together an outstanding poster**

Cleaved Laminin- $\alpha 5\beta 1\gamma 1$ Fragment Activates EGFR in Prostate Cancer Cells

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Abstract

There is a crucial need for increased information about the mechanism by which some prostate cancers become invasive. Laminin- $\alpha 5\beta 1\gamma 1$ (laminin-511) is a major component of the basal lamina surrounding low grade prostate cancer. It is one of the only laminins whose expression persists during the progression from a normal prostate gland to prostate cancer. In order for prostate cancer to metastasize, it must invade through a laminin-511 rich barrier. We have previously shown that the matrix metalloprotease, MT1-MMP, which is expressed in prostate cancer but not in normal prostate tissue, is capable of cleaving the laminin-511 $\alpha 5$ chain into four distinct fragments. These fragments are 45 kDa, 160 kDa, 190 kDa, and 310 kDa in size. This cleavage allows for increased prostate cancer cell migration *in vitro*. Laminin-511 cleavage also occurs *in vivo* in human prostate tissue. Cleavage of laminin-511 and release of laminin-511 fragments leads to altered cell function leading to increased cell migration and invasion in *in vitro* assays. Here, we demonstrate that prostate cancer cells treated with laminin-511 that has been cleaved by MT1-MMP have increased EGFR phosphorylation compared with cells grown on tissue culture plastic or intact laminin-511 in a Western blot. We have purified a recombinant 45kDa laminin-511 N-terminal cleavage fragment, which contains laminin EGF-like domains. Treatment of prostate cancer cells with soluble recombinant fragment demonstrates that the cleaved laminin fragment acts as a matrine, activating the EGFR on prostate cancer cells in a Western blot. This work demonstrates that increased MT1-MMP expression in prostate cancer not only cleaves the major laminin surrounding prostate cancer to clear a path for migration, but also releases active fragments from the laminin-511 that signal for increased migration.

Prostate Cancer Progression



Nagle RB, Kwon ED, Wei C, Bowden GT, Gross AE, J Cell Biochem. 19:232-237, 1994

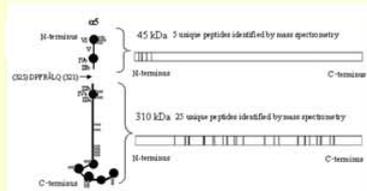


Figure 1. Laminin fragments resulting from MT1-MMP treatment. Treating LM-511 with recombinant catalytic domain produced 4 identifiable cleavage products in a Coomassie stained gel. Bands were excised and analyzed by mass spectrometry to identify LM- $\alpha 5$ chain coverage of each fragment. Results are shown for the 45 kDa band and the 310 kDa band. Potential cleavage site was identified based on fragment peptide coverage of total protein and MT1-MMP consensus cleavage sites in the LM- $\alpha 5$ chain.

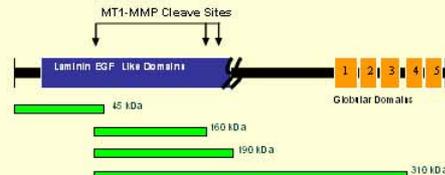


Figure 2. Potentially biologically active LM-511 fragments. A schematic of the LM- $\alpha 5$ chain is shown. Potential MT1-MMP cleavage sites are indicated and fragments with potential biologic activity are indicated in green. Fragments produced by MT1-MMP cleavage contain LM-EGF-like domains, suggesting these fragments may be biologically active independent of the intact LM-511 protein.



Figure 3. Activation of EGFR on cells plated on cleaved LM-511. LM-511 (μ g) was coated on a 6-well tissue culture plate and treated with catalytic domain of MT1-MMP (2.1 nmol) or left intact. DU-146 cells were then plated on uncoated and LM-511 coated wells. EGF was added to an uncoated well as a control for activation. 16 hours after plating, cells were lysed and analyzed for EGFR activation at Tyr 1068. Results demonstrate EGFR activation when cells were plated on intact LM-511, with an increase in EGFR activation when cells were plated on cleaved LM-511.



Figure 4. Activation of EGFR with soluble LM-511 fragments. DU-146 cells were seeded in a 6-well tissue culture plate and stimulated with: EGF, LM-511 fragments produced by incubating intact LM-511 with catalytic domain of MT1-MMP 16 hr, intact LM-511, or were left untreated. Results demonstrate that cleaved LM-511 fragments activate EGFR to a greater extent than intact LM-511, and that the receptor is not activated with no treatment. Laminin A/B protein was used as a loading control.



Figure 5. Structural representation of LM- $\alpha 5$ chain 45 kD fragment. The LM- $\alpha 5$ chain 45 kDa fragment we have produced in E. Coli has been modeled based on its amino acid sequence. The fragment contains a complete EGF-like domain at its C-terminus.

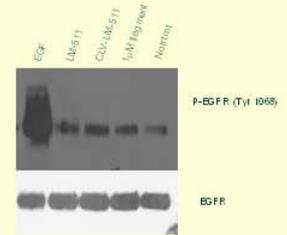


Figure 6. LM- $\alpha 5$ 45 kD fragment activates EGFR. DU-146 cells were treated with EGF, intact soluble LM-511, cleaved soluble LM-511, 45 kDa soluble fragment, or left untreated. Results indicate that the 45 kD fragment activates EGFR above endogenous levels (untreated). Total EGFR protein is shown as a loading control.

Conclusions

- LM-511 cleaved with MT1-MMP contains potentially bio-active fragments.
- Cleaved LM-511 fragments activate the EGFR at Tyr 1068.
- Recombinant 45 kDa LM- $\alpha 5$ chain demonstrates some activity in activating the EGFR.

Future Directions

- Test fragments for activation of other phosphorylation sites on EGFR.
- Examine downstream signaling pathway activation after treatment with fragments.
- Examine bio-activity of higher MW cleavage recombinant LM- $\alpha 5$ fragments (160, 190, 310 kDa).
- Determine which fragment(s) is/are responsible for activation of migration in prostate cancer cells.

Selected References

- Nagle RB, et al. *J Cell Biochem Suppl*. 1994. 19: 232-237.
 Bair EL et al. *Neoplasia* 2005. 7(4): 380-389.
 Shank S, et al. *J Cell Biol*. 2003. 161(1): 197-209.

This work is supported by DOD PC050430 and NIH PO1 CA56666



- Prostate cancer is surrounded by a basal lamina consisting predominantly of LM-511.
- We have previously demonstrated that MT1-MMP can cleave the LM-511 $\alpha 5$ chain into 4 distinct fragments: 45, 160, 190, and 310 kD.
- LM-511 cleavage by MT1-MMP resulted in decreased prostate cancer cell adhesion.
- LM-511 cleavage by MT1-MMP also resulted in an increase in prostate cancer cell migration.
- Prostate cancer cells that express MT1-MMP can cleave purified LM-511 coated on a tissue culture plate. When the LM-511 is cleaved by cells, the 45 kD LM- $\alpha 5$ chain fragment is released into the medium.

Hypothesis

- LM-511 cleavage by MT1-MMP results in decreased adhesion and increased migration of prostate cancer cells due to release of biologically active LM- $\alpha 5$ fragments containing EGF-like domains that function as matrine, can bind and activate the EGF receptor.

Making a terrific poster is only the first step

- How you present yourself and discuss your work is critical
- Wear a name tag & make sure to introduce yourself
- Practice! People will wander away if you are long-winded and hard to follow
- Listen carefully to questions; be prepared to provide detail for experts and an overview for interested novices
- When it counts bring additional data, reprints, business cards, etc