Creating and Presenting Dynamic Posters

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Poster sessions are a lot like farmers markets
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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
- are visually appealing & readable
- are succinct
- presented clearly & with enthusiasm
- tell a story
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
The most important rules

- Have your poster fit in the space provided
- Make your poster readable from 4 feet away
- Don’t try to put EVERYTHING on your poster
- Limit text
More tips for readable posters

- Use dark text on a light background
- Avoid garish colors and complicated backgrounds
- Use one font and style for the whole poster
- Leave space between columns
- Try to use bullet points and pictures instead of text
- Label figures clearly
How large should the fonts be?

- **Title 72 pt**
- **Section heading 48 pt**
- **Figure heading 30 pt**
- **General text 28 pt**
- **Text for labels 20 pt**
Typical parts of a poster

- Title
- Authors and affiliations
- Introduction
- Goals (optional)
- Methods
- Results
- Conclusions
- Future directions (optional)
- References (optional)
- Acknowledgements
# Short descriptive poster title

## Authors & affiliations

<table>
<thead>
<tr>
<th>Introduction</th>
<th>Data 1</th>
<th>Data 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goals</td>
<td>Data 2</td>
<td>Conclusions</td>
</tr>
<tr>
<td>Methods</td>
<td>Data 3</td>
<td>Other stuff</td>
</tr>
</tbody>
</table>

- 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 reverses the epigenetic and subsequently the differentiation identity of a cell to a pluripotent embryonic soma-cell state. Embryonic soma-cells (ESC) derived from the inner cell mass of the blastocyst, are pluripotent. They are immortalized, possess long term renewal ability and can give rise to the entire embryo excluding the extra-embryonic tissues. As such they are highly prized for patient specific disease replacement. The birth of Dolly in 1997, by somatic cell nuclear transfer is a good example of reticular gene flipping in vivo. In 2006, Yamanaka et al. showed that this “reprogramming environment” for epigenetic memory of cells is overwhelmed to a pluripotent-like state. A somatic cell becomes pluripotential-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 induce four transcription factor genes, which when transfected into somatic cells give rise to induced pluripotent stem (iPS) cells.

Objective

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

- Baculovirus mediated fusion of two ESC lines to induce embryonic fibroblasts (MEFs) reprogramming
  1. In reprogramming ability of different ESC lines, as measured by the overall number of tetraploid hybrids obtained. “In vivo”?
  2. Are chromatin remodeling markers involved in mediating this phenotype and if so how?

Materials and Methods

Fusion procedure

IPS generation

IPS derived from MEF's differentiate into mesodermal lineages

Conclusion

- Reprogrammed hybrids exhibit pluripotent-like characteristics such as morphology, long term renewal ability, embryoid body formation, gene expression profile and chromosomal patterns
- Different ESC lines display characteristic higher-order chromatin structures. While it is true that one can design specific modifications to a single nuclear donor, to have minimal modulation necessarily translates to an identical biological outcome
- The Yamanaka group has shown that chromatin remodeling is likely the key mechanism underlying the transition between differentiated cells and iPSCs
- When iPSCs are fused again with somatic cells from which they themselves originated, they reprogram them, although the efficiency of this reprogramming further needs investigation
- iPSC differentiation into MSC's but flow cytometry analysis indicates that there are significant differences in the cellular differentiation markers accessed in standard to vitro MEF's

Future

- iPSC heterogeneity with respect to pluripotency. Is attempts to “quantify” such enormous differences we will investigate iPSC chromatin epigenetic remodeling
- The in vivo aspect of our work will focus on examining the functional potential of iPSC-derived differentiated cells

Present iPSC generating methods use such that these “golden cells” are still diseased for translational use due to its increased oncogenic potential. We are working on finding new strategies to efficiently generate clinically relevant iPSCs

References

The Title

- Your whole project boiled down to just a few words
- Used by many to decide whether to visit your poster
- 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 and last names
- Spell out affiliations that may not be universally recognized
- Street addresses are not necessary
- Smaller than title, but still big
- Logos and pictures can be nice, but not if they clutter up your poster
Introduction

- Gets the viewers 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:

• Keep it brief
• Use figures & diagrams if possible
• Use bullet points if possible
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 protein 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

Biosynthesis & Trafficking

Regulation

Endocytosis/Recycling

Degradation

Protein interactions regulate all aspects of CFTR biology.
Our Goals:

- To use mass spectrometry to identify proteins that associate with CFTR
- To use biochemical and cell-based assays to study novel CFTR-interacting proteins
- To study novel interacting proteins in cells expressing WT and mutant versions of CFTR

We focused on 3 mutations:
- N-terminal, S13F
- R domain, ΔF508
- C-terminal, ΔCT
Methods

- As brief as possible
- Use graphics and flow charts, rather than text, if possible
- No need to describe 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

Elution

Sample Buffer

Acid Elution

Separation

SDS-PAGE GEL/Silver stain

Online HPLC

Analysis

MALDI-TOF

MALDI-TOF/TOF

Electrospray TOF/TOF
Results

- Include only a few key figures or tables
- Each figure should have a title that summarizes the results
- Figures should be large, labeled clearly and be easy to understand without a long legend
- All text should be visible from several feet away

REMEMBER:
- Figures should make sense even when you’re not at your poster
- Put your best foot forward!
PP2A subunits & activity co-precipitate with CFTR in airway cells

A. Western blot analysis

B. Phosphatase activity assay

![Western blot analysis and phosphatase activity assay](image_url)
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
- Consider displaying a model
- Possible to use summary paragraph or summary bullet points instead

REMEMBER:
Less is more!
CONCLUSIONS –

We used affinity purification to identify proteins that associate with CFTR and found that 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.
Isn’t this better?

- The B’ε subunit of PP2A directly associates with the COOH-terminus of CFTR
- PP2A protein and activity co-immunoprecipitate 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
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
Future Directions

- Optional section
- Use bullet points
- Be brief
**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 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. 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, sequestration, 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. 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 trimmer of heterodimers at ~500 kD.

**Methodology**

1. **Fast p14 - small viral fusion protein**
2. **Targeting sequences added**
3. **Expression, purification, and reconstitution**
4. **Test for fusion in mammalian cells**
5. **Efficacy testing in cell culture and animal studies**

**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 innovation in nanomedicine.

**References**


**Collaborators**

Roy Duncan, Faculty of Medicine, Department of Microbiology and Immunology, Dalhousie University, Nova Scotia, Canada

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

Dimiter Dimitrov, Center for Cancer Research Nanobiology Program, NCI-Frederick, Frederick, MD
A relationship between yeast nuclear morphology, large structural nucleoporins, and the SUN domain protein MPS3

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1NIDDK, National Institutes of Health. Bethesda, Maryland 20892.
2Stowers Institute for Medical Research, Kansas City, MO 64110.

Abstract:
Cells lacking the non-essential SPOT gene have increased phospholipid synthesis, altered lipid composition, and expansion of the nuclear envelope. SPOT has genetic interactions with genes encoding nuclear pore complex (NPC) components, suggesting that NPCs are affected by the altered nuclear envelope in spo7Δ cells. In order to identify other nuclear envelope components sensitive to SPOT deletion, we adopted a candidate approach. One interesting candidate is the SUN-domain gene MPS3. The Mps3p protein localizes to the spindle pole body (SPB) and around the nuclear periphery, and MPS3 has been implicated in SPB duplication as well as other nuclear functions. Cells lacking SPOT were compromised for viability when combined with mps3Δ mutations specifically affecting Mps3p function at the SPB. This suggests that SPOT deletion exacerbates the SPB defect of mps3Δ mutants, rather than affecting its other nuclear functions. High-copy suppressors of the synthetic lethality between spo7Δ and mps3Δ were identified, including dominant-negative forms of the nucleoporins NUP97 and NIC96. Strikingly, deletion of NUP157 completely rescues the SPB defect of mps3Δ mutants. Furthermore, deletion of NUP157 or either of the pore membrane (pom) nucleoporins POM152 or POM34 rescues temperature-sensitivity of a subset of SPB mutants, including mps3Δ. Based on our data, we propose 3 models for the relationship between nucleoporins, MPS3 and the SPB.

Working hypothesis:
Altered nuclear envelope lipid composition in spo7Δ cells affects the function of proteins and complexes that reside in the nuclear membrane.

Candidate gene: MPS3

- Nuclear pore protein
- Required for SPB duplication
- Important for nuclear periphery

Figure 1: Genetic interactions between SPOT and MPS3

<table>
<thead>
<tr>
<th>Gene</th>
<th>Function</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>NUP97</td>
<td>SPB duplication</td>
<td>++</td>
</tr>
<tr>
<td>NUP157</td>
<td>SPB duplication</td>
<td>++</td>
</tr>
<tr>
<td>NUP97</td>
<td>NUP157</td>
<td>++</td>
</tr>
<tr>
<td>NIC96</td>
<td>SPB duplication</td>
<td>++</td>
</tr>
</tbody>
</table>

Figure 2: Genetic interactions between SPOT and other SPB genes

<table>
<thead>
<tr>
<th>Gene</th>
<th>Function</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>mps2-381</td>
<td>mps2Δ</td>
<td>++</td>
</tr>
<tr>
<td>ksr1Δ</td>
<td>ksrΔ</td>
<td>++</td>
</tr>
<tr>
<td>brrΔ</td>
<td>brrΔ</td>
<td>++</td>
</tr>
<tr>
<td>spc22Δ</td>
<td>spc22Δ</td>
<td>++</td>
</tr>
<tr>
<td>spo42Δ</td>
<td>spo42Δ</td>
<td>++</td>
</tr>
</tbody>
</table>

Figure 3: Suppressor screen schematic and results:

<table>
<thead>
<tr>
<th>Gene</th>
<th>Function</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPC22</td>
<td>High-copy suppressor screen</td>
<td>++</td>
</tr>
<tr>
<td>mps2-381</td>
<td>mps2Δ</td>
<td>++</td>
</tr>
<tr>
<td>ksr1Δ</td>
<td>ksrΔ</td>
<td>++</td>
</tr>
<tr>
<td>brrΔ</td>
<td>brrΔ</td>
<td>++</td>
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Figure 4: Truncated forms of NUP157 and NIC96 partially suppress synthetic lethality

<table>
<thead>
<tr>
<th>Gene</th>
<th>Function</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
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<td>ksrΔ</td>
<td>++</td>
</tr>
<tr>
<td>brrΔ</td>
<td>brrΔ</td>
<td>++</td>
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</table>

Figure 5: Both suppressors rescue ts of mps3Δ mutants

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<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>mps2-381</td>
<td>mps2Δ</td>
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<td>ksr1Δ</td>
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<td>++</td>
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<tr>
<td>brrΔ</td>
<td>brrΔ</td>
<td>++</td>
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Summary:
- There are strong genetic interactions between SPOT and MPS3, specifically affecting MPS3 function at the SPB.
- SPOT interacts with other SPB mutants, suggesting that SPOT influences SPB structure or function.
- High-copy expression of truncated forms of NUP157 and NIC96 rescues synthetic lethality between spo7Δ and mps3Δ and partially rescues the temperature-sensitivity of mps3-1 mutants.
- Deletion of NUP157, POM152, or POM34 rescues the temperature-sensitivity of mps3Δ mutants and a subset of other SPB mutants.
- Deletion of NUP157 restores SPB function to mps3-1 mutants, establishing a functional link between nuclear pore proteins and the SPB.

Models:
1. Nucleoporins affect the relative localization of Mps3p to the nuclear envelope vs the SPB. In the absence of NUP157, Mps3p preferentially localizes to the SPB, and this rescues Mps3p function at the SPB.
2. Nuclear pore complexes and the SPB compete for insertion into the nuclear envelope, perhaps because of a shared insertion factor. When NPC biogenesis is compromised, defects stemming from improper SPB duplication or insertion are ameliorated.
3. Nuclear pore complexes affect nuclear membrane dynamics, which affects SPB duplication or insertion.
Poster Formats

- It is important to talk in advance with your mentor about options for poster design and printing

- Examples of format options:
  - One large page printed on a poster printer
  - Individual panels printed on 8 ½ x 11 paper and mounted on colorful poster board
Before you print

- Discuss options with your mentor way ahead of time—you may need an appointment
- Print out a draft in 8½ x 11 format to proofread and edit
- Make sure you’re using standard fonts and high quality images
- Think about saving a pdf version (this is especially useful if switching from a Mac to PC)
If you are using a graphics arts service

- Inform the service what program and platform you used to prepare your poster
- 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
Common mistakes

- Too much material
- Too much text
- Poor layout
- Blocks of text longer than 10 sentences
- Waiting until last minute to print
- Neglecting to prepare your presentation
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
Making a terrific poster is only the first step

- How you present yourself and discuss your work is critical
- Wear a name tag & 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
- If necessary, bring additional data, reprints, business cards, etc
New this year!

This year all posters will be judged by NIH scientists who will be looking at the following:

- Poster Content
- Poster Appearance
- Student Presentation