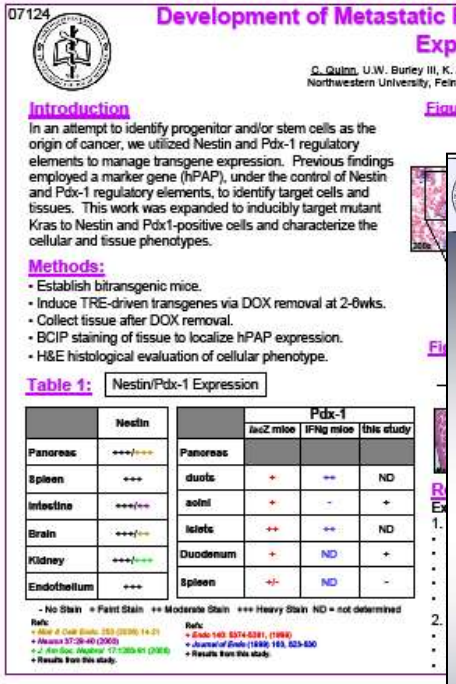
Collaborative Learning and Integrated Mentoring in the Biosciences

Posters-Best Practices

Posters are a critical component of science communication – an important element in a successful scientific career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Development of Metastatic Precursor Lesions in Murine Pancreas following Mutant Kras Expression in Adult Pdx-1 Positive Cells

C. Quinn, U.W. Burley III, K. Adrian, M.R. Barron, C. Pelham, T.E. Adrian, R.H. Bell Jr., R.J. MacDonald, G.Y. Yang, P.J. Grippo
Northwestern University, Feinberg School of Medicine, Department of Surgery and Robert H Lurie Comprehensive Cancer Center

Introduction

In an attempt to identify progenitor and/or stem cells as the origin of cancer, we utilized Nestin and Pdx-1 regulatory elements to manage transgene expression. Previous findings employed a marker gene (hPAP), under the control of Nestin and Pdx-1 regulatory elements, to identify target cells and tissues. This work was expanded to inducibly target mutant Kras to Nestin and Pdx1-positive cells and characterize the cellular and tissue phenotypes.

Methods:

- Establish bitransgenic mice.
- Induce TRE-driven transgenes via DOX removal at 2-6wks.
- Collect tissue after DOX removal.
- BCIP staining of tissue to localize hPAP expression.
- H&E histological evaluation of cellular phenotype.

Table 1: Nestin/Pdx-1 Expression

	Nestin
Pancreas	+++/+++
Spleen	+++
Intestine	+++/++
Brain	+++/++
Kidney	+++/+++
Endothelium	+++

	Pdx-1		
	lacZ mice	IFNg mice	this study
Pancreas			
ducts	+	++	ND
acini	+	-	+
islets	++	++	ND
Duodenum	+	ND	+
Spleen	+/-	ND	-

- No Stain + Faint Stain ++ Moderate Stain +++ Heavy Stain ND = not determined

Figure 1: hPAP Expression in Mouse Pancreas

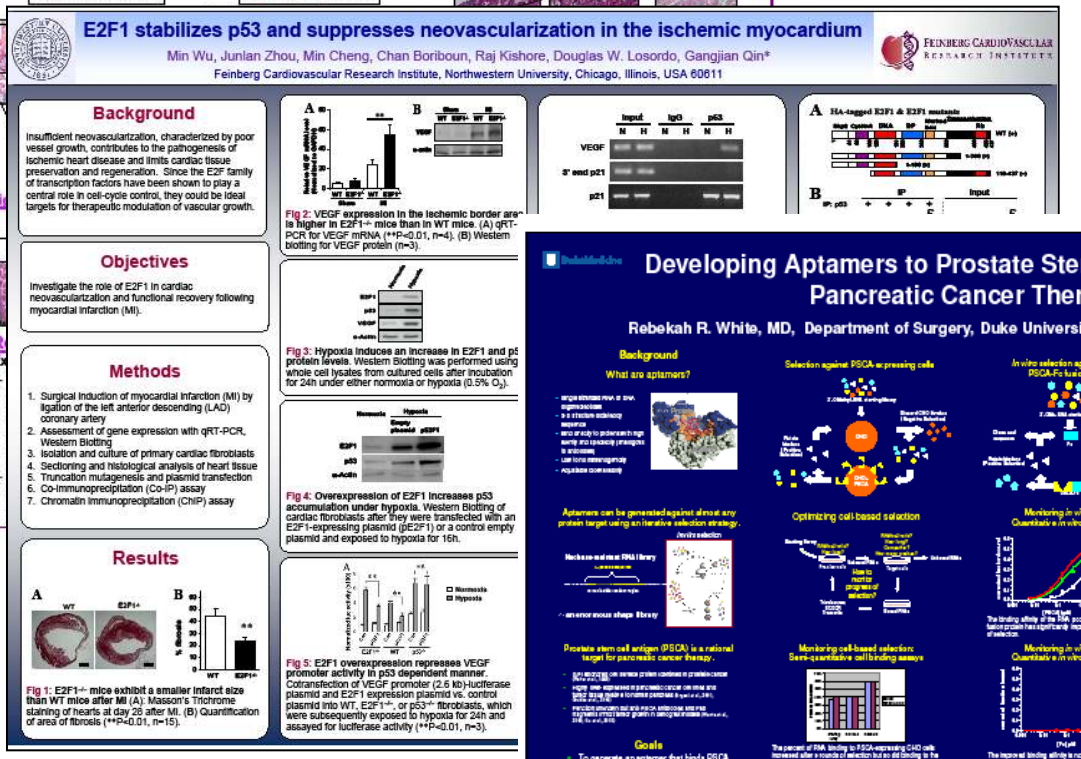
Nest-tTA/TRE-hPAP

Pdx-1tTA/TRE-hPAP

Figure 3: Nestin Targeting: Intestinal and Pancreatic lesions

intestine

pancreas


E2F1 stabilizes p53 and suppresses neovascularization in the ischemic myocardium

Min Wu, Junlan Zhou, Min Cheng, Chan Boriboun, Raj Kishore, Douglas W. Losordo, Gangjian Qin*
Feinberg Cardiovascular Research Institute, Northwestern University, Chicago, Illinois, USA 60611

Feinberg Cardiovascular Research Institute

Background

Insufficient neovascularization, characterized by poor vessel growth, contributes to the pathogenesis of ischemic heart disease and limits cardiac tissue preservation and regeneration. Since the E2F family of transcription factors have been shown to play a central role in cell-cycle control, they could be ideal targets for therapeutic modulation of vascular growth.

Objectives

Investigate the role of E2F1 in cardiac neovascularization and functional recovery following myocardial infarction (MI).

Methods

1. Surgical induction of myocardial infarction (MI) by ligation of the left anterior descending (LAD) coronary artery
2. Assessment of gene expression with qRT-PCR, Western Blotting
3. Isolation and culture of primary cardiac fibroblasts
4. Sectioning and histological analysis of heart tissue
5. Truncation mutagenesis and plasmid transfection
6. Co-Immunoprecipitation (Co-IP) assay
7. Chromatin Immunoprecipitation (ChIP) assay

Results

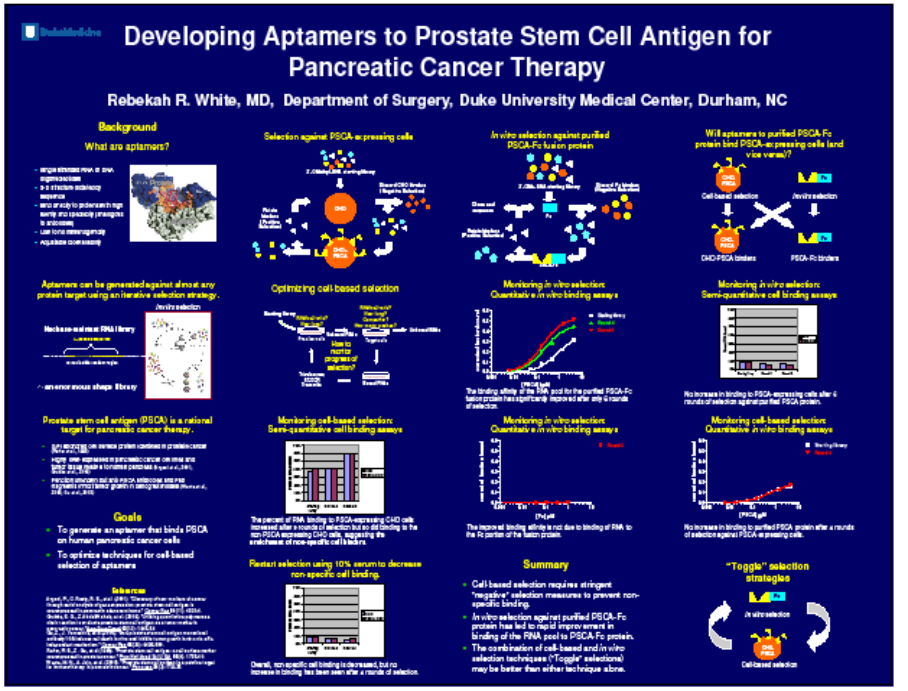
Fig 1: E2F1$^{-/-}$ mice exhibit a smaller infarct size than WT mice after MI (A): Masson's Trichrome staining of hearts at day 28 after MI. (B) Quantification of area of fibrosis (**P<0.01, n=15).

Fig 2: VEGF expression in the ischemic border area is higher in E2F1$^{-/-}$ mice than in WT mice. (A) qRT-PCR for VEGF mRNA (**P<0.01, n=4). (B) Western blotting for VEGF protein (n=3).

Fig 3: Hypoxia induces an increase in E2F1 and p53 protein levels. Western Blotting was performed using whole cell lysates from cultured cells after incubation for 24h under either normoxia or hypoxia (0.5% O_2).

Fig 4: Overexpression of E2F1 increases p53 accumulation under hypoxia. Western Blotting of cardiac fibroblasts after they were transfected with an E2F1-expressing plasmid (pE2F1) or a control empty plasmid and exposed to hypoxia for 16h.

Fig 5: E2F1 overexpression represses VEGF promoter activity in p53 dependent manner. Cotransfection of VEGF promoter (2.6 kb)-luciferase plasmid and E2F1 expression plasmid vs. control plasmid into WT, E2F1$^{-/-}$, or p53$^{-/-}$ fibroblasts, which were subsequently exposed to hypoxia for 24h and assayed for luciferase activity (**P<0.01, n=3).


Developing Aptamers to Prostate Stem Cell Antigen for Pancreatic Cancer Therapy

Rebekah R. White, MD, Department of Surgery, Duke University Medical Center, Durham, NC

Background

What are aptamers?

Prostate stem cell antigen (PSCA) is a rational target for pancreatic cancer therapy.

Goals

- To generate an aptamer that binds PSCA on human pancreatic cancer cells
- To optimize techniques for cell-based selection of aptamers

Selection against PSCA-expressing cells

Aptamers can be generated against almost any protein target using an iterative selection strategy.

Optimizing cell based selection

Monitoring cell-based selection: Semi-quantitative cell binding assays

Restart selection using 10% serum to decrease non-specific cell binding.

In vitro selection against purified PSCA-Fc fusion protein

Monitoring in vitro selection: Quantitative in vitro binding assays

Monitoring cell-based selection: Quantitative in vitro binding assays

Summary

- Cell based selection requires stringent "negative" selection measures to prevent non-specific binding.
- In vitro selection against purified PSCA-Fc protein has led to rapid improvement in binding of the RNA pool to PSCA-Fc protein.
- The combination of cell-based and in vitro selection techniques ("Toggle" selections) may be better than either technique alone.

Will aptamers to purified PSCA-Fc protein bind PSCA-expressing cells (and vice versa)?

Monitoring in vitro selection: Semi-quantitative cell binding assays

Monitoring cell-based selection: Quantitative in vitro binding assays

"Toggle" selection strategies

Posters sessions provide opportunities for:

- Personal interaction
  - Networking
    - Potential collaboration

Agenda

1 – Writing Poster Titles & Figure Titles

Writing titles as "takeaways" – i.e., complete thoughts that cue the reader to the meaning of your overall poster and each figure

2 – Designing for Readability

Developing discrete sections; eliminating words so your poster is not "text heavy"; cueing the reader how to view your information

3 – Telling the Story

Creating "flow;" organizing your introduction; framing your methods, conclusions, and next steps

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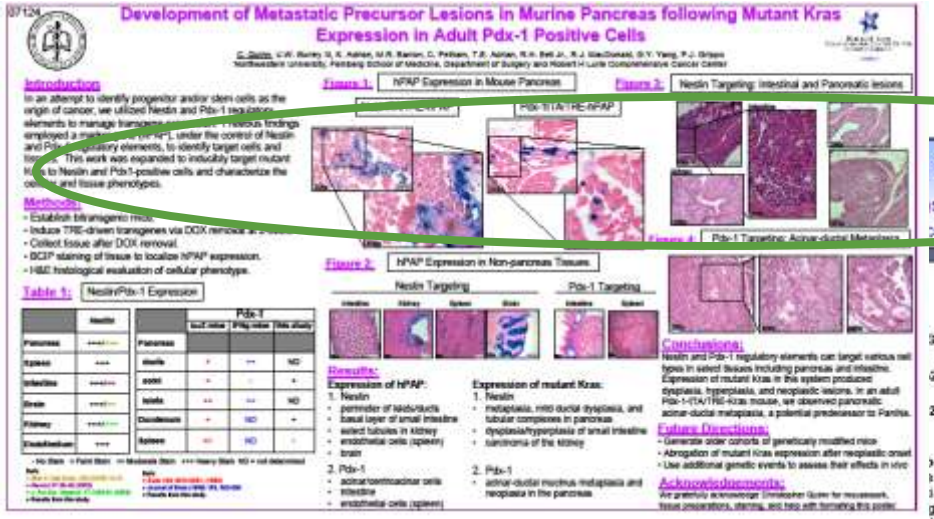
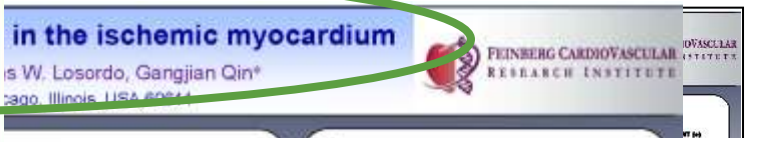
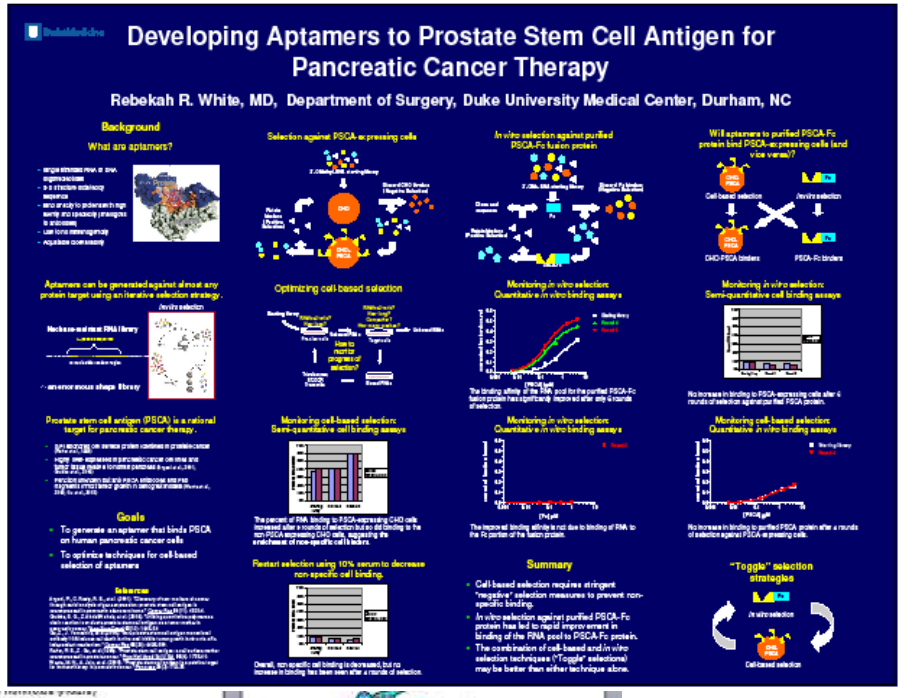
Best posters are snapshots of your work, and good titles—main and figure– are critical to communicating your research







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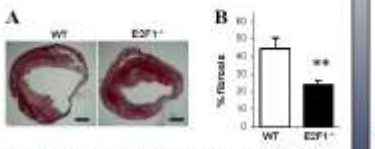

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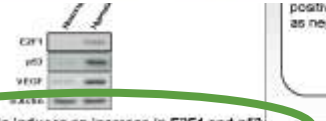

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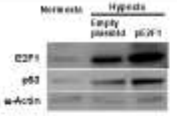

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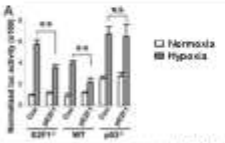

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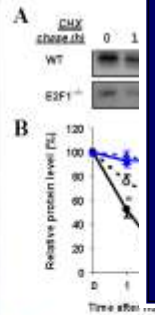


Fig 7: Under hypoxia, E2F1 prolongs the p53 decay half-life and attenuates its degradation via ubiquitin-proteasomal pathway. WT and E2F1$^{-/-}$ cardiac fibroblasts were cultured under hypoxia for 6h then cyclohexamide (CHX) was added (40 mg/mL) with or without the proteasome inhibitor lactacystin (L). Cell lysates were prepared at the indicated time points after the addition of CHX and analyzed by immunoblotting with anti-p53 Ab. (**A**) Representative immunoblots of p53. (**B**) p53 levels were quantified by densitometry and plotted versus time (n=4, *p<0.05, **P<0.01).

Future Plan

1. Fine-map p53 binding site on E2F1
2. Engineer small E2F1 peptide to block E2F1:p53 interaction, de-repress VEGF expression and enhance neovascularization for ischemic heart disease

First, what is the difference between poster title #1 and poster title #2?

Title 1

E2F1 effects on p53 and neovascularization in the ischemic myocardium

Title 2

E2F1 stabilizes p53 and suppresses neovascularization in the ischemic myocardium

What makes poster title #1 a *Topic* title and poster title #2 a *Message* title?

Topic title

E2F1 effects on p53 and neovascularization in the ischemic myocardium

1. Key word – descriptive noun with no relationship or causality

2. Not a sentence

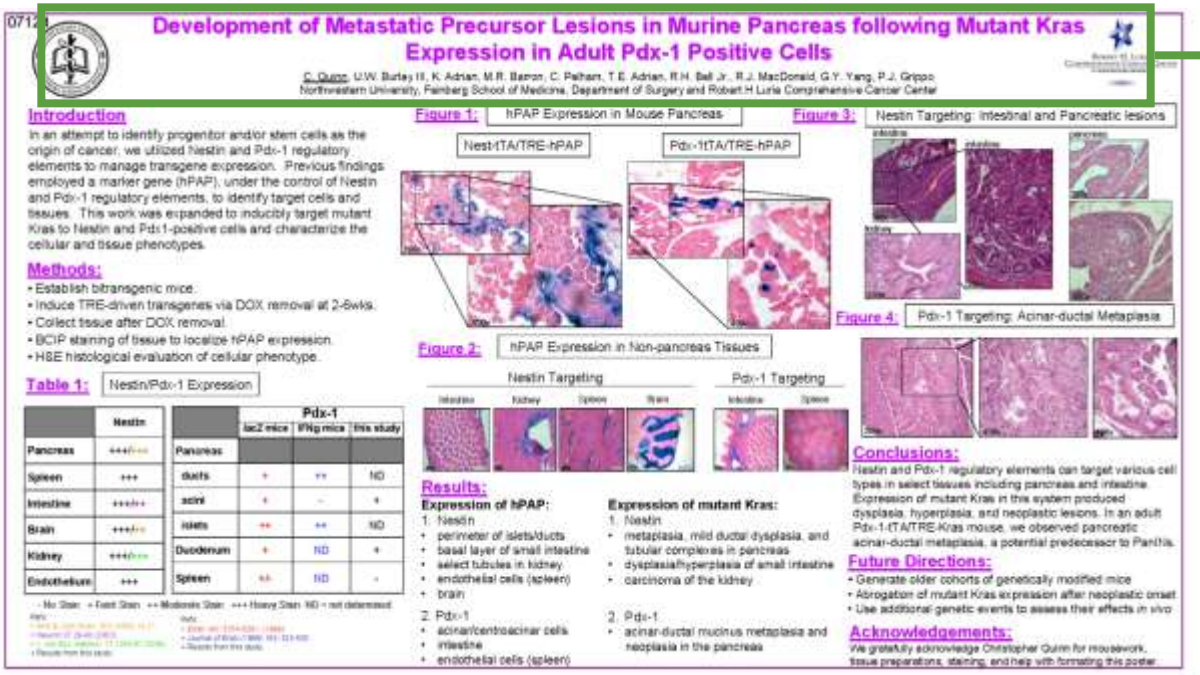
Message title

E2F1 stabilizes p53 and suppresses neovascularization in the ischemic myocardium

1. Key words – active verbs showing a relationship or causality

2. A complete sentence

Now, good poster titles don't necessarily HAVE to be sentences. But they should describe the poster's main point



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	Pdx-1		
	lacZ mice	IPlg mice	this study
Pancreas			
ducts	+	++	ND
acini	+	-	+
islets	++	++	ND
Duodenum	+	ND	+
Spleen	++	ND	-

Figure 1: hPAP Expression in Mouse Pancreas

Nest/rTA/TRE-hPAP | Pdx-1rTA/TRE-hPAP

Figure 2: hPAP Expression in Non-pancreas Tissues

Nestin Targeting | Pdx-1 Targeting

Figure 3: Nestin Targeting: Intestinal and Pancreatic lesions

Figure 4: Pdx-1 Targeting: Acinar-ductal Metaplasia

Results:

Expression of hPAP:

1. Nestin
   - perimeter of islets/ducts
   - basal layer of small intestine
   - select tubules in kidney
   - endothelial cells (spleen)
   - brain
2. Pdx-1
   - acinar/centroacinar cells
   - intestine
   - endothelial cells (spleen)

Expression of mutant Kras:

1. Nestin
   - metaplasia, mild ductal dysplasia, and tubular complexes in pancreas
   - dysplasia/hyperplasia of small intestine
   - carcinoma of the kidney
2. Pdx-1
   - acinar-ductal mucinus metaplasia and neoplasia in the pancreas

Conclusions:

Nestin and Pdx-1 regulatory elements can target various cell types in select tissues including pancreas and intestine. Expression of mutant Kras in this system produced dysplasia, hyperplasia, and neoplastic lesions. In an adult Pdx-1-rTA/TRE-Kras mouse, we observed pancreatic acinar-ductal metaplasia, a potential predecessor to PanIIa.

Future Directions:

- Generate older cohorts of genetically modified mice
- Abrogation of mutant Kras expression after neoplastic onset
- Use additional genetic events to assess their effects *in vivo*

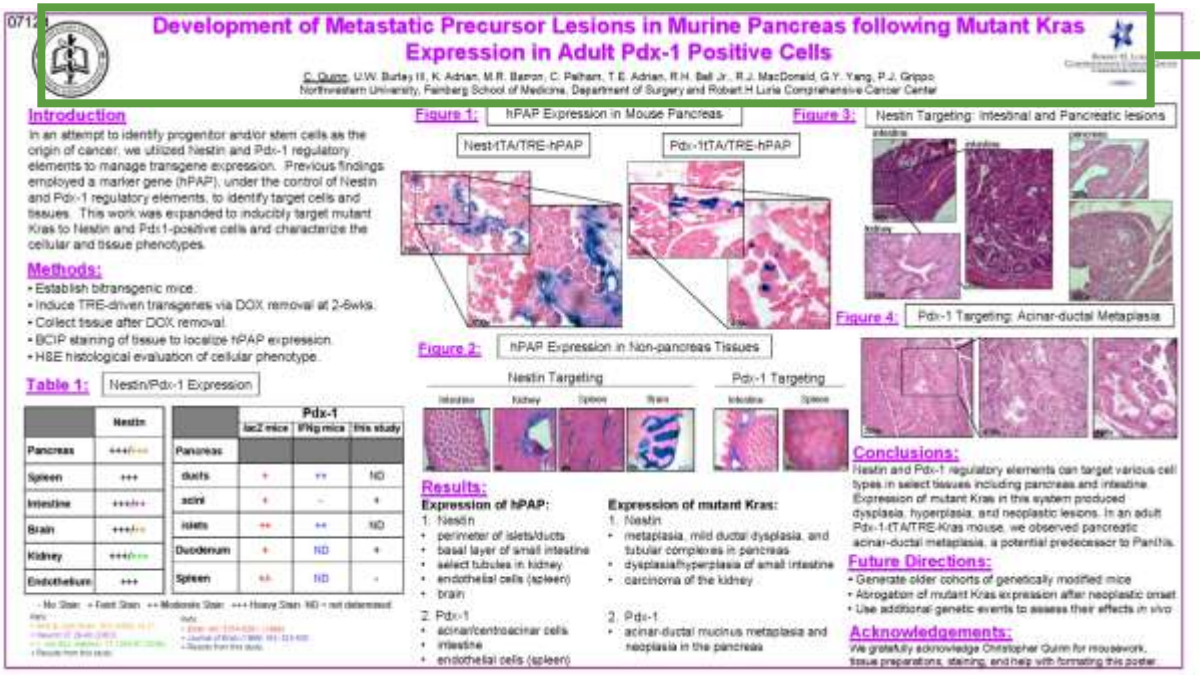
Acknowledgements:

We gratefully acknowledge Christopher Quinn for mousework, tissue preparations, staining, and help with formatting this poster.

Development of Metastatic Precursor Lesions in Murine Pancreas following Mutant Kras Expression in Adult Pdx-1 Positive Cells

With this title we can quickly process the main point of the poster: precursor lesions came about as a result of a specific gene expression

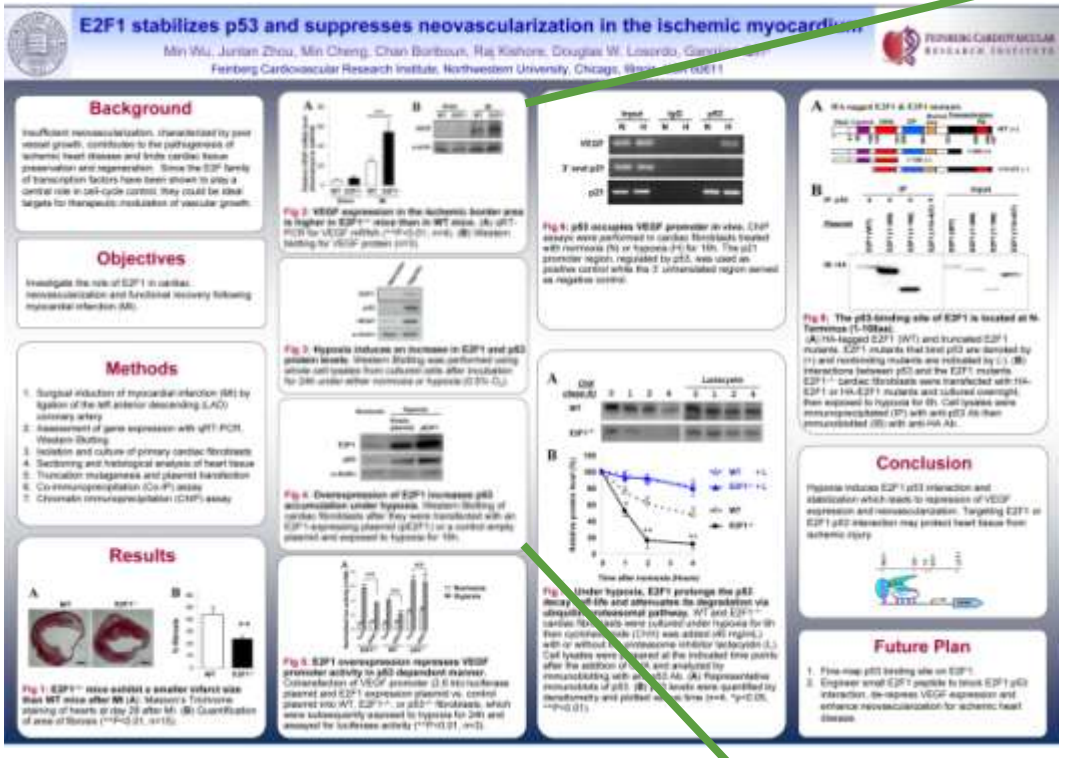
Of course, it is very easy to transform this title into a sentence



Development of Metastatic Precursor Lesions in Murine Pancreas following Mutant Kras Expression in Adult Pdx-1 Positive Cells

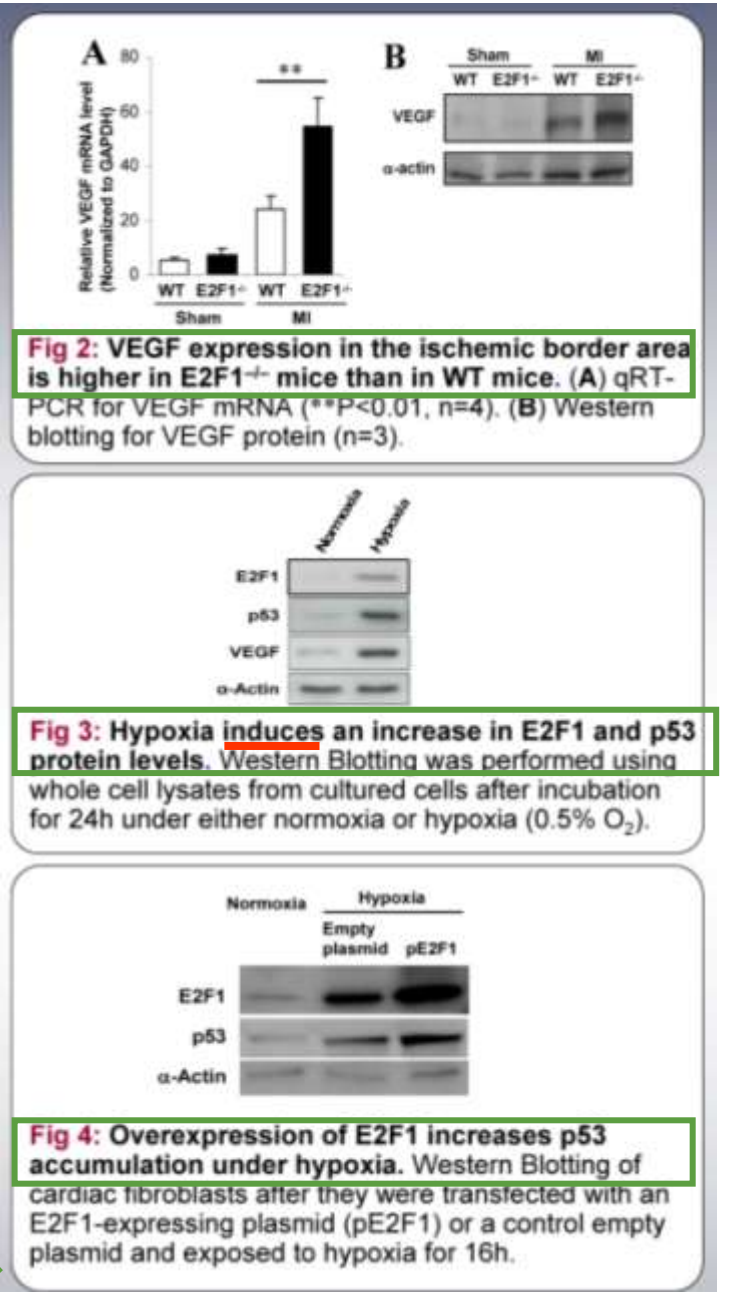
Metastatic Precursor Lesions in Murine Pancreas Developed following Mutant Kras Expression in Adult Pdx-1 Positive Cells

Finally, use message titles in your figures as well – avoid topic titles – and number your figures



Each figure title:

- **is a sentence** that tells us exactly what we should "takeaway" from the figure
- **uses verbs,** showing relationship or causality
- **is numbered** so we know where we are in the "sequence" of the poster's story and for reference



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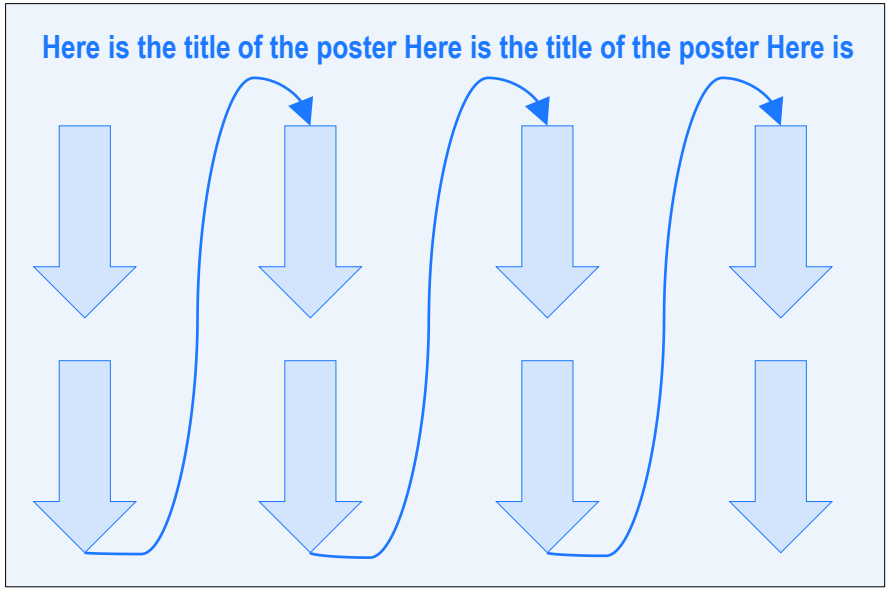
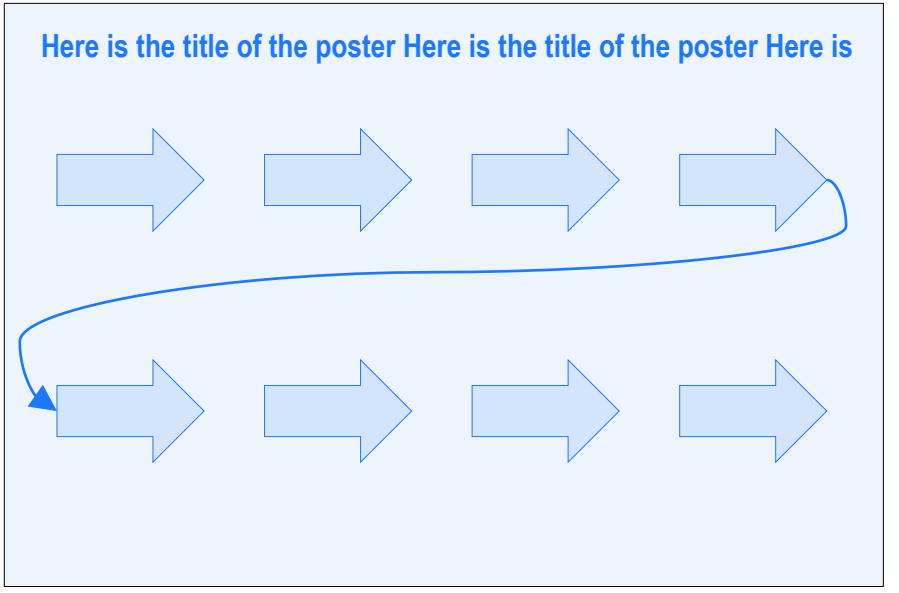
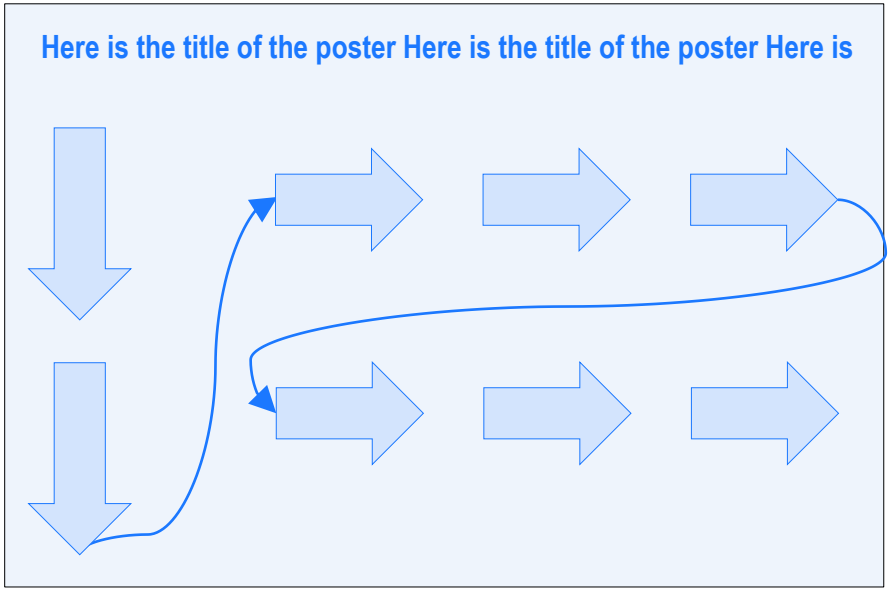
How do you want your viewers to read your poster? Think of the directional “flow” of your poster’s story

Pure Column



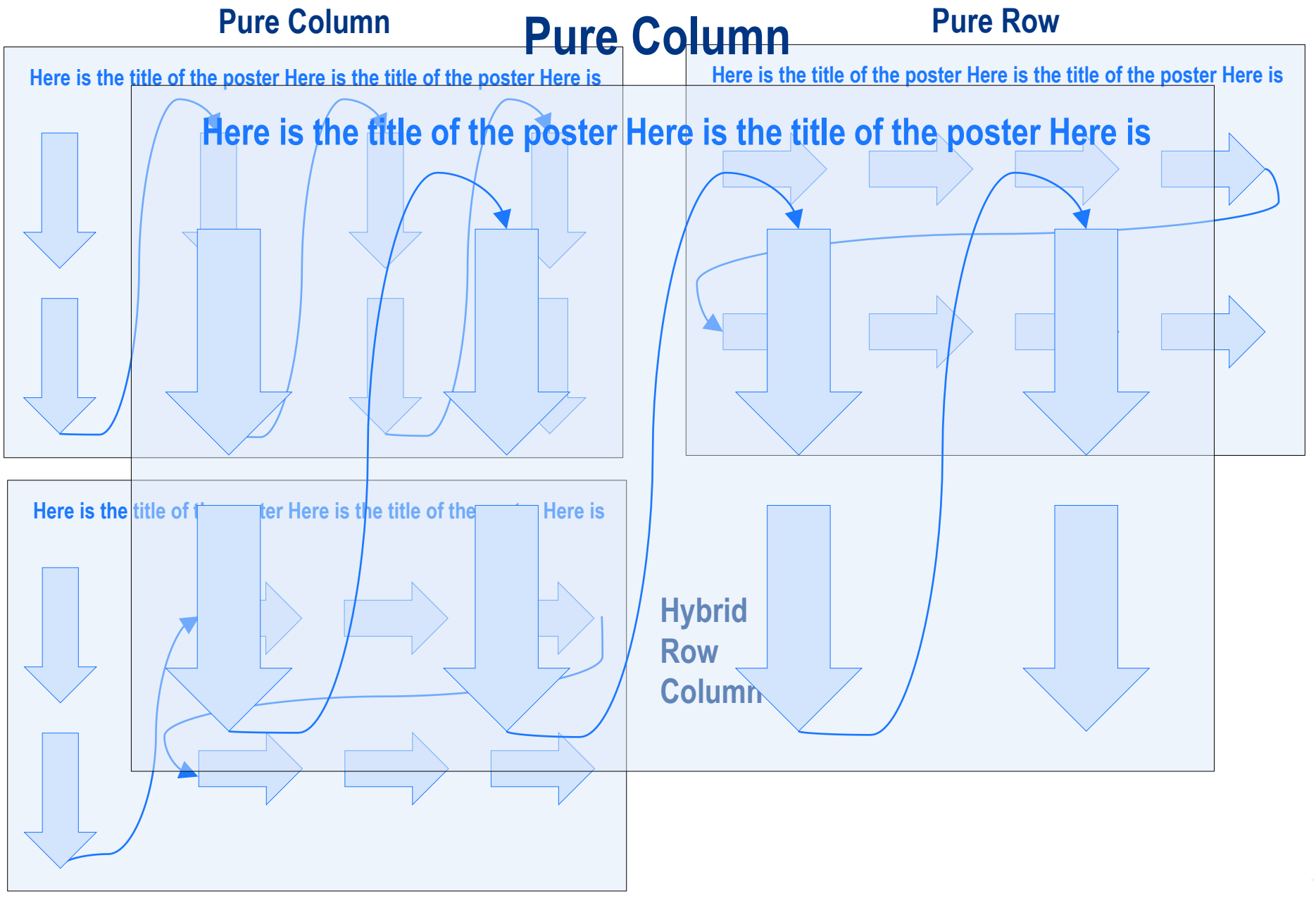
Pure Row





Hybrid
Row
Column

The “Pure Column” is the standard classic approach to poster design



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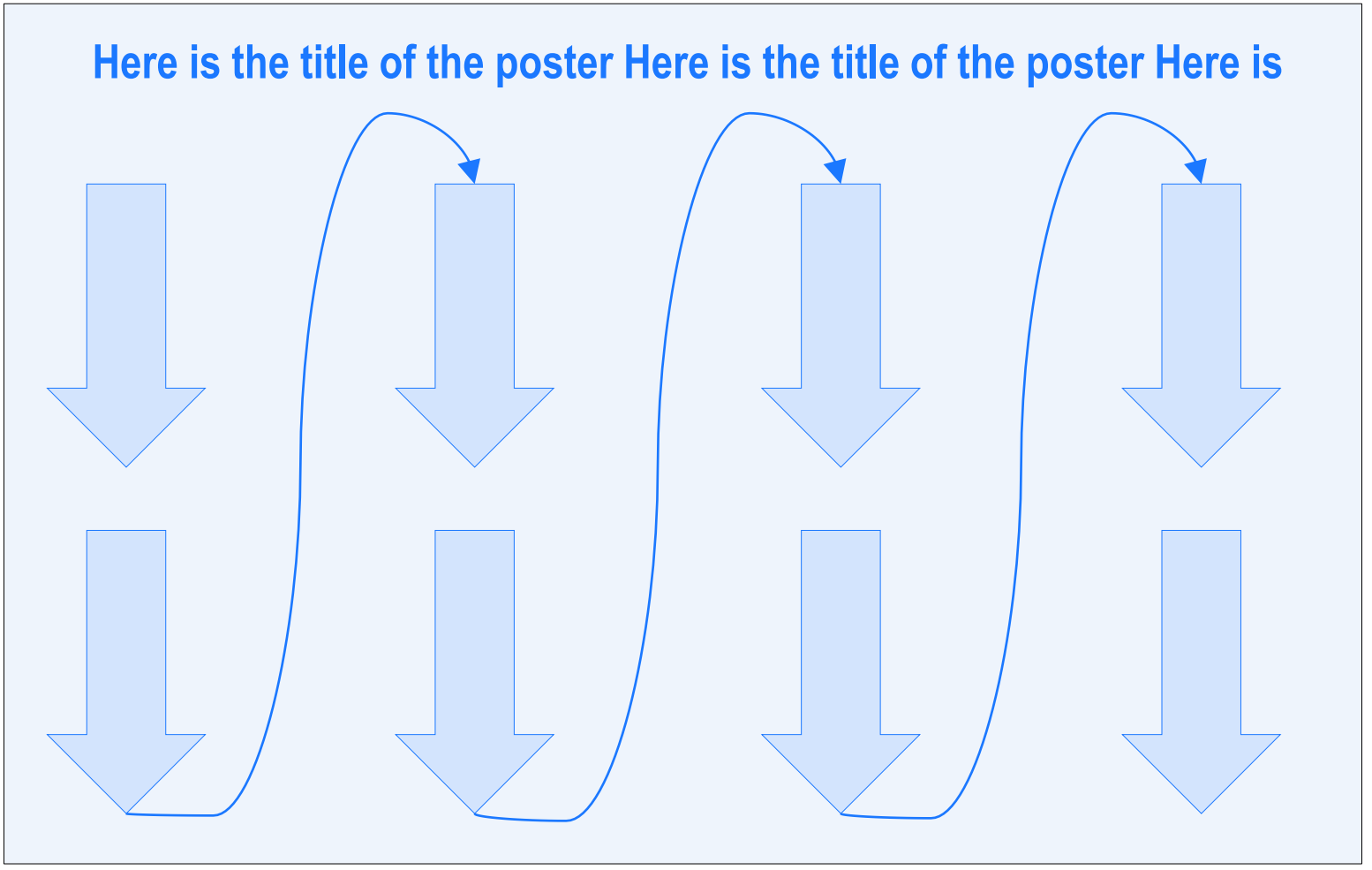
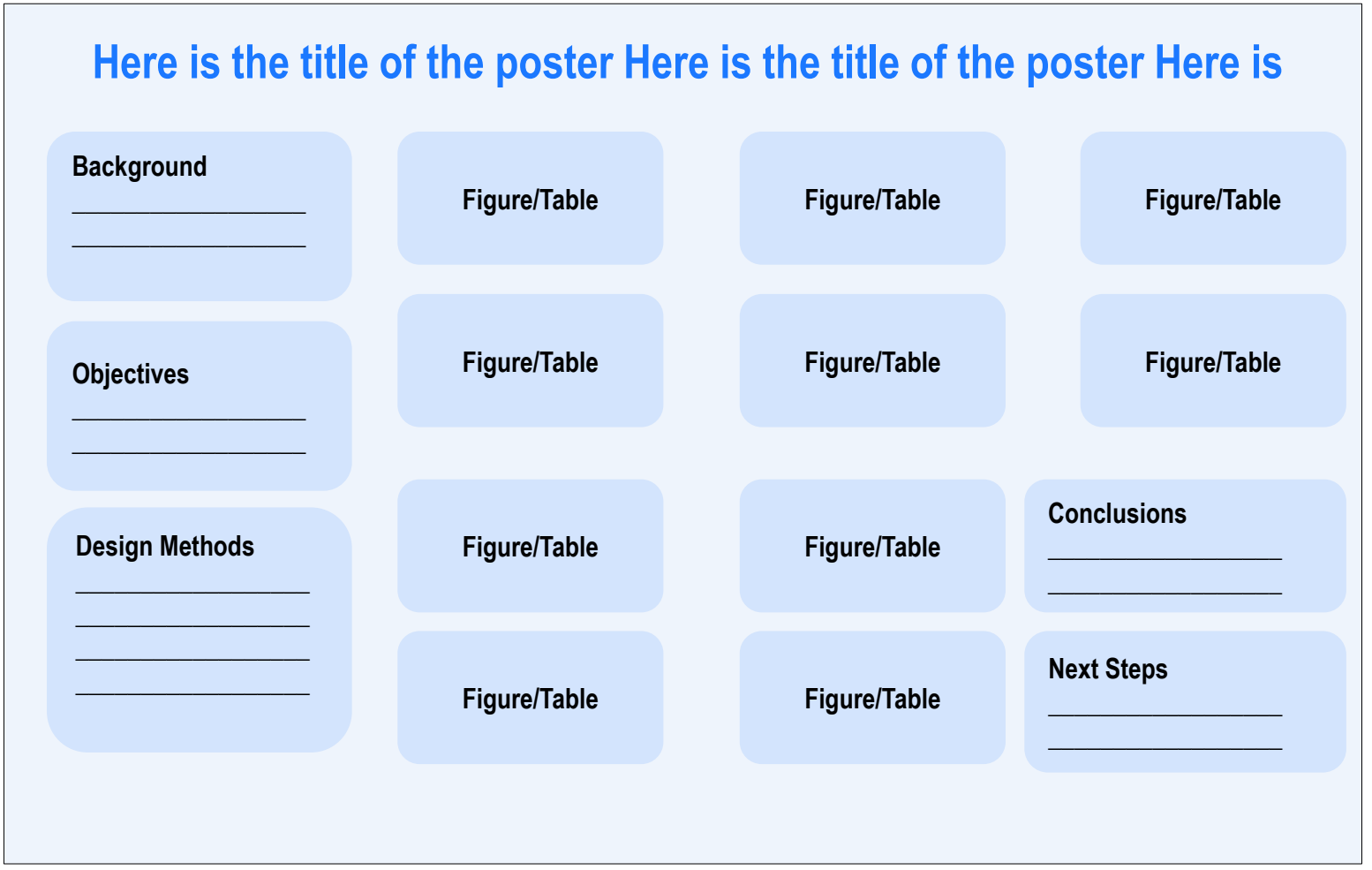


And here are some typical parts of a Pure Column poster, which can be three or four columns



Here's another Pure Column approach that uses the terms "Introduction" and "Hypothesis" among others

Pure Column

Here is the title of the poster Here is the title of the poster Here is

Introduction

_____
_____
_____

Hypothesis

_____
_____
_____

Design/Methods

_____
_____
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_____

Figure/Table

Figure/Table

Figure/Table

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Figure/Table

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Figure/Table

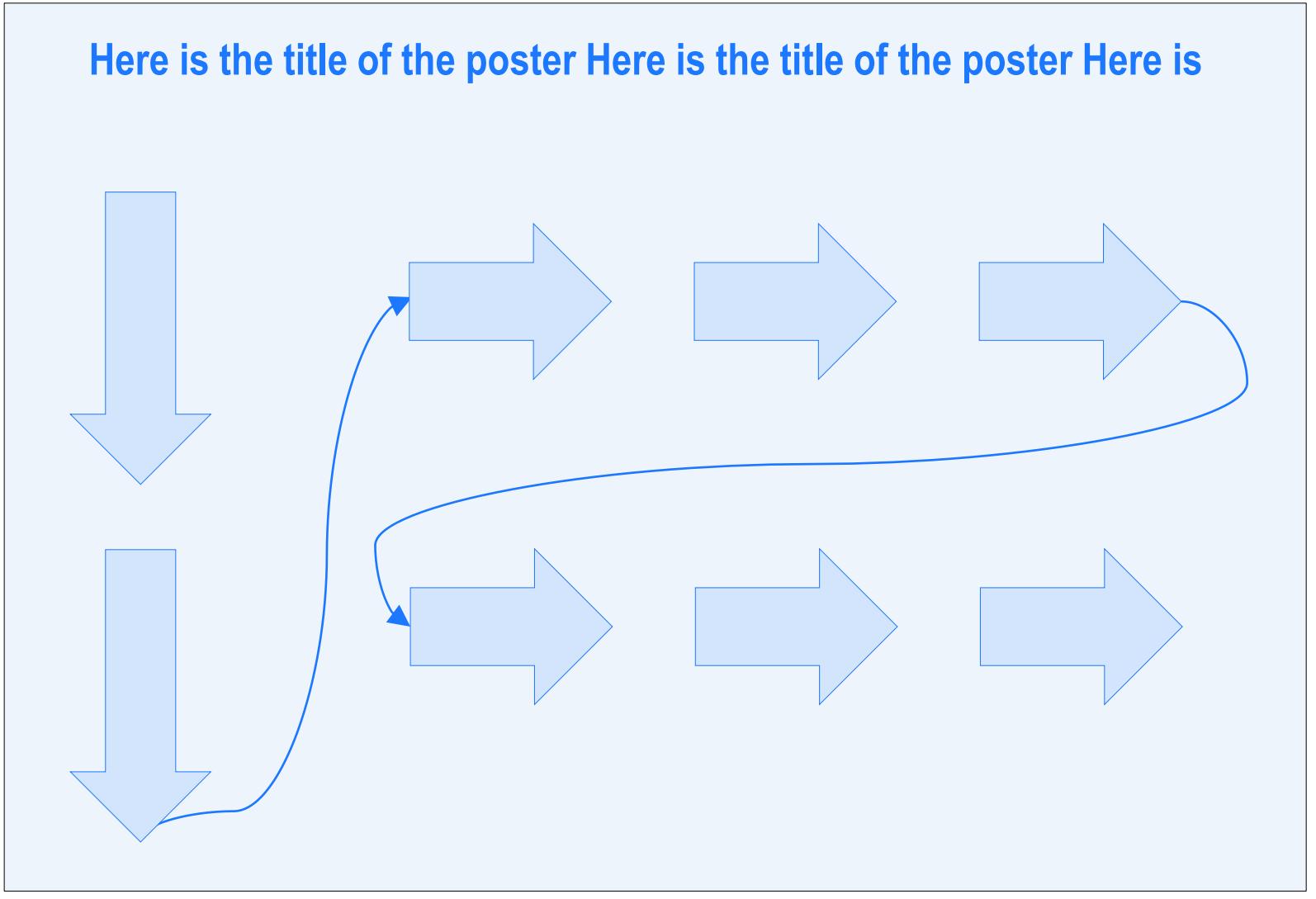
Conclusions

_____
_____

Next Steps

_____
_____

Consider Hybrid Row Column especially if figures reflect a sequential dependent time pattern in experimental results



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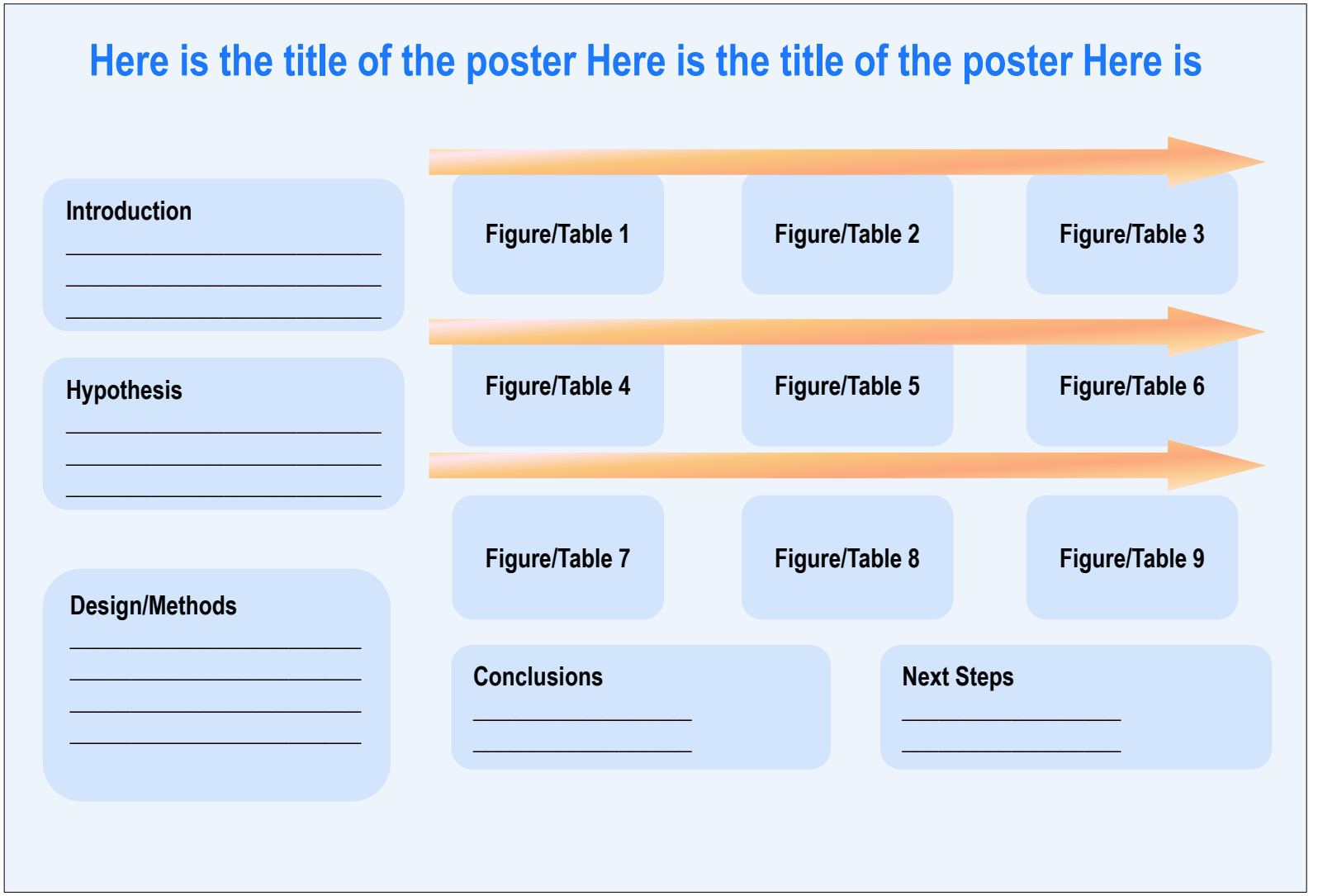
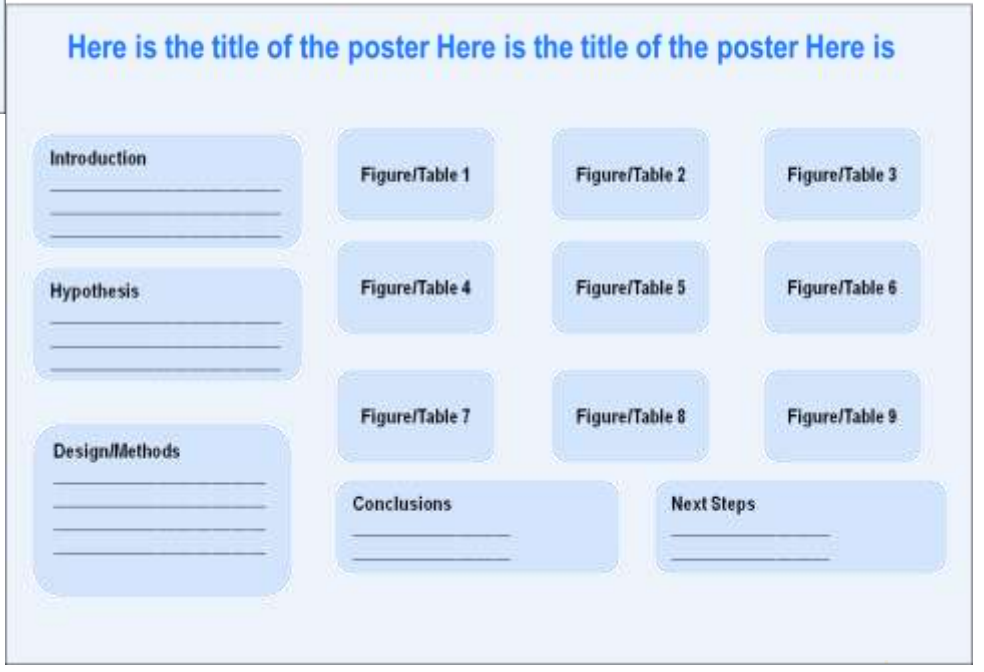


Hybrid Row Column

But note that looking at layouts, a reader STILL may not know how to read it – or have to work to figure out how to read it

Pure Column

Hybrid Row Column

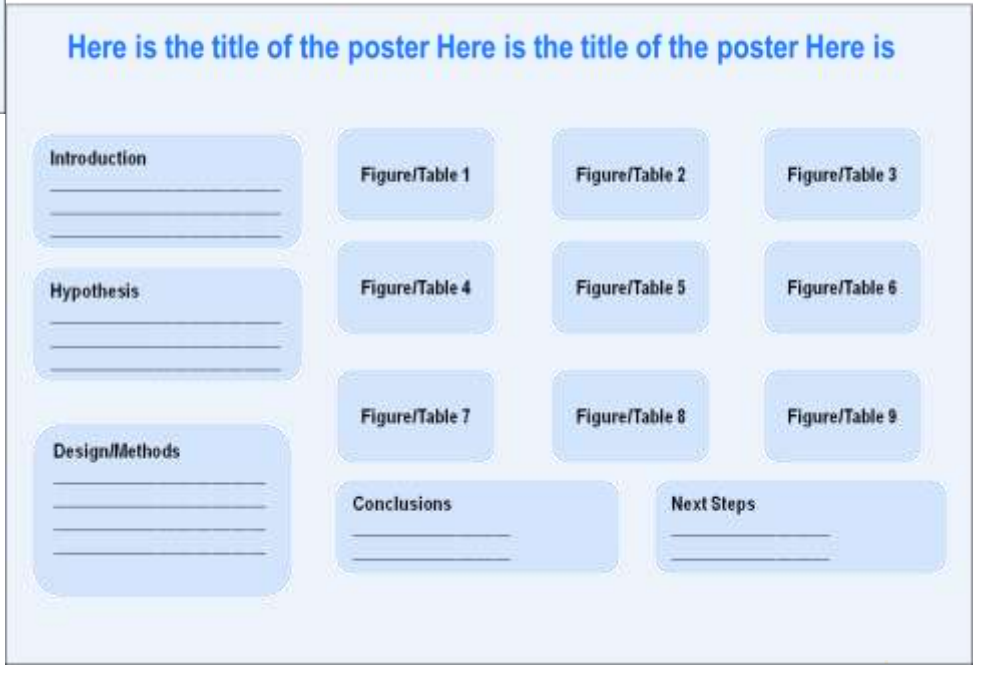


So what's the solution?

But note that looking at layouts, a reader STILL may not know how to read it – or have to work to figure out how to read it

Pure Column

Hybrid Row Column



To assist readers

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Introduction

_____
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Figure/Table

Figure/Table

Figure/Table

Figure/Table

Figure/Table

Figure/Table

Figure/Table

Figure/Table

Figure/Table

Conclusions

_____
_____

Next Steps

_____
_____

To assist readers, number your key sections and figures, create distinguishing headers, and thin rules to separate sections

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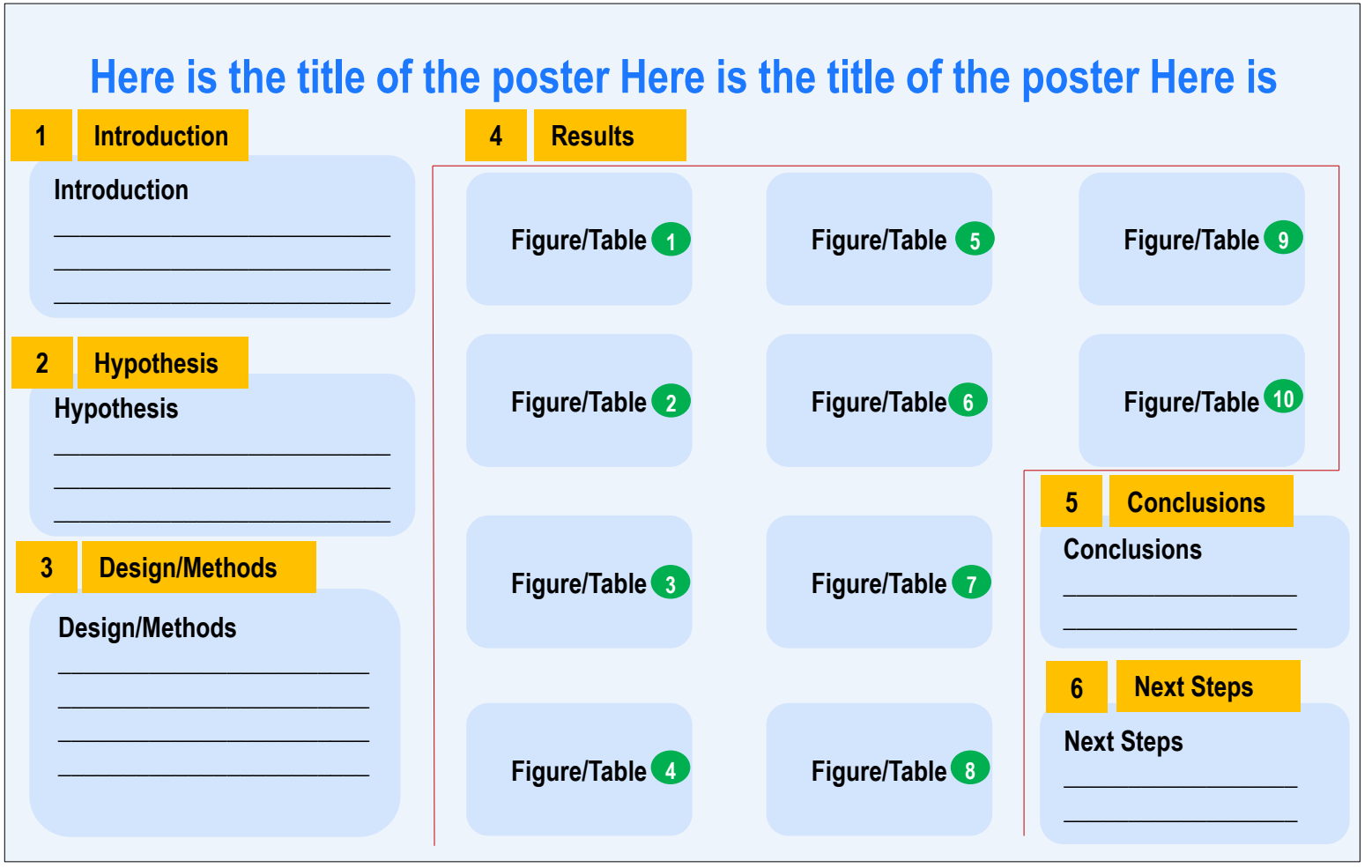
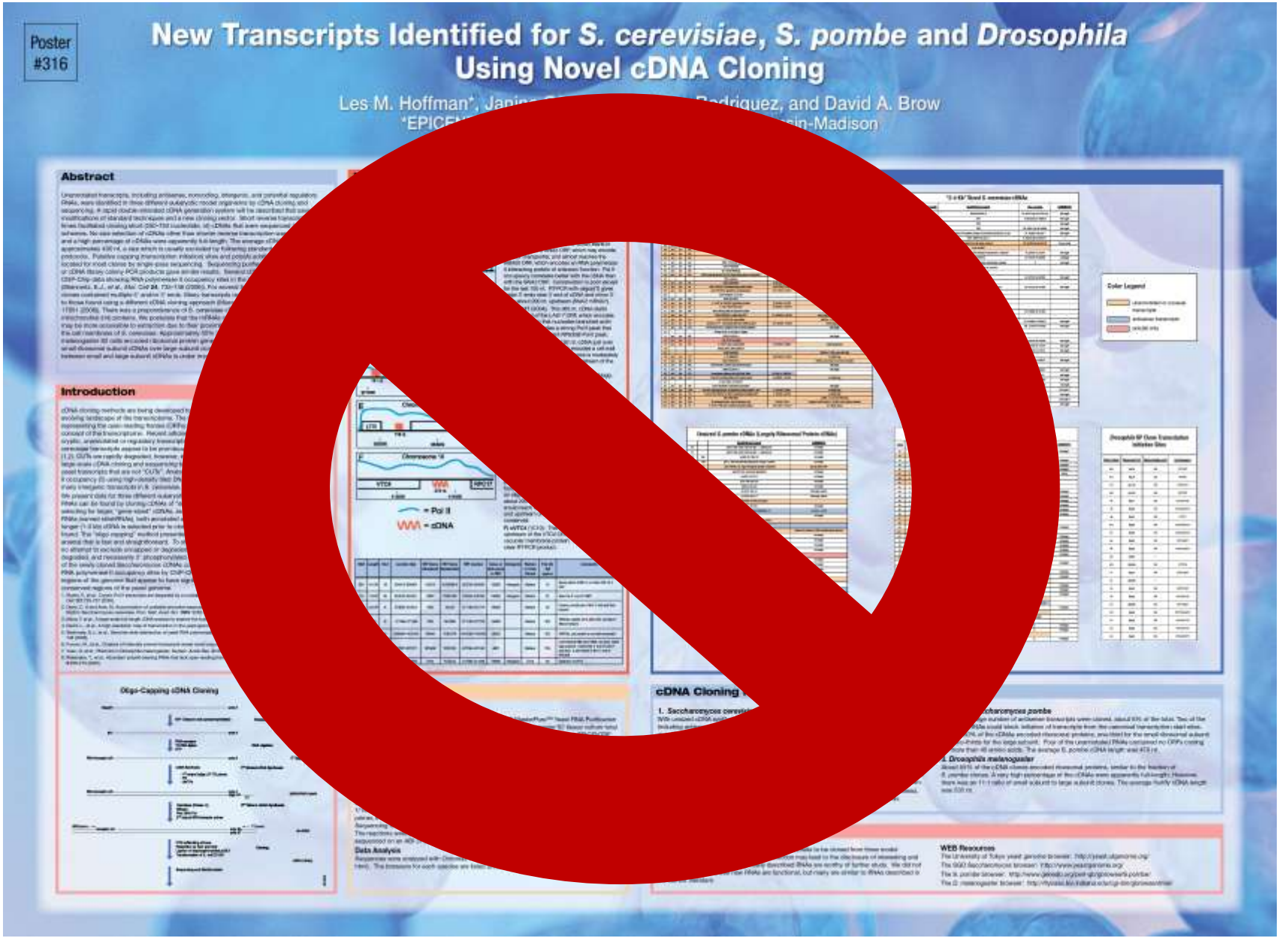
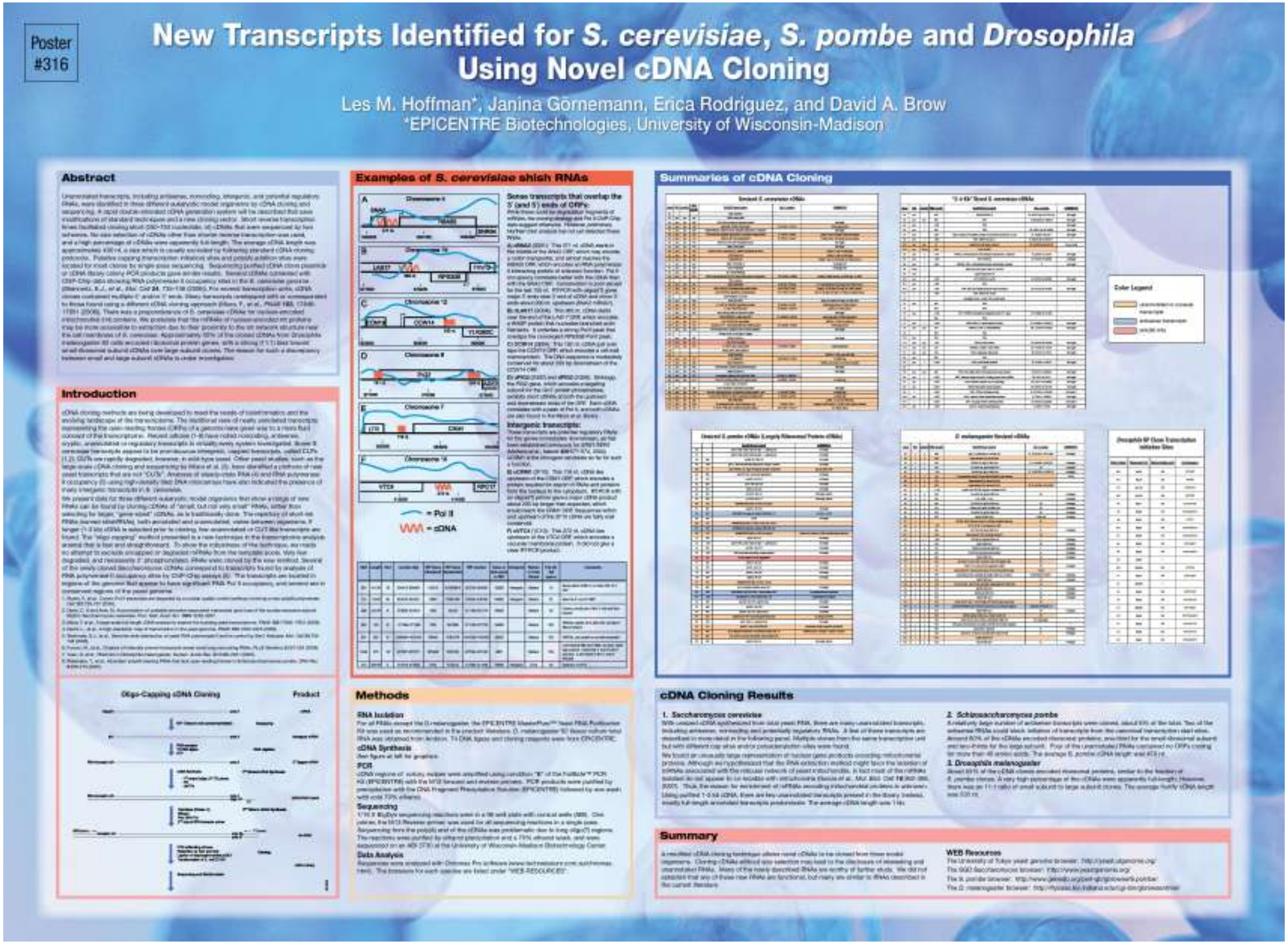


Now, let's look at text. How many posters do you see that look like this?



So what are the flaws here?



Overriding principle: Make your poster "skimmable"

Changes sentences to bullets, phrases

Not:
cDNA cloning methods are being developed to meet the needs of bioinformatics and the evolving landscape of the transcriptome

Instead:
cDNA cloning methods can:
--meet needs of bioinformatics
--enhance understanding of transcriptome

Highlight key categories with boldface

For example, in Methods Section, have a three or four word "Title" of each method that's bolded and then a brief description of the method

Lots of white space

Do a word count – do you have more than 1,000 words? Then think about cutting. Do NOT drop what amounts to a scientific paper onto your poster.

Let your figures tell the story

A good sequence of figures, with explanatory titles, should carry the bulk of your story; keep additional narrative to a minimum

Agenda

1 – Writing Poster Titles & Figure Titles

Writing titles as "takeaways" – i.e., complete thoughts that cue the reader to the meaning of your overall poster and each figure

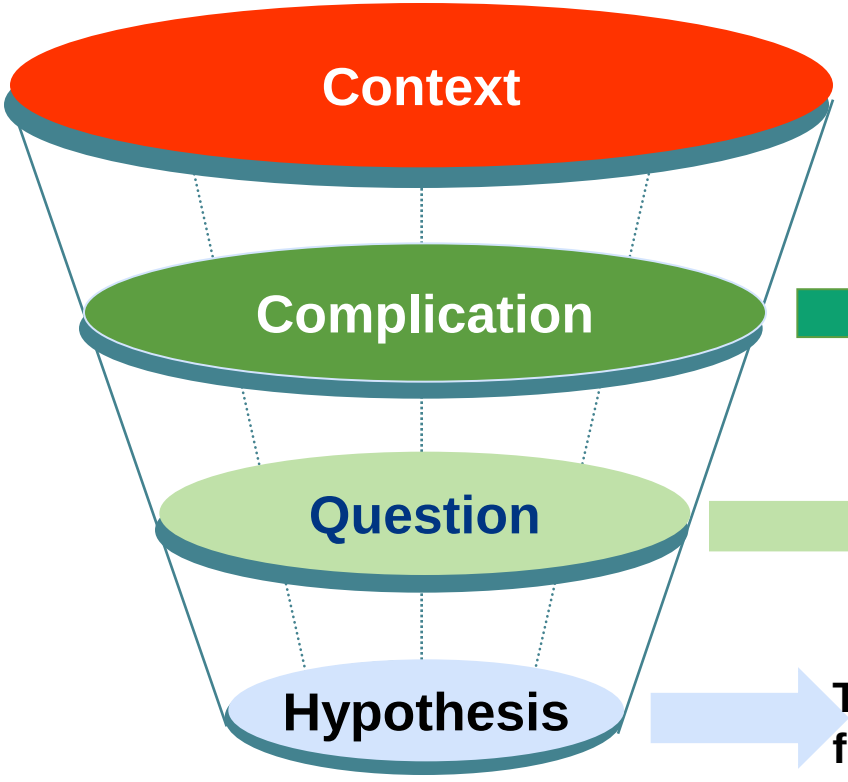
2 – Designing for Readability

Developing discrete sections; eliminating words so your poster is not "text heavy"; cueing the reader how to view your information

3 – Telling the Story

Creating "flow;" organizing your introduction; framing your methods, conclusions, and next steps

To create flow within your poster, you must organized opening sections using the CCQH "funnel" pattern



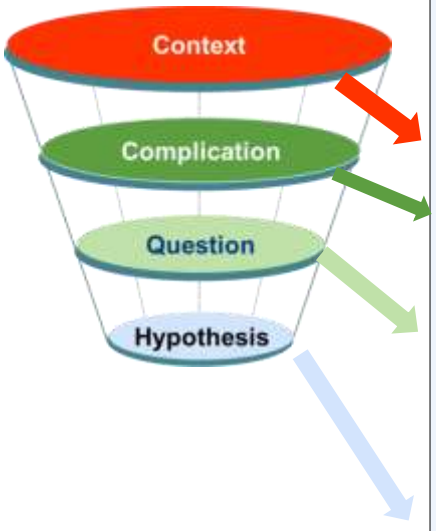
Context gives the big picture—what everyone knows, agrees to

Complication tells us what's unknown or uncertain about the issues raised by the context

The Question raised by the complication should be answerable in a way that will resolve the complication

The Hypothesis is the narrowest point of the funnel – the hypothesis focuses not only the work itself but the presentation

Here's one way you can map the funnel onto a set of poster sections



Here is the title of the poster Here is the title of the poster Here is

1 Introduction

Introduction

2 Research Question

3 Hypothesis

4 Design/Methods

5 Results

Figure/Table 1 | Figure/Table 5 | Figure/Table 9

Figure/Table 2 | Figure/Table 6 | Figure/Table 10

Figure/Table 3 | Figure/Table 7

Figure/Table 4 | Figure/Table 8

6 Conclusions

Conclusions

7 Next Steps

Next Steps

Let's look at an example poster. Does it follow our principles?

E2F1 stabilizes p53 and suppresses neovascularization in the ischemic myocardium

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Background

Insufficient neovascularization, characterized by poor vessel growth, contributes to the pathogenesis of ischemic heart disease and limits cardiac tissue preservation and regeneration. Since the E2F family of transcription factors have been shown to play a central role in cell-cycle control, they could be ideal targets for therapeutic modulation of vascular growth.

Objectives

Investigate the role of E2F1 in cardiac neovascularization and functional recovery following myocardial infarction (MI).

Methods

1. Surgical induction of myocardial infarction (MI) by ligation of the left anterior descending (LAD) coronary artery
2. Assessment of gene expression with qRT-PCR, Western Blotting
3. Isolation and culture of primary cardiac fibroblasts
4. Sectioning and histological analysis of heart tissue
5. Truncation mutagenesis and plasmid transfection
6. Co-Immunoprecipitation (Co-IP) assay
7. Chromatin Immunoprecipitation (ChIP) assay

Results

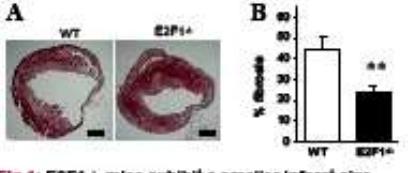


Fig 1: E2F1$^{-/-}$ mice exhibit a smaller infarct size than WT mice after MI (A): Masson's Trichrome staining of hearts at day 28 after MI. (B) Quantification of area of fibrosis (**P<0.01, n=15).

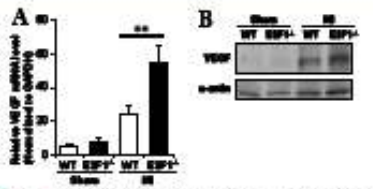


Fig 2: VEGF expression in the ischemic border area is higher in E2F1$^{-/-}$ mice than in WT mice. (A) qRT-PCR for VEGF mRNA (**P<0.01, n=4). (B) Western blotting for VEGF protein (n=3).

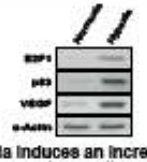


Fig 3: Hypoxia induces an increase in E2F1 and p53 protein levels. Western Blotting was performed using whole cell lysates from cultured cells after incubation for 24h under either normoxia or hypoxia (0.5% O_2).

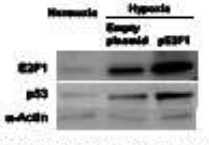


Fig 4: Overexpression of E2F1 increases p53 accumulation under hypoxia. Western Blotting of cardiac fibroblasts after they were transfected with an E2F1-expressing plasmid (pE2F1) or a control empty plasmid and exposed to hypoxia for 16h.

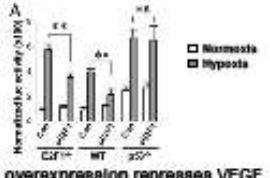


Fig 5: E2F1 overexpression represses VEGF promoter activity in p53 dependent manner. Cotransfection of VEGF promoter (2.6 kb)-luciferase plasmid and E2F1 expression plasmid vs. control plasmid into WT, E2F1$^{-/-}$, or p53$^{-/-}$ fibroblasts, which were subsequently exposed to hypoxia for 24h and assayed for luciferase activity (**P<0.01, n=3).

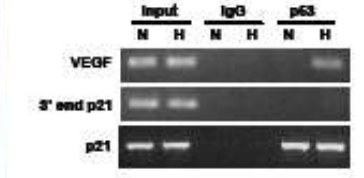


Fig 6: p53 occupies VEGF promoter *in vivo*. ChIP assays were performed in cardiac fibroblasts treated with normoxia (N) or hypoxia (H) for 16h. The p21 promoter region, regulated by p53, was used as positive control while the 3' untranslated region served as negative control.

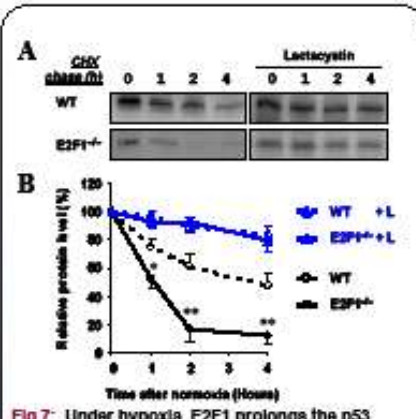


Fig 7: Under hypoxia, E2F1 prolongs the p53 decay half-life and attenuates its degradation via ubiquitin-proteasomal pathway. WT and E2F1$^{-/-}$ cardiac fibroblasts were cultured under hypoxia for 6h then cycloheximide (*CHX*) was added (40 mg/mL) with or without the proteasome inhibitor lactacystin (L). Cell lysates were prepared at the indicated time points after the addition of CHX and analyzed by immunoblotting with anti-p53 Ab. (A) Representative immunoblots of p53. (B) p53 levels were quantified by densitometry and plotted versus time (n=4, *p<0.05, **P<0.01).

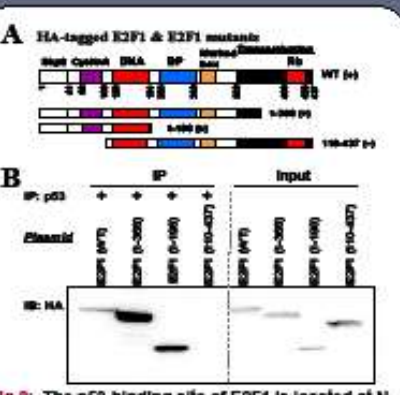
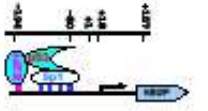


Fig 8: The p53-binding site of E2F1 is located at N-Terminus (1-108aa).
(A) HA-tagged E2F1 (WT) and truncated E2F1 mutants. E2F1 mutants that bind p53 are denoted by (+) and nonbinding mutants are indicated by (-). (B) Interactions between p53 and the E2F1 mutants. E2F1$^{-/-}$ cardiac fibroblasts were transfected with HA-E2F1 or HA-E2F1 mutants and cultured overnight, then exposed to hypoxia for 6h. Cell lysates were immunoprecipitated (IP) with anti-p53 Ab then immunoblotted (IB) with anti-HA Ab.

Conclusion

Hypoxia induces E2F1:p53 interaction and stabilization which leads to repression of VEGF expression and neovascularization. Targeting E2F1 or E2F1:p53 interaction may protect heart tissue from ischemic injury.



Future Plan

1. Fine-map p53 binding site on E2F1.
2. Engineer small E2F1 peptide to block E2F1:p53 interaction, de-repress VEGF expression and enhance neovascularization for ischemic heart disease.