

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

07124 **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. Peilham, 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 induced 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 Pdx-1-positive cells and characterize the cellular and tissue phenotypes.

Methods:

- Establish btransgenic 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	Pdx-1		
		lacZ mice	IFNg mice	this study
Pancreas	+++/-++	Pancreas		
Spleen	+++	Spleen	-	ND
Intestine	+++/-++	Intestine	-	+
Brain	+++/-++	Brain	++	ND
Kidney	+++/-++	Duodenum	ND	+
Endothelium	+++	Spleen	-/-	ND

- No Stain + Faint Stain ++ Moderate Stain +++ Heavy Stain ND = not determined
 Refs:
 • *Cell* 133(2006):14-21
 • *Am J Pathol* 163(2003):140-150
 • *J Clin Invest* 113(2004):1025-1035
 • *Results from this study*
 • *Diabetes* 149(2004):1188-1194
 • *Journal of Virology* 78(2004):163-170
 • *Results from this study*

Figure 1: hPAP Expression in Mouse Pancreas Figure 3: Nestin Targeting: Intestinal and Pancreatic lesions

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

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 has been shown to play a central role in cell-cycle control, they could be ideal targets for therapeutic modulation of vascular growth.

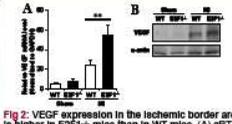


Fig 2: VEGF expression in the ischemic border areas is higher in E2F1^{-/-} mice than in WT mice. (A) qRT-PCR for VEGF mRNA (**P<0.01, n=4). (B) Western blotting for VEGF protein (n=3).

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

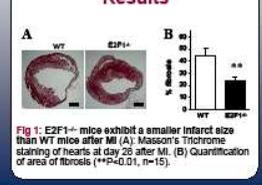


Fig 5: E2F1^{-/-} mice exhibit a smaller infarct size than WT mice after MI (A). Masson's Trichrome staining of hearts at day 28 after MI. (B) Quantification of area of fibrosis (**P<0.01, n=15).

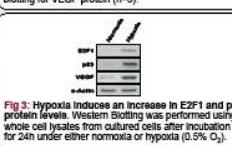


Fig 3: Hypoxia induces an increase in E2F1 and p53 protein levels. Western blotting was performed using whole cell lysates from cultured cells after incubation for 24h under either normoxia or hypoxia (0.5% O₂).

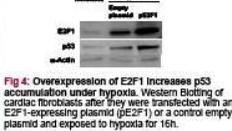


Fig 4: Overexpression of E2F1 increases p53 accumulation under hypoxia. Western blotting of cardiac fibroblasts after they were transfected with an E2F1-expressing plasmid (E2F1) or a control empty plasmid and exposed to hypoxia for 16h.

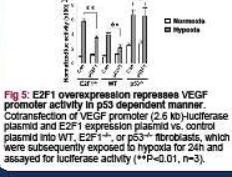


Fig 5: E2F1 overexpression represses VEGF promoter activity in p53 dependent manner. Cotransfection of VEGF promoter (2.6 kb)-luciferase plasmid and E2F1 expression plasmid vs. control plasmid into WT, E2F1^{-/-}, or p53^{-/-} fibroblasts, which were subsequently exposed to hypoxia for 24h and assayed 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?
 Single stranded DNA or RNA
 1-1000 nucleotides
 Natural
 Not strictly to potassium ions
 Easy and efficient synthesis
 Low cost (sequencing)
 Requires no chemistry expertise

Prostate stem cell antigen (PSCA) is a rational target for pancreatic cancer therapy.
 PSCA is overexpressed in prostate cancer
 High level expression in pancreatic cancer cells
 PSCA is a novel target for pancreatic cancer therapy
 PSCA is overexpressed in pancreatic cancer cells
 PSCA is a novel 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

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

Results
 The percent of the library to PSCA-expressing cells was increased after 10 cycles of selection. The enrichment of non-specific cell binding was reduced after 10 cycles of selection.

Summary
 Cell-based selection requires stringent "Tuggle" 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 ("Tuggle" selection) may be better than either technique alone.

"Tuggle" selection strategy
 In vitro selection
 Cell-based selection

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

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

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

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

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 lineage-specific gene expression. Previous findings employed a similar strategy to Nestin under the control of Nestin and Pdx-1 regulatory elements, to identify target cells and tissues. This work was expanded to include target mutant Kras to Nestin and Pdx-1-positive cells and characterize the cellular and tissue phenotypes.

Methods
• Establish intragenetic cross.
• Induce TRS-driven transgenes via DOX removal in pancreas.
• Collect tissue after DOX removal.
• IHC/P staining of tissue to localize NPAP expression.
• H&E histological evaluation of cellular phenotypes.

Figure 1: NPAP Expression in Mouse Pancreas
Figure 2: NPAP Expression in Non-pancreas Tissues
Figure 3: Pdx-1 Targeting: Acinar-driven Metastasis
Figure 4: Nestin Targeting: Intestinal and Pancreatic Lesions

Table 1: Nestin/Pdx-1 Expression

Gene	Nestin	Pdx-1
Pancreas	+++	+++
Small Intestine	+++	++
Large Intestine	+++	++
Brain	+++	++
Liver	+++	++
Spleen	+++	++
Testis	+++	++
Uterus	+++	++
Bladder	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++
Stomach	+++	++
Adipose	+++	++
Colon	+++	++
Prostate	+++	++
Thyroid	+++	++
Salivary Gland	+++	++
Adipose	+++	++
Heart	+++	++

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

0712

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

S. Duggo, U.W. Burley II, K. Adrian, M.R. Baron, C. Pihani, 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 Pdx-1-positive cells and characterize the cellular and tissue phenotypes.

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

Table 1: Nestin/Pdx-1 Expression

	Nestin		Pdx-1	
	lacZ mice	IFNg mice	lacZ mice	IFNg mice
Pancreas	+++	+++	+++	+++
Spleen	+++	+++	+++	+++
Intestine	+++	+++	+++	+++
Brain	+++	+++	+++	+++
Kidney	+++	+++	+++	+++
Endothelium	+++	+++	+++	+++

Figure 1: hPAP Expression in Mouse Pancreas
Nestin-TA/TRE-hPAP, Pdx-1-TA/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 mucinous 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-TA/TRE-Kras mouse, we observed pancreatic acinar-ductal metaplasia, a potential predecessor to PanINs.

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 rework, 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

0712

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

S. Duggo, U.W. Burley II, K. Adrian, M.R. Baron, C. Pihani, 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 Pdx-1-positive cells and characterize the cellular and tissue phenotypes.

Methods:
• Establish bitergenic 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		Pdx-1	
	lacZ mice	hPAP mice	lacZ mice	hPAP mice
Pancreas	+++	+++	+	++
Spleen	+++	+++	+	+
Intestine	+++	+++	+	+
Brain	+++	+++	+	+
Kidney	+++	+++	+	+
Endothelium	+++	+++	+	+

Legend: - No Stain; + Faint Stain; ++ Moderate Stain; +++ Heavy Stain; ND = not determined

Figure 1: hPAP Expression in Mouse Pancreas
Nestin1TA/TRE-hPAP, Pdx-1TA/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 mucinous 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-TA/TRE-Kras mouse, we observed pancreatic acinar-ductal metaplasia, a potential predecessor to PanINs.

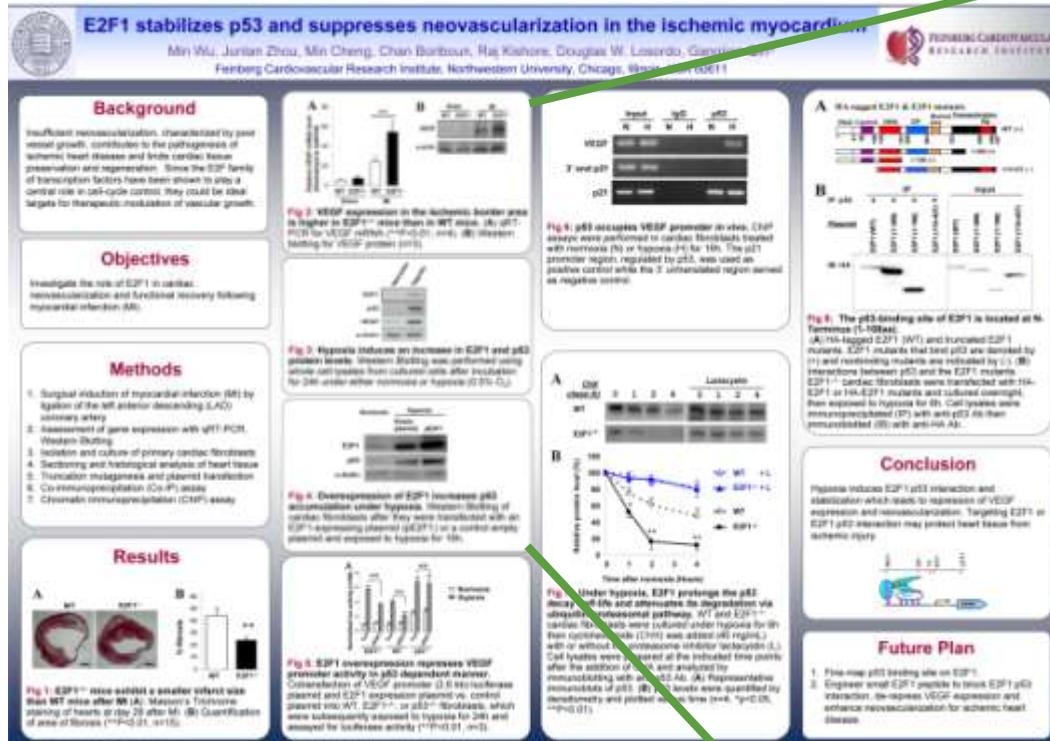
Future Directions:
• Generate older cohorts of genetically modified mice
• Abrogation of mutant Kras expression after neoplastic onset
• Use additional genetic events to assess their effects in vivo

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

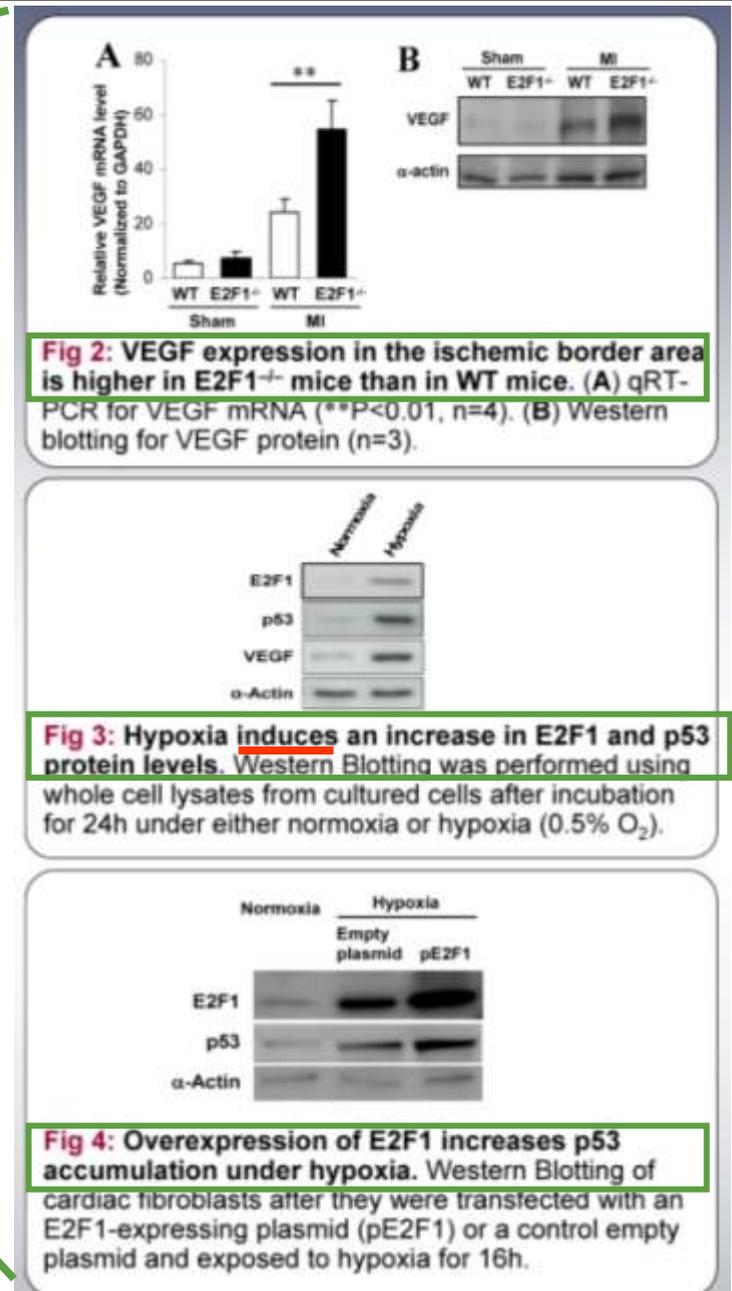
Metastatic Precursor Lesions in Murine Pancreas Developed following Mutant Kras Expression in Adult Pdx-1 Positive Cells

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



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

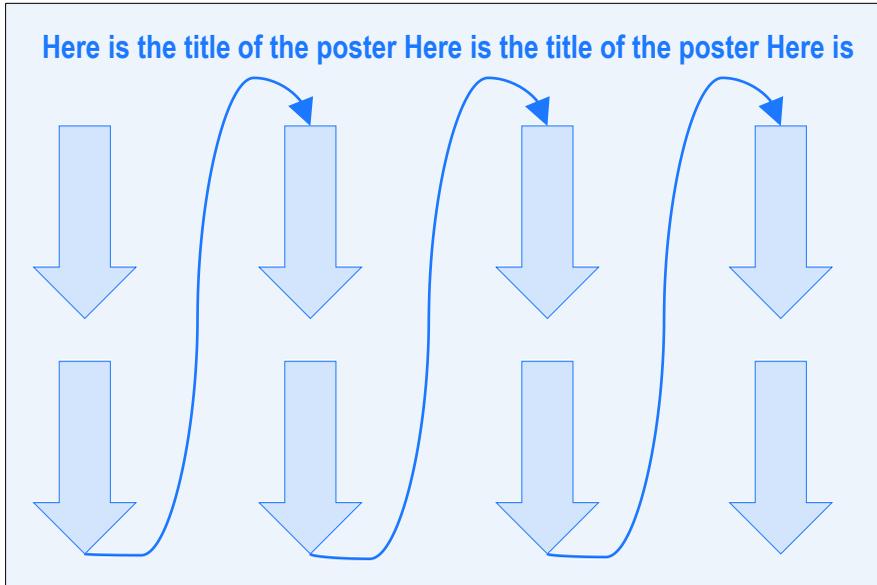
Developing discrete sections; eliminating words so your poster is not “text heavy”; cueing the reader how to view your information

3 – Telling the Story

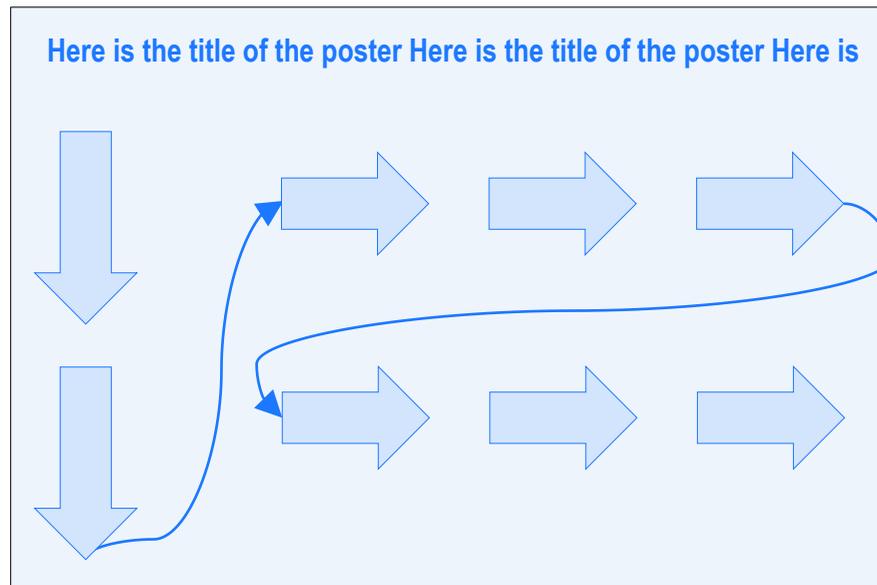
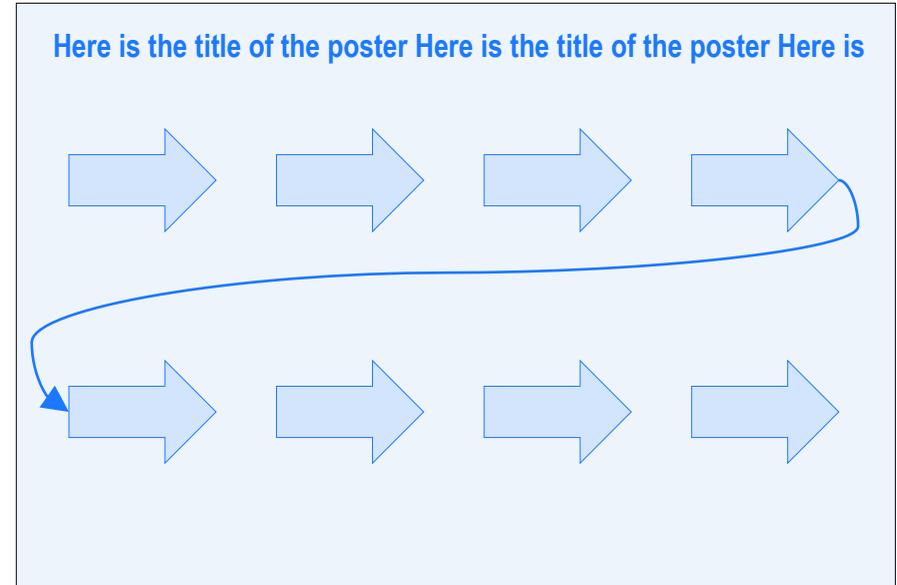
Creating “flow;” organizing your introduction; framing your methods, conclusions, and next steps

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

Pure Column



Pure Row



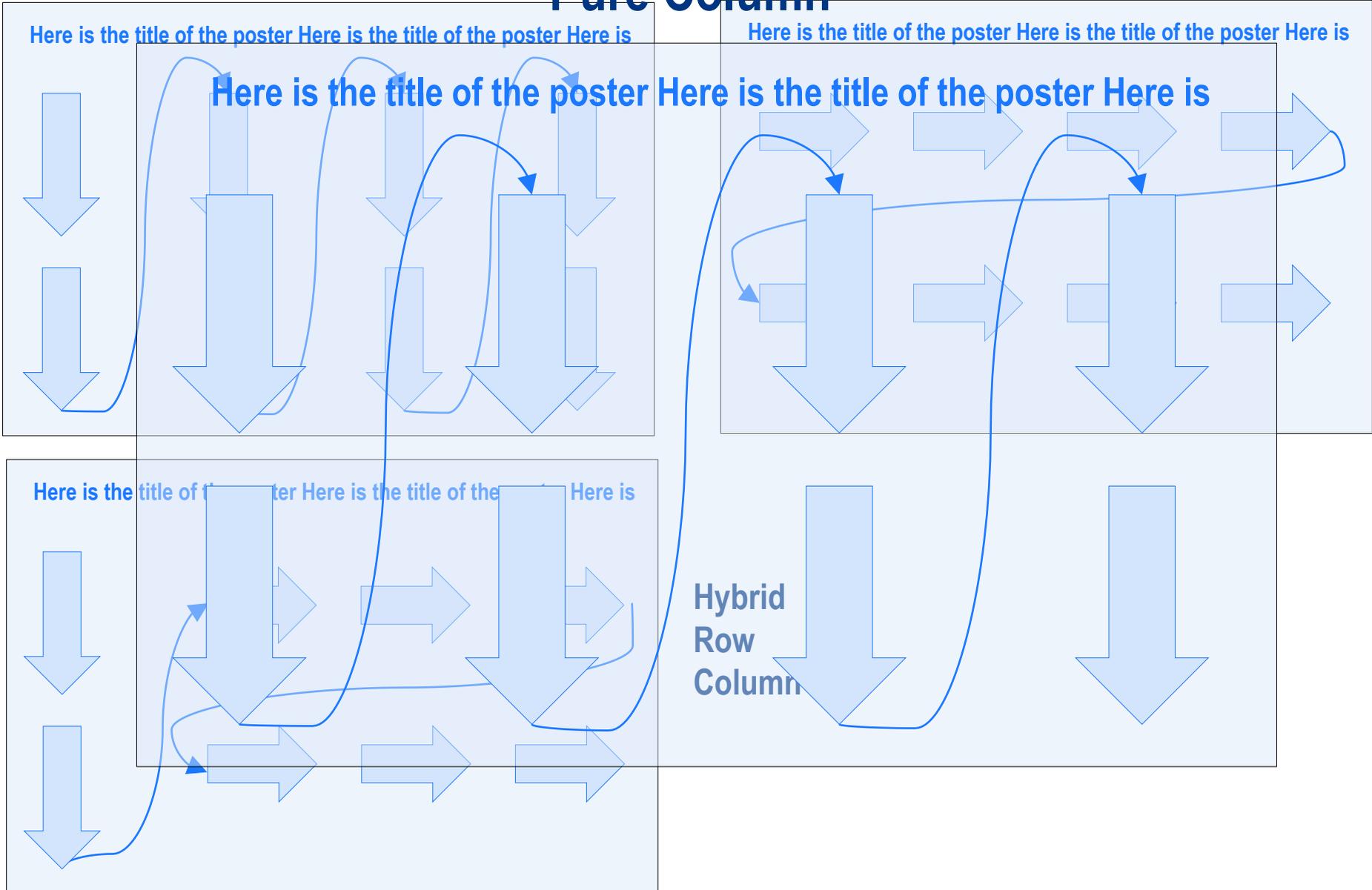
Hybrid
Row
Column

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

Pure Column

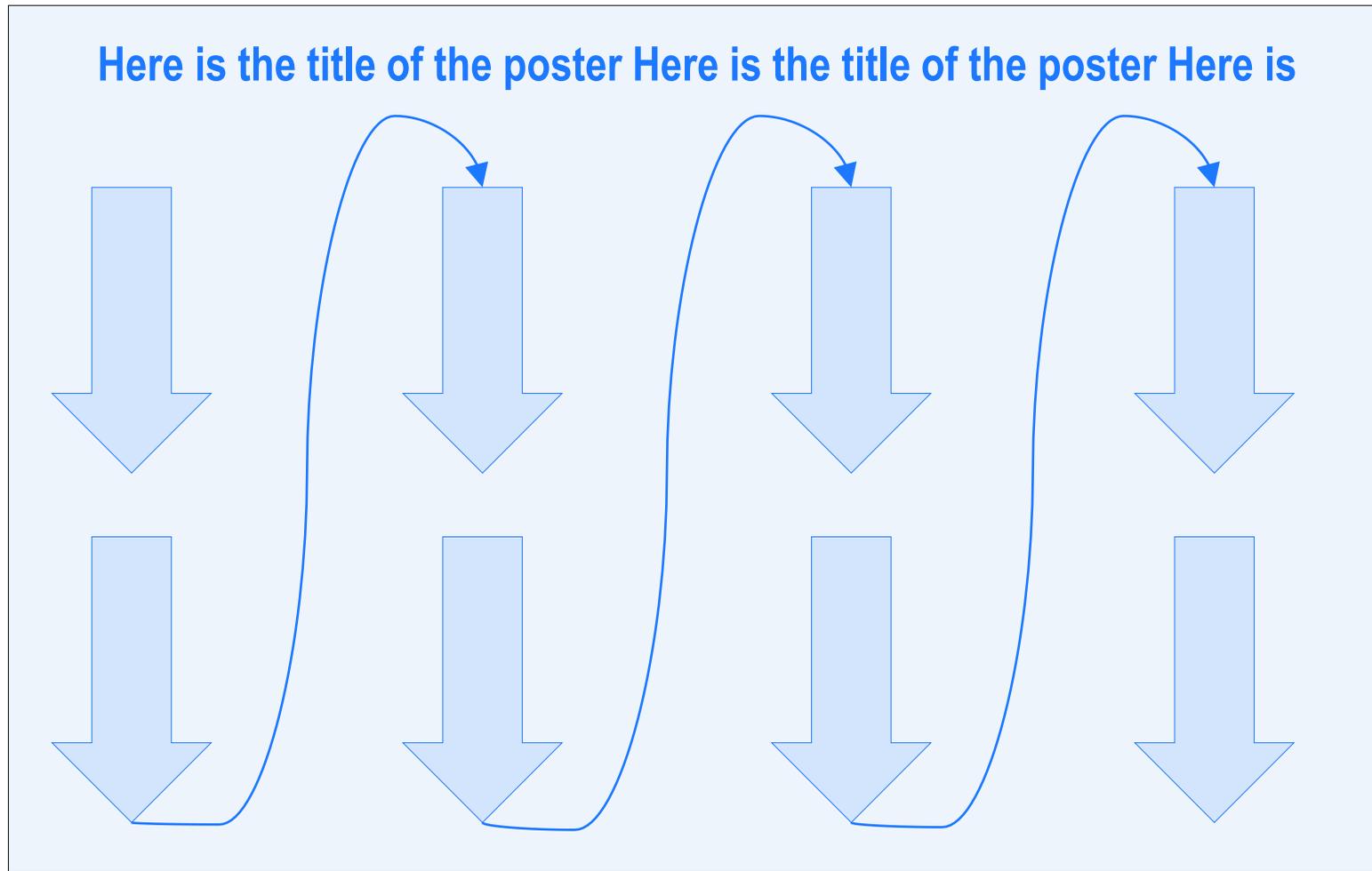
Pure Column

Pure Row



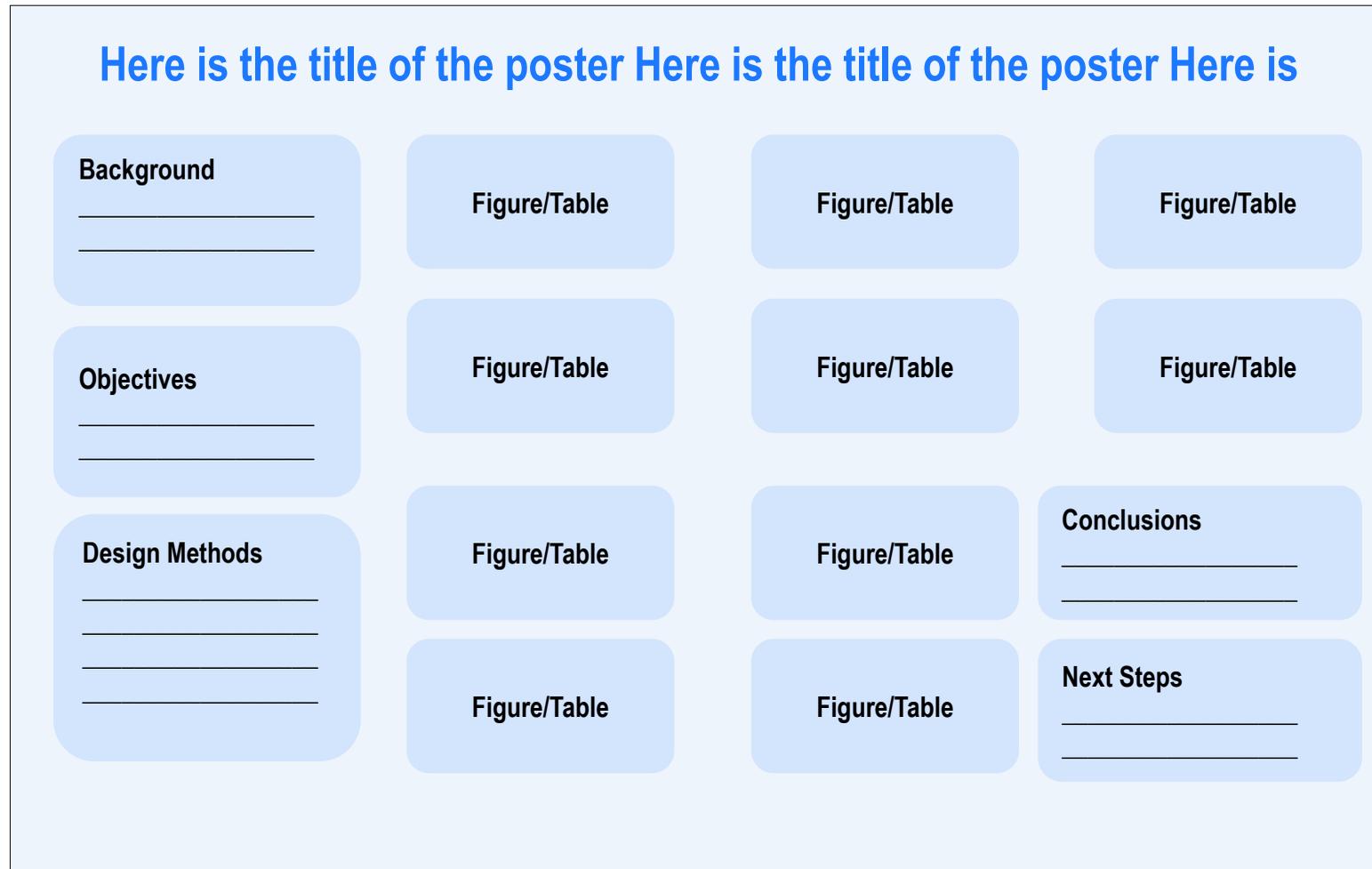
The “Pure Column” is the standard classic approach to poster design

Pure Column



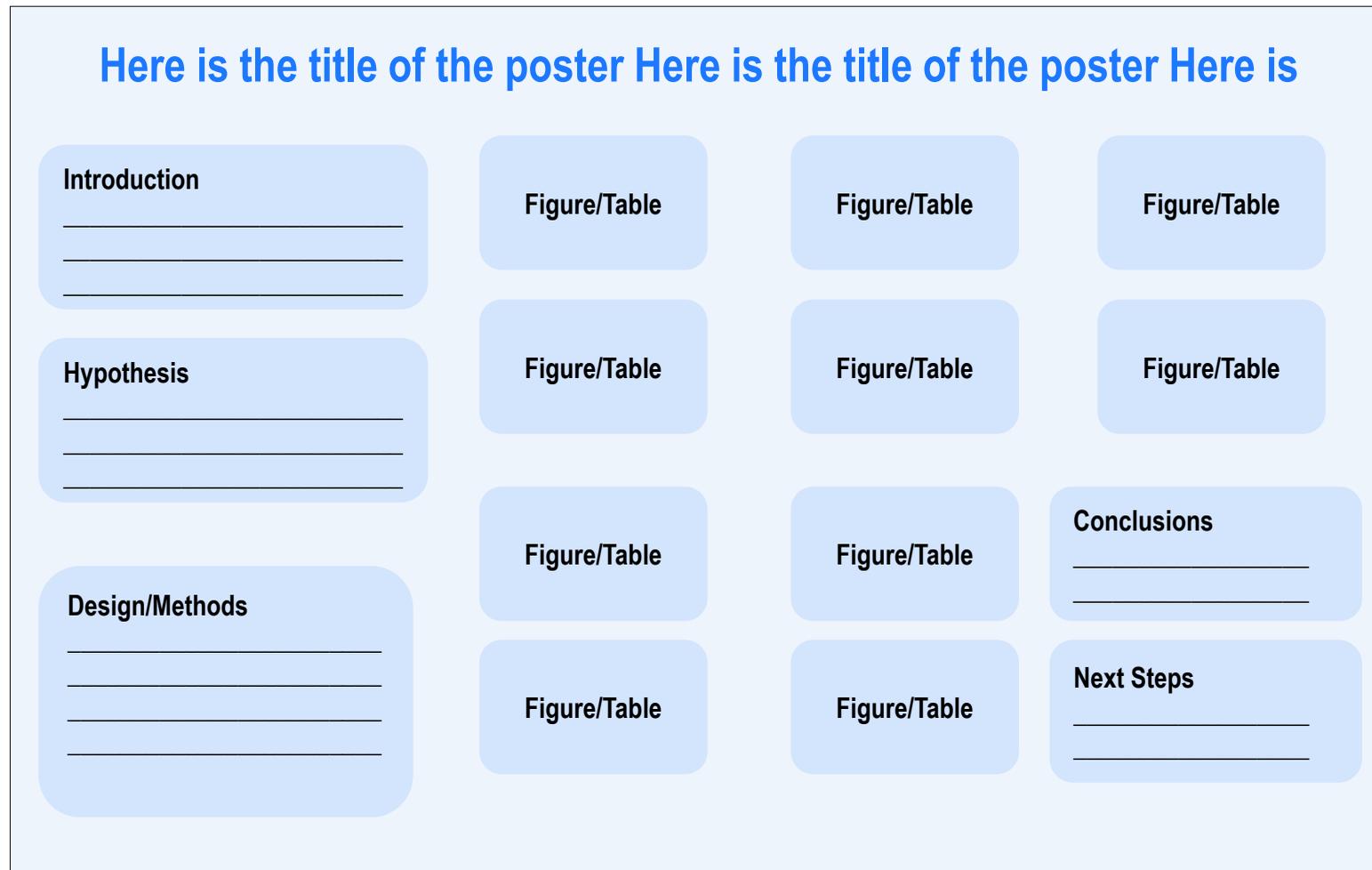
And here are some typical parts of a Pure Column poster, which can be three or four columns

Pure Column

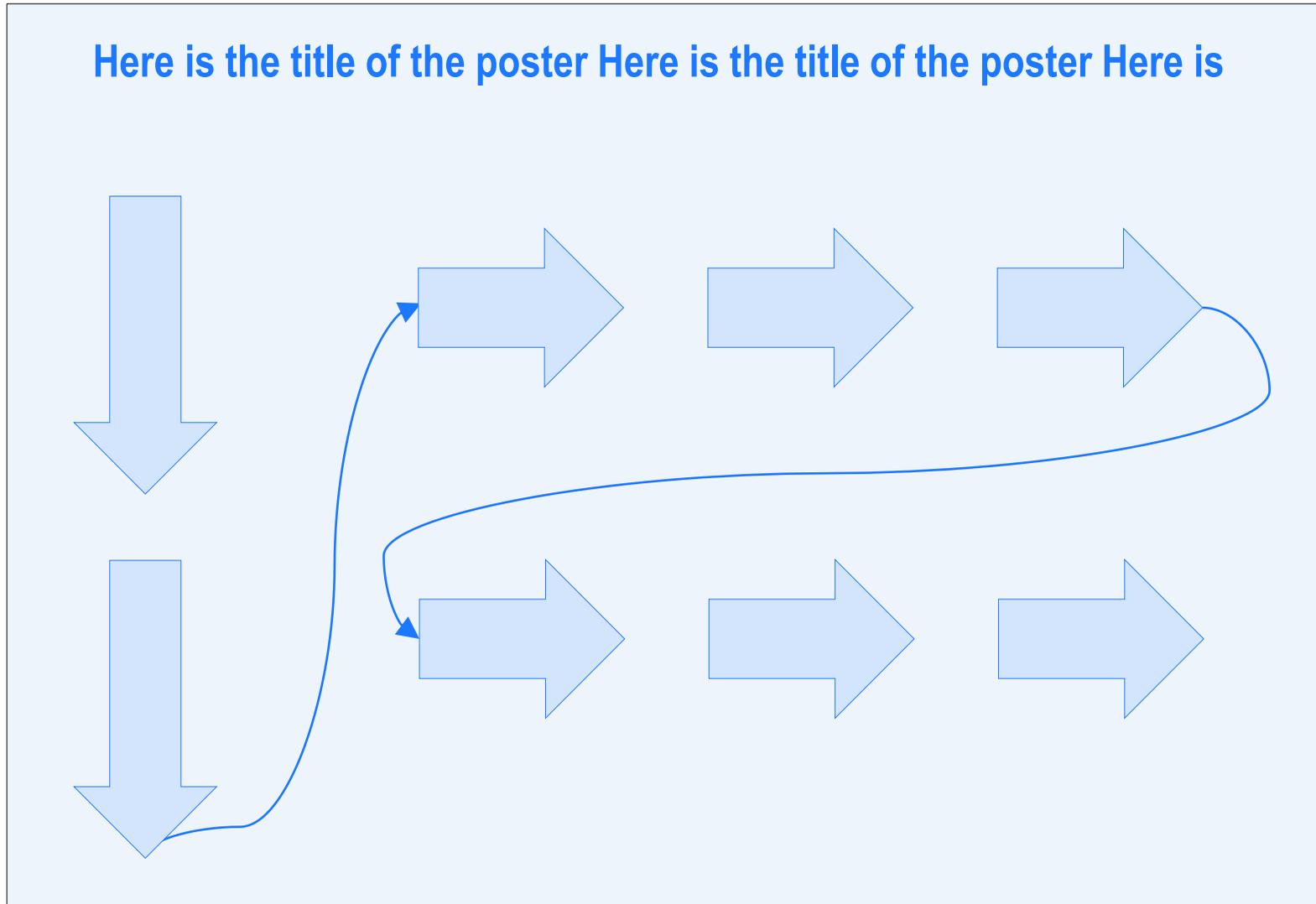


Here's another Pure Column approach that uses the terms "Introduction" and "Hypothesis" among others

Pure Column

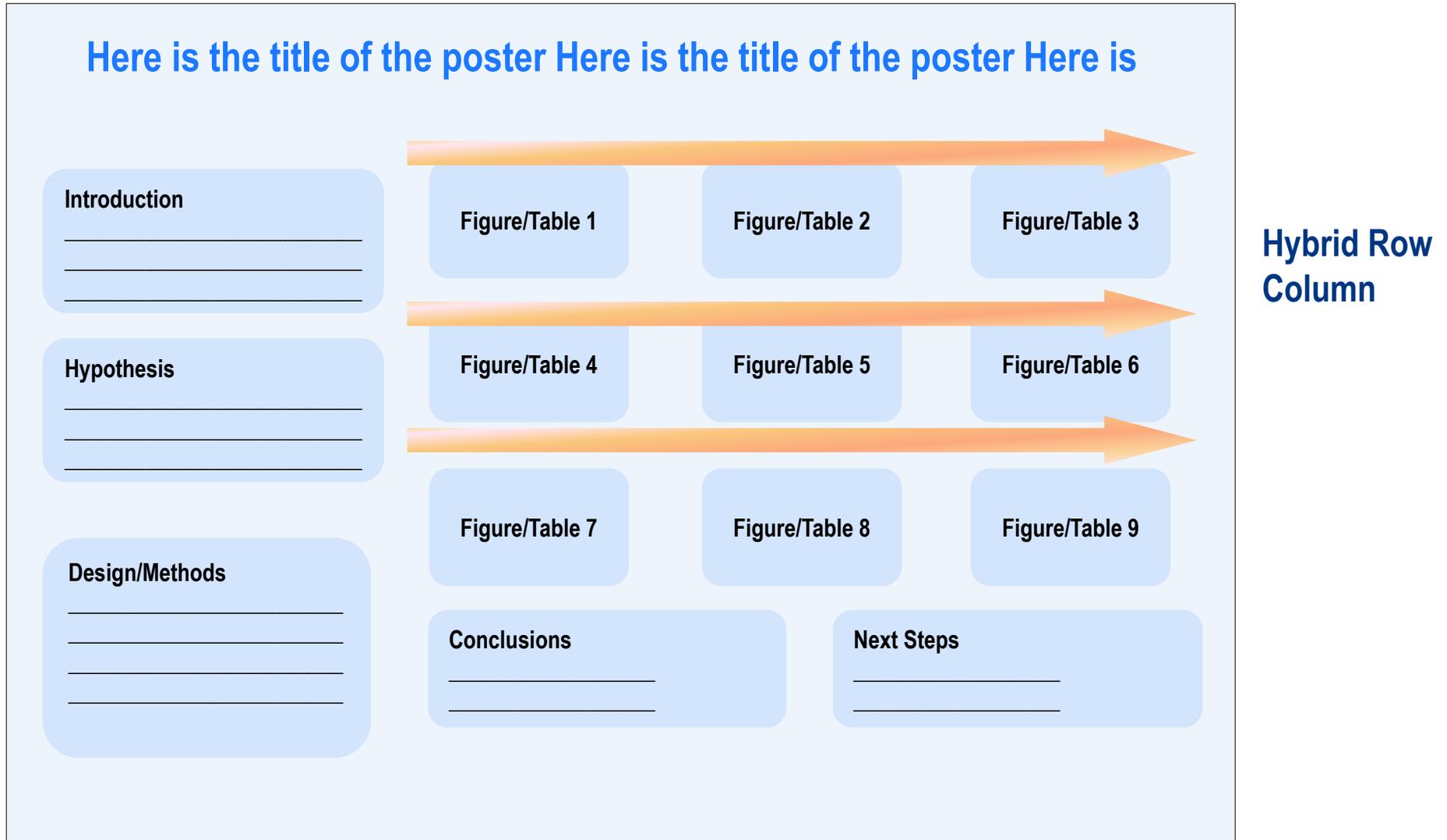


Consider Hybrid Row Column especially if figures reflect a sequential dependent time pattern in experimental results



Hybrid Row
Column

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

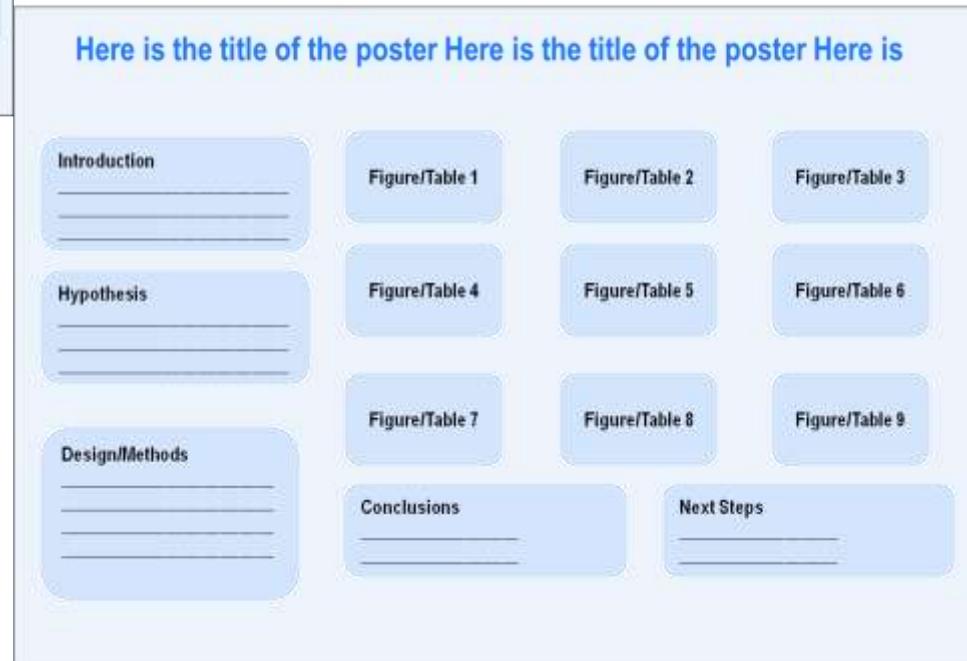


But note that looking at layouts, a reader **STILL** may not know how to read it – or have to work to figure out how to read it

Pure Column



Hybrid Row Column



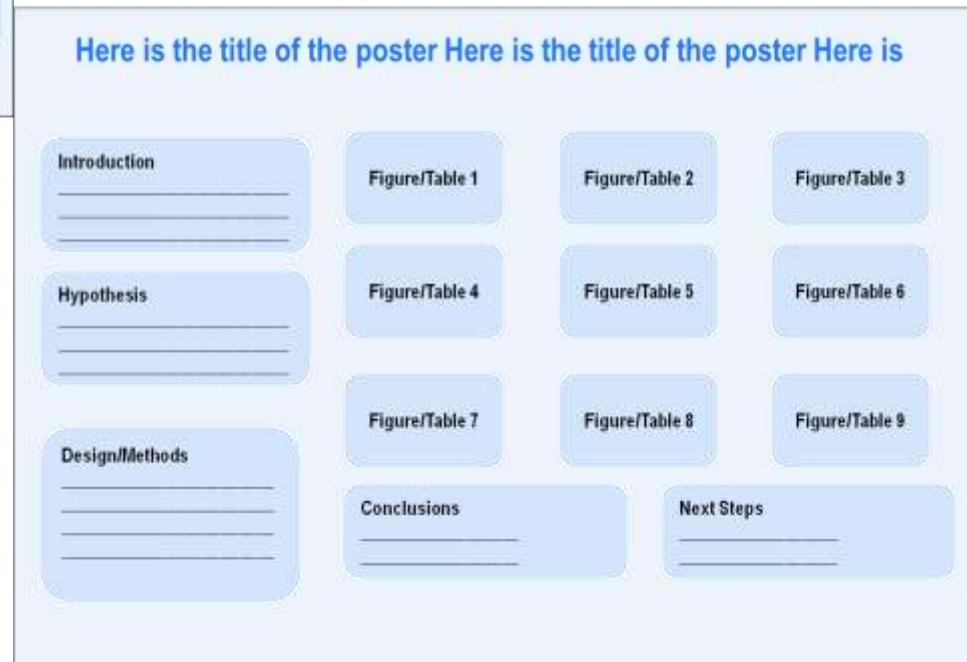
So what's the solution?

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

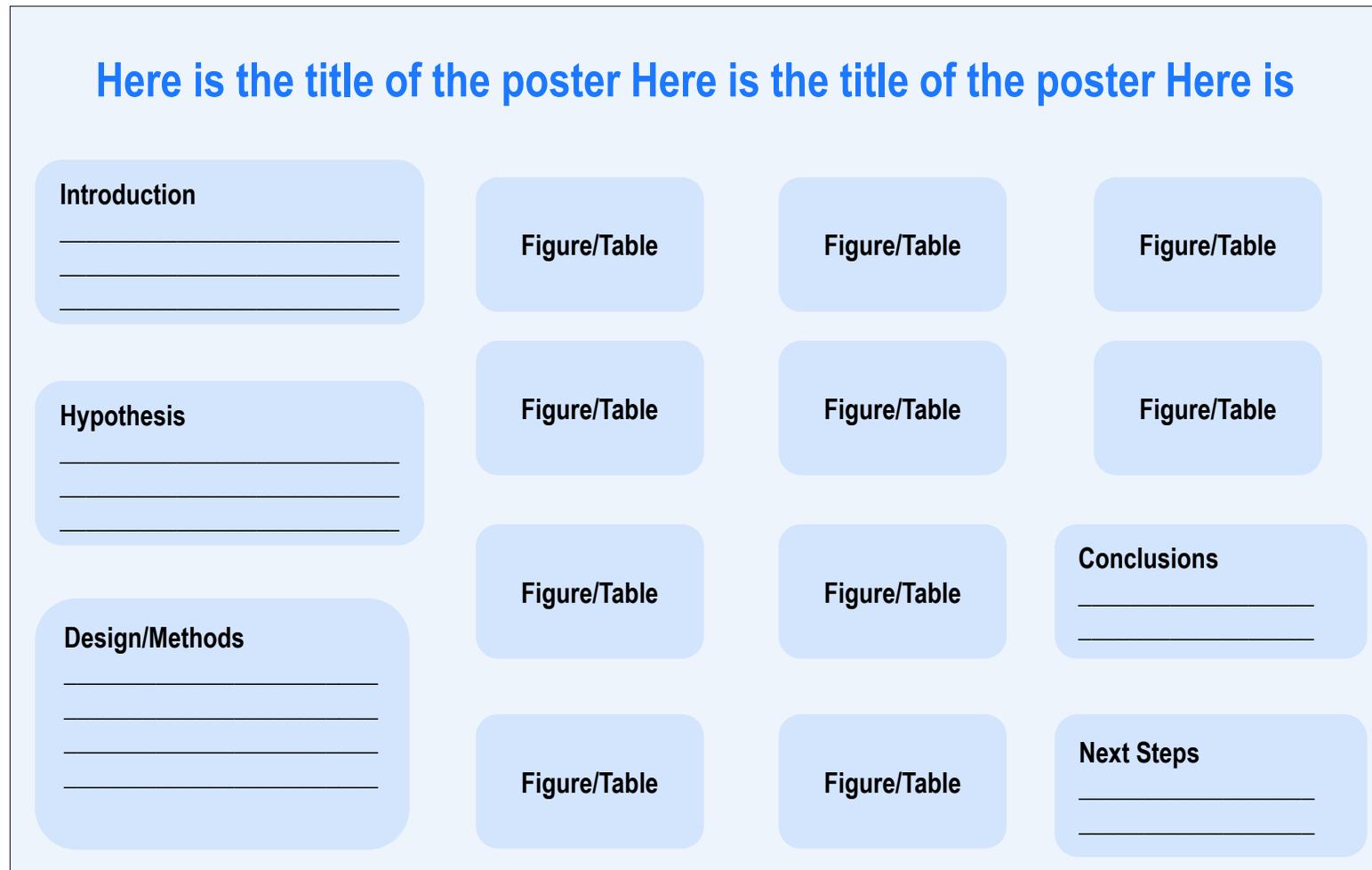


Hybrid Row Column



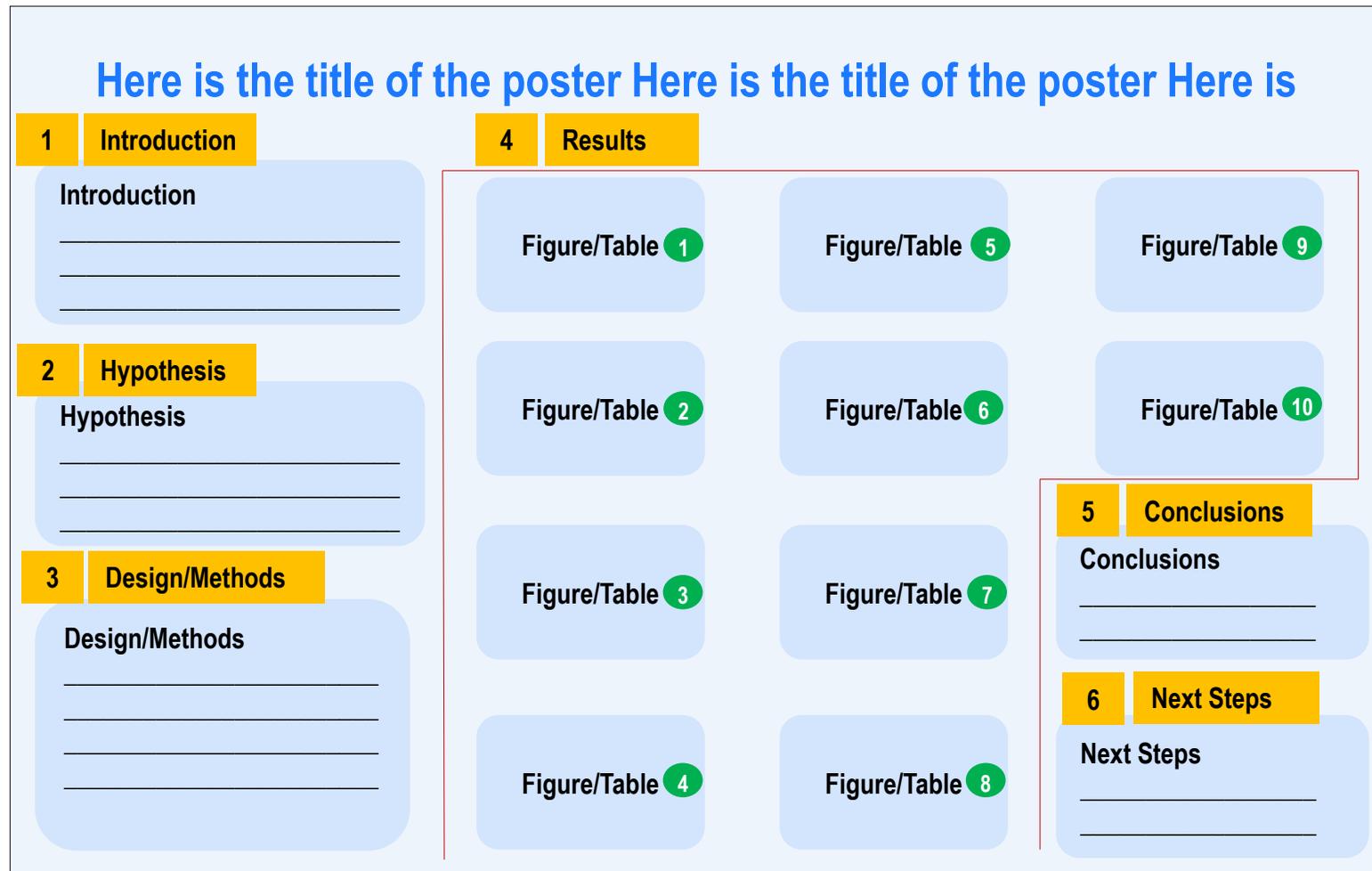
To assist readers

Pure Column



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

Pure Column



So what are the flaws here?

Poster
#316

New Transcripts Identified for *S. cerevisiae*, *S. pombe* and *Drosophila* Using Novel cDNA Cloning

Les-M. Hoffman*, Janina Görnemann, Erica Rodriguez, and David A. Brow
*EPICENTRE Biotechnologies, University of Wisconsin-Madison

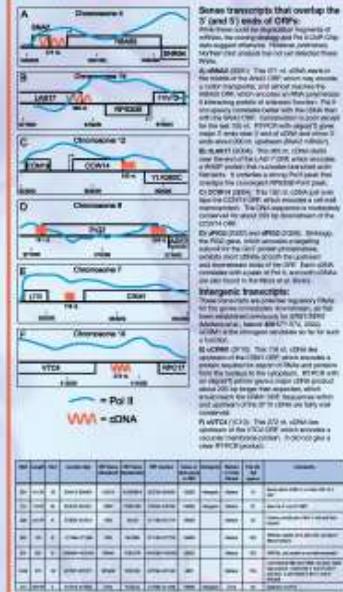
Abstract

Unannotated transcripts, including antisense, noncoding, and unusual regulatory RNAs, were identified using different nucleic acid approaches to cDNA cloning and sequencing. A rapid clone-enriched cDNA generation system will be described that uses microarrays of standard backsplices and a new cloning vector. Short reverse transcripts were used to facilitate cloning of 5' UTRs, including all cDNAs, but were essential for best outcomes. No size selection of cDNAs other than size-exclusion chromatography was used, and a high percentage of cDNAs were sequenced by length. The average cDNA length was approximately 430 nt, a size which is usually considered to favoring standard cDNA cloning protocols. Full-length transcription initiation sites and poly(A) addition sites were located for most clones by 5' and 3' end sequencing. Sequencing confirmed cDNA clone presence or cDNA library cloning PCR products gave similar results. Several cDNAs contained well-known 5' UTRs, including 5' UTRs from polyoma virus, SV40, and other viruses (Hoffman, B.J., et al., *Mol. Cell* 24, 729-738 (2006)). We moved transcription start sites, cDNAs, and some contained multiple 5' ends. If results show two separate transcription start sites, we have found using a different cDNA cloning strategy (Hoffman, F., et al., *PLoS Biol* 4, e183 (2006)). There was a preponderance of 5' sequences of cDNAs for various protein-coding genes in the cDNA library. We propose that the 5' ends of cDNAs are not primarily for the use of network structure, but the last nucleotide of a cDNA. Approximately 1/3 of the cDNAs have 5' nucleotide nucleotides for only one nucleotide protein genes, with a strong 1:1:1 bias toward all well-developed strand cDNAs over large strand cDNAs. The reason for such a discrepancy between small and large strand cDNAs is under investigation.

Introduction

cDNA cloning methods are being developed to meet the needs of investigators and the growing database of the transcriptome. The traditional view of newly published transcripts representing the main coding, sense cDNAs of a genome has given way to a more full account of the transcriptome. Inward addition of 5' and 3' end nucleotides, antisense, cryptic, unannotated or regulatory transcripts in cellular systems investigated. Some 3' sequences have been added to the transcriptome, including transcription factors, C/EBP, E2F, GATA, and other transcription factors. Other recent studies, such as the large-scale cDNA cloning and sequencing by Hoffman et al. (2), have identified a portion of the most transcripts that are not "GCY". Analysis of whole RNA-seq data and RNA-seq evidence of occupancy of using high-quality RNA-seq data has also indicated the presence of many transcripts that are not GCY. We present data for three different eukaryotic model organisms that show a range of new RNAs can be found by cloning cDNAs of "total cDNA" or "total RNA", rather than selecting for "total" "sense" cDNAs, as is traditionally done. The transcriptome of about 50,000 genes is annotated, both annotated and unannotated, across the genome. It ranges from 1.5 kb cDNAs to selected genes for cloning, the unannotated cDNAs transcripts are found. The "total transcript" method presented as a new technique in the transcriptome analysis, which is fast and straightforward. To allow the reproducibility of the technique, we need to change to include unannotated or unannotated transcripts from the transcriptome. We have designed, and necessary, 5' and 3' end nucleotides. RNA-seq data, which is used for the newly published transcriptome cDNAs, compared to the transcriptome found by cloning of RNA-seq data is largely affected by cDNA cloning (3). The transcripts are located in regions of the genome that appear to have significant PAF, but it is important, and better than unannotated regions of the genome.

Examples of *S. cerevisiae* shish RNAs



Summaries of the cDNA Cloning

Species	Library	Clones	Sequenced	Annotations
S. cerevisiae	Standard cDNA	100,000	100,000	100,000
	5' and 3' end cDNA	100,000	100,000	100,000
S. pombe	Standard cDNA	100,000	100,000	100,000
	5' and 3' end cDNA	100,000	100,000	100,000
Drosophila	Standard cDNA	100,000	100,000	100,000
	5' and 3' end cDNA	100,000	100,000	100,000

Color Legend
 unannotated cDNA
 sense cDNA
 shish RNA



Methods

RNA Isolation
 Total RNA was isolated using the RNeasy Lysis Buffer (Qiagen) and RNeasy Lysis Buffer (Qiagen). Total RNA was isolated using the RNeasy Lysis Buffer (Qiagen) and RNeasy Lysis Buffer (Qiagen). Total RNA was isolated using the RNeasy Lysis Buffer (Qiagen) and RNeasy Lysis Buffer (Qiagen).

cDNA Synthesis
 Total RNA was isolated using the RNeasy Lysis Buffer (Qiagen) and RNeasy Lysis Buffer (Qiagen). Total RNA was isolated using the RNeasy Lysis Buffer (Qiagen) and RNeasy Lysis Buffer (Qiagen). Total RNA was isolated using the RNeasy Lysis Buffer (Qiagen) and RNeasy Lysis Buffer (Qiagen).

PCR
 cDNA regions of interest were amplified using standard PCR conditions. PCR products were purified by gel extraction and ligated into the pUC19 vector. PCR products were purified by gel extraction and ligated into the pUC19 vector. PCR products were purified by gel extraction and ligated into the pUC19 vector.

Sequencing
 cDNA regions of interest were amplified using standard PCR conditions. PCR products were purified by gel extraction and ligated into the pUC19 vector. PCR products were purified by gel extraction and ligated into the pUC19 vector. PCR products were purified by gel extraction and ligated into the pUC19 vector.

5' and 3' end sequencing
 cDNA regions of interest were amplified using standard PCR conditions. PCR products were purified by gel extraction and ligated into the pUC19 vector. PCR products were purified by gel extraction and ligated into the pUC19 vector. PCR products were purified by gel extraction and ligated into the pUC19 vector.

cDNA Cloning Results

1. Saccharomyces cerevisiae
 We cloned cDNAs from total yeast RNA, from a highly enriched transcriptome, including antisense, non-coding, and unusual regulatory RNAs. A set of these transcripts are presented in the following panel. Multiple clones from the same transcriptome are shown with different cap sites and polyadenylation sites were found. We found an unusually large representation of nuclear gene products encoding mitochondrial proteins. Although we hypothesized that the RNA extraction method might favor the isolation of RNAs associated with the ribosomal network of yeast mitochondria, in fact most of the RNAs isolated did appear to be located with mitochondrial DNA (4). See also: *Mol. Cell* 18, 1003 (2005). Thus, the reason for the isolation of RNAs encoding mitochondrial proteins is unknown, although we have 1-5 kb cDNAs. There are few unannotated transcripts present in the yeast library, mostly full-length annotated transcripts. The average cDNA length was 475 nt.

2. Schistosoma mansoni
 A highly enriched transcriptome from total yeast RNA, from a highly enriched transcriptome, including antisense, non-coding, and unusual regulatory RNAs. A set of these transcripts are presented in the following panel. Multiple clones from the same transcriptome are shown with different cap sites and polyadenylation sites were found. We found an unusually large representation of nuclear gene products encoding mitochondrial proteins. Although we hypothesized that the RNA extraction method might favor the isolation of RNAs associated with the ribosomal network of yeast mitochondria, in fact most of the RNAs isolated did appear to be located with mitochondrial DNA (4). See also: *Mol. Cell* 18, 1003 (2005). Thus, the reason for the isolation of RNAs encoding mitochondrial proteins is unknown, although we have 1-5 kb cDNAs. There are few unannotated transcripts present in the yeast library, mostly full-length annotated transcripts. The average cDNA length was 475 nt.

3. Drosophila melanogaster
 A highly enriched transcriptome from total yeast RNA, from a highly enriched transcriptome, including antisense, non-coding, and unusual regulatory RNAs. A set of these transcripts are presented in the following panel. Multiple clones from the same transcriptome are shown with different cap sites and polyadenylation sites were found. We found an unusually large representation of nuclear gene products encoding mitochondrial proteins. Although we hypothesized that the RNA extraction method might favor the isolation of RNAs associated with the ribosomal network of yeast mitochondria, in fact most of the RNAs isolated did appear to be located with mitochondrial DNA (4). See also: *Mol. Cell* 18, 1003 (2005). Thus, the reason for the isolation of RNAs encoding mitochondrial proteins is unknown, although we have 1-5 kb cDNAs. There are few unannotated transcripts present in the yeast library, mostly full-length annotated transcripts. The average cDNA length was 475 nt.

Summary

A modified cDNA cloning technique allows novel cDNAs to be cloned from three model organisms. Cloning cDNAs without size selection may lead to the discovery of unannotated and unannotated RNAs. Many of the newly described RNAs are worthy of further study. We did not discover that any of these new RNAs are functional, but many are either 5' RNAs described in the current literature.

Web Resources
 The University of Wisconsin-Madison: <http://www.epicentre.org/>
 The USFQ/EPICENTRE website: <http://www.epicentre.org/>
 The cDNA cloning website: <http://www.epicentre.org/cdna-cloning/>
 The cDNA cloning website: <http://www.epicentre.org/cdna-cloning/>

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

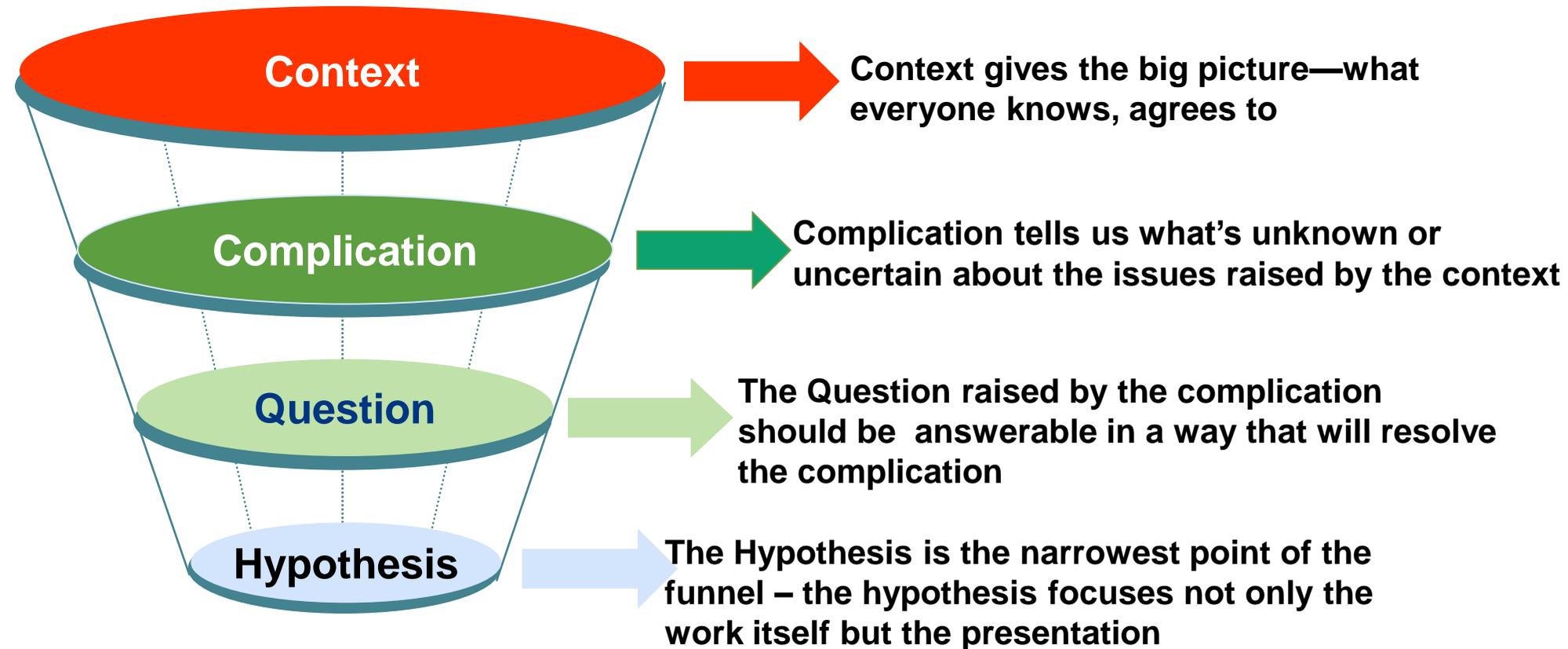
2 – Designing for Readability

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

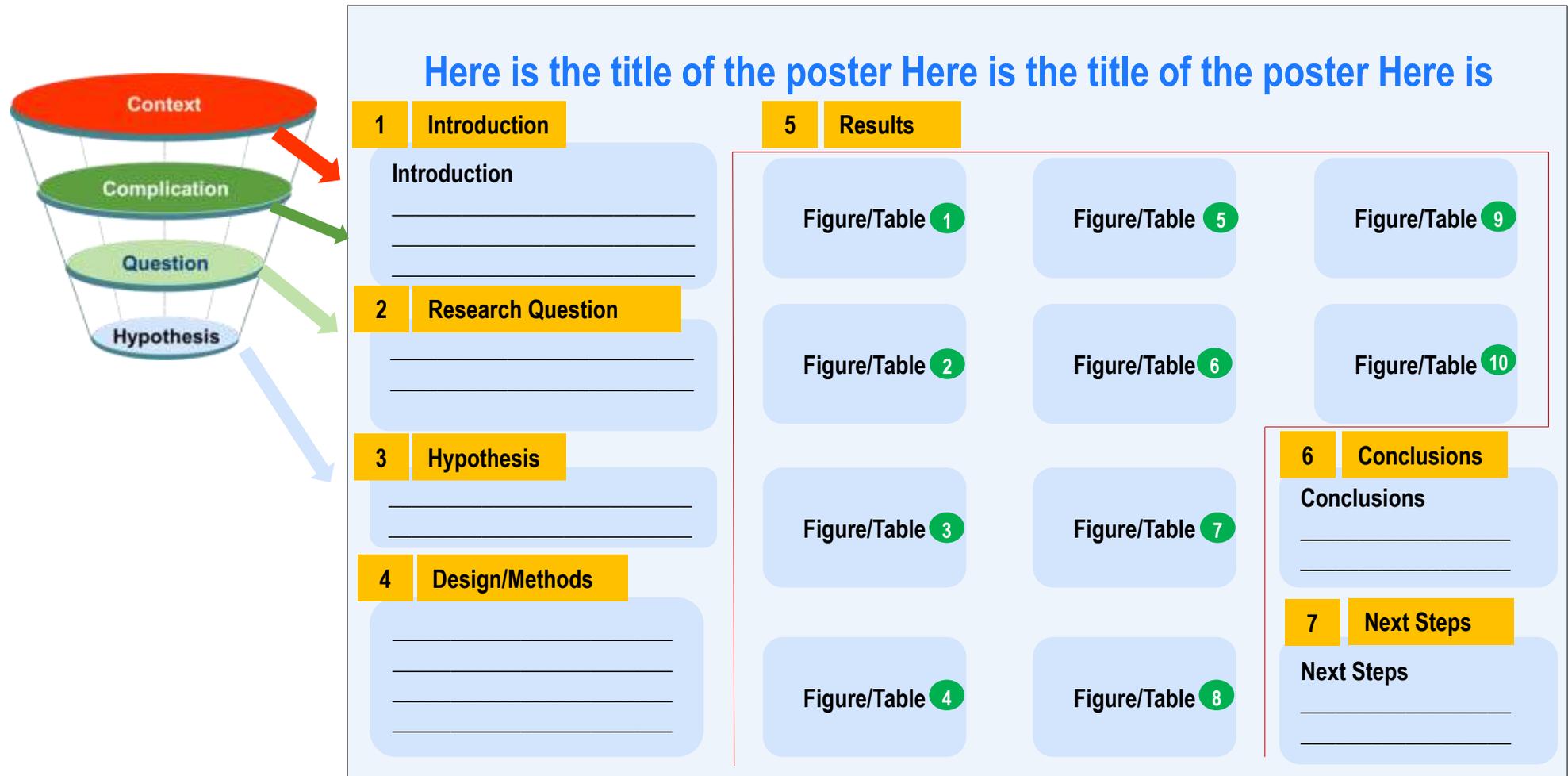
3 – Telling the Story

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



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

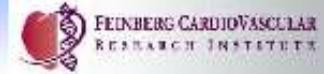


Let's look at an example poster. Does it follow our principles?



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



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

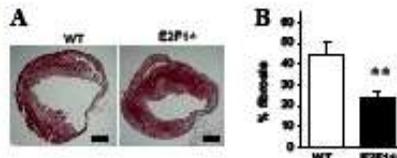


Fig 1: E2F1⁺ mice exhibit a smaller infarct size than WT mice after MI (A). Masson's Trichrome staining of hearts at day 28 after MI. (B) Quantification of area of fibrosis (**P<0.01, n=15).

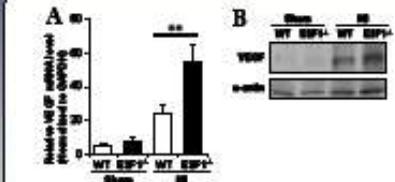


Fig 2: VEGF expression in the ischemic border area is higher in E2F1⁺ mice than in WT mice. (A) qRT-PCR for VEGF mRNA (**P<0.01, n=4). (B) Western blotting for VEGF protein (n=3).

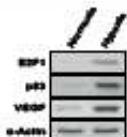


Fig 3: Hypoxia induces an increase in E2F1 and p53 protein levels. Western Blotting was performed using whole cell lysates from cultured cells after incubation for 24h under either normoxia or hypoxia (0.5% O₂).

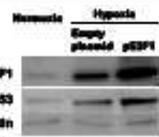


Fig 4: Overexpression of E2F1 increases p53 accumulation under hypoxia. Western Blotting of cardiac fibroblasts after they were transfected with an E2F1-expressing plasmid (pE2F1) or a control empty plasmid and exposed to hypoxia for 16h.

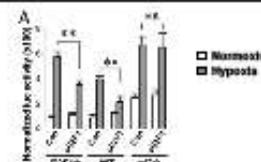


Fig 5: E2F1 overexpression represses VEGF promoter activity in p53 dependent manner. Cotransfection of VEGF promoter (2.6 kb)-luciferase plasmid and E2F1 expression plasmid vs. control plasmid into WT, E2F1⁺, or p53^{-/-} fibroblasts, which were subsequently exposed to hypoxia for 24h and assayed for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

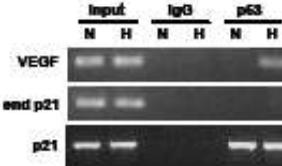


Fig 6: p53 occupies VEGF promoter *in vivo*. ChIP assays were performed in cardiac fibroblasts treated with normoxia (N) or hypoxia (H) for 16h. The p21 promoter region, regulated by p53, was used as positive control while the 3' untranslated region served as negative control.



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 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).

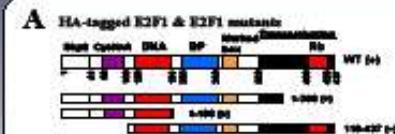


Fig 8: The p53-binding site of E2F1 is located at N-Terminus (1-108aa).

(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.