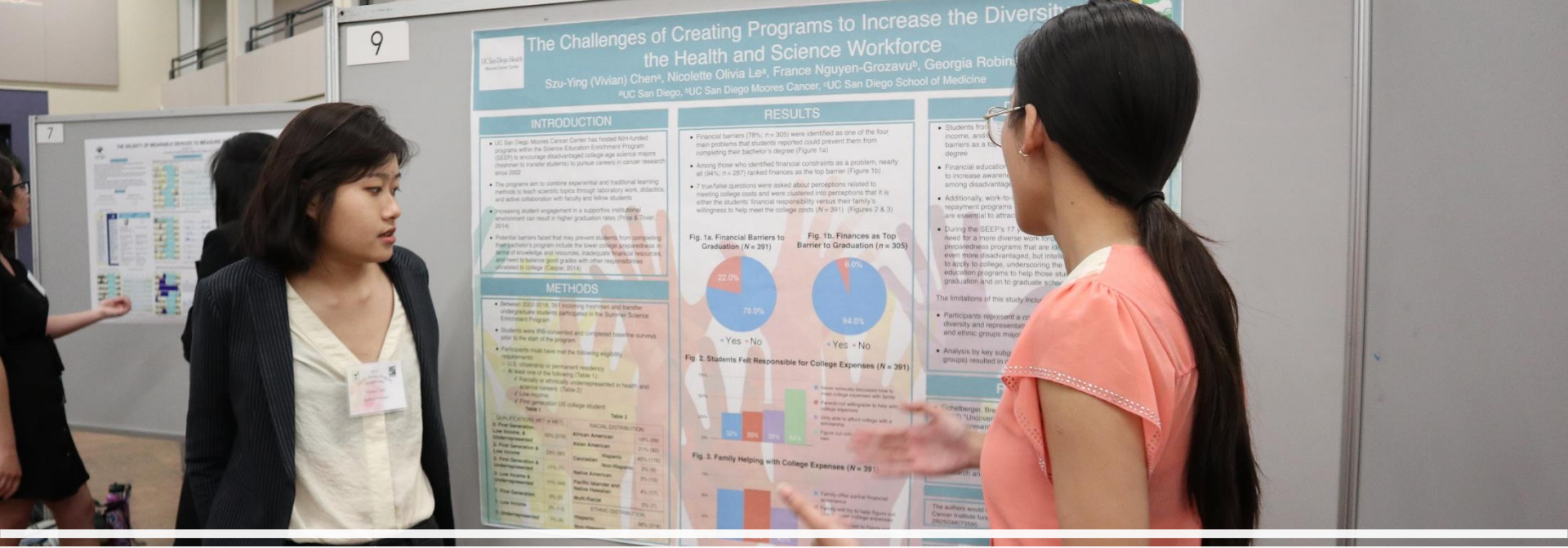
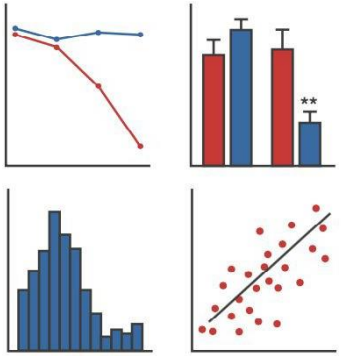
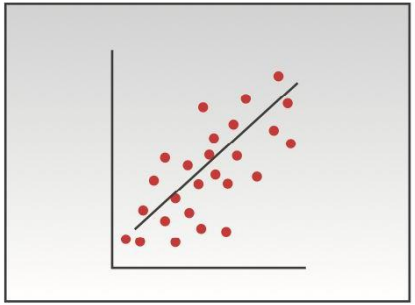


Poster Presentations

Undergraduate Research Hub





Scientific Content + Visual Information + Delivery

Your ideas, experiments, results, discussion, etc. Anything you want to communicate to an audience.

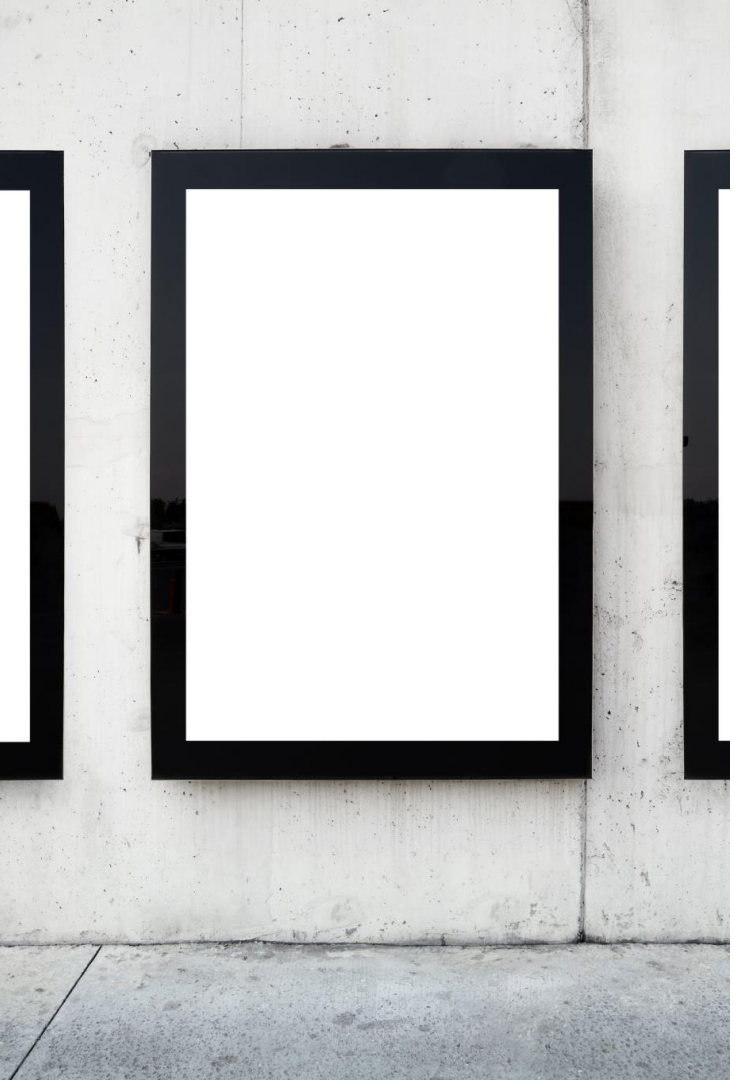
All of the visual aids you use to communicate information. In a paper, these are your figures; in a slide presentation, these are your slides; and in a poster presentation, this is your poster.

Your narrative that leads the presentation of your visual information. In a paper, your narrative is written on the page. In a slide and poster presentation, you deliver your narrative orally and with nonverbal communication (body language).

First step

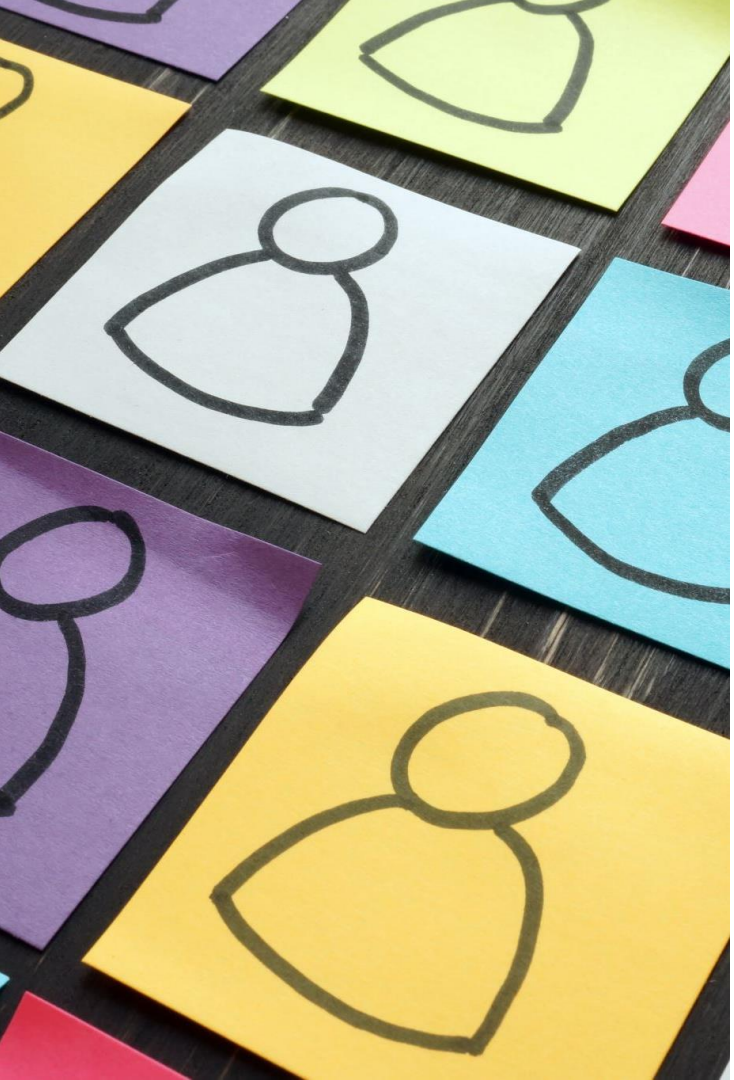
The Purpose of Poster Presentations

- Visually present a summary of your research
- Serve as a visual aid that **supports** your oral presentation



Balancing Act

- Detailed and complete AND concise
- Poster should stand on its own
- Poster shouldn’t be overwhelming with text



Design Tips

Plan

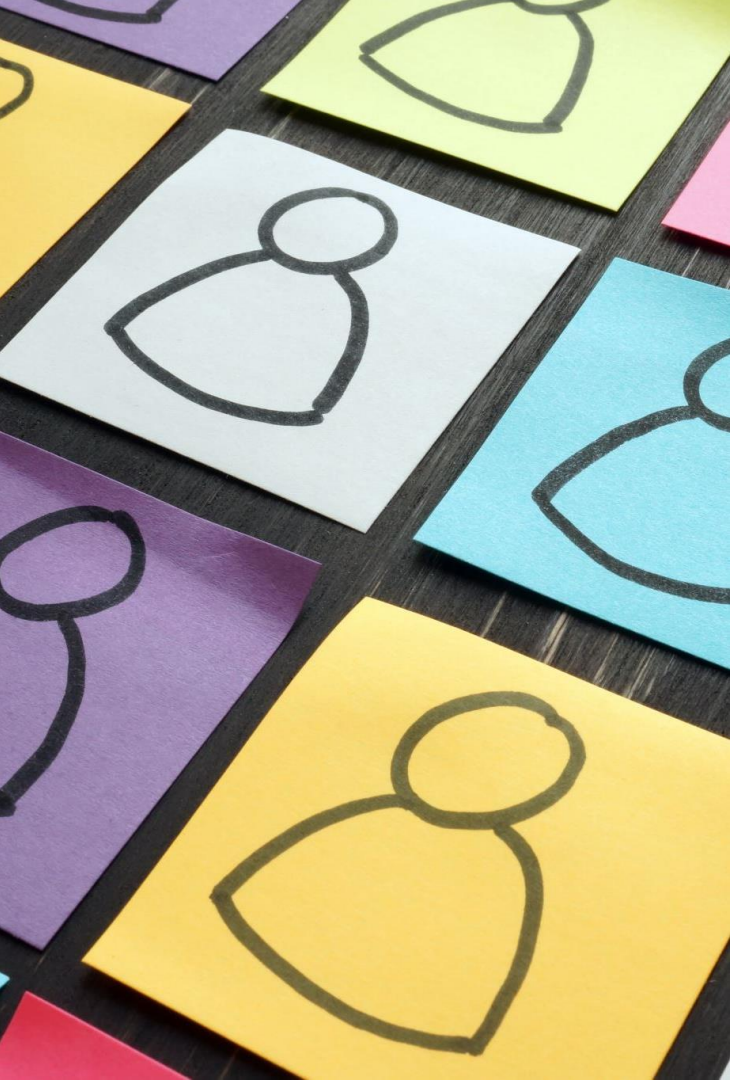
- Results
- Charts
- Bullet Points/Summaries
- Layout

Flow of sections

- Logical

Size

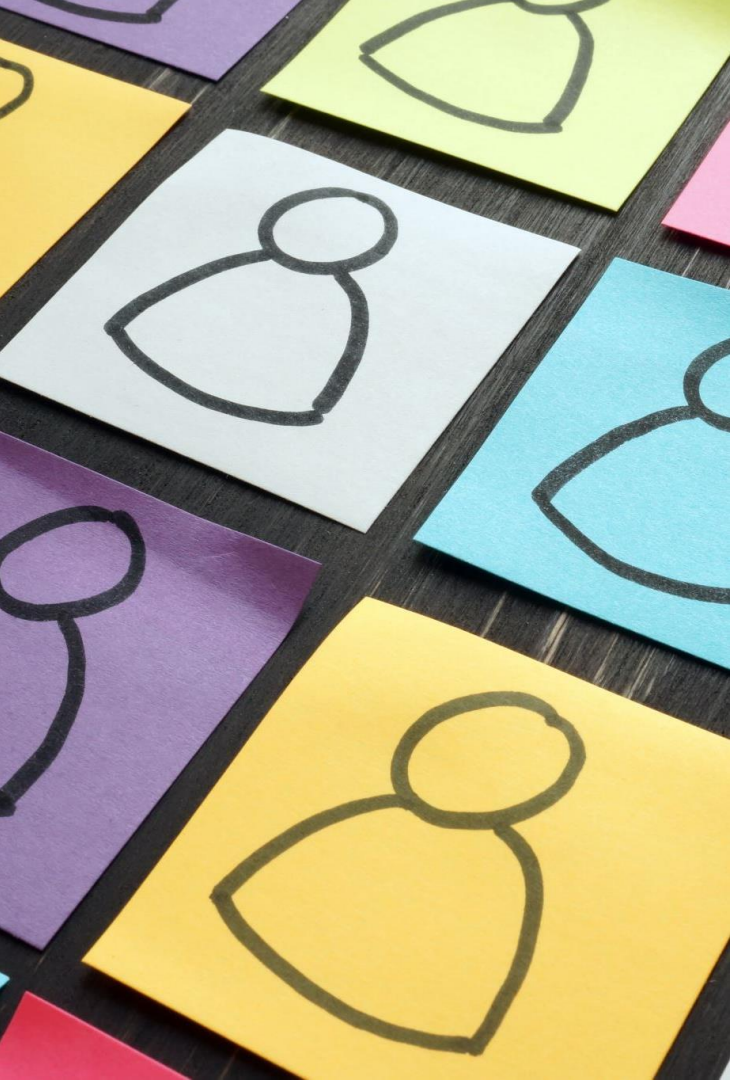
- Conferences dictate size
- 48" x 60“ is typical



Design Tips

Text

- Get to the point
- Use bullet points
- Font
  - Use standard font
  - Headings: 32 pt
  - Text: 24 pt
  - Figure Details: 18 pt
- Balance with images
- Word Count
  - ~100 words / section
  - ~1000 words total



Design Tips

Images

- Photos/Figures
- Use to tell the story (e.g. models, charts)
- Use with purpose
- Balance with text

Color

- ~2-3 colors
- Use white space

Title with Authors and Affiliations

Background/Introduction

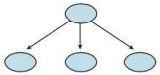
Results

Lorem ipsum dolor sit amet, consectetur adipiscing elit

Lucious Aurelius, Titus Crassus, Oenomaus Gannicus, Lucrecia Glaber
Universitatis Scientia

Background

Phasellus bibendum lacinia viverra. Sed suscipit mi id lectus molestie mattis fermentum lacus vulputate.

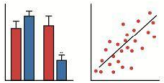


Hypothesis

Quisque non massa turpis, quis pretium eros. Pellentesque habitant morbi tristique senectus et netus et malesuada fames ac turpis egestas.

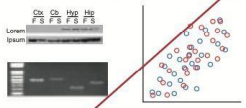
Result

Integer volutpat neque at erat imperdiet sit amet elementum tellus sodales.



Result

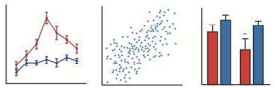
Mauris commodo suscipit quam, sed luctus ligula scelerisque aliquet.



Proin enim nisl, aliquam vitae sodales non, sagittis id nulla. Quisque et tempor nibh.

Result

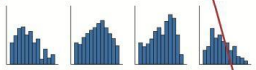
Duis id elit non neque faucibus tincidunt id nec nisl.



Cras consequat sem at augue hendrerit non malesuada elit accumsan. Pellentesque id lectus ligula. Vivamus ac erat id lorem posuere hendrerit tincidunt.

Result

Nam vitae vestibulum enim. Aliquam erat volutpat.



Result

Pellentesque diam nunc, euismod in tristique et, ornare vel neque.

Summary

Pellentesque nec turpis sem. Pellentesque adipiscing erat a ipsum laoreet vel consectetur mi placerat. Vivamus a purus nunc. Aliquam elementum, sapien vitae posuere malesuada, augue purus vehicula ligula, at posuere tellus ligula eget orci.

Cras aliquam turpis nec turpis adipiscing ut ornare nisi luctus. Curabitur gravida viverra massa, ut placerat purus sollicitudin non.

Vivamus euismod tristique consectetur. Nulla tempor faucibus dui, et elementum erat condimentum eget. Nunc ornare. Nulla vitae elementum ultrices, nunc nunc mollis lacus.

Specific Hypothesis/Question

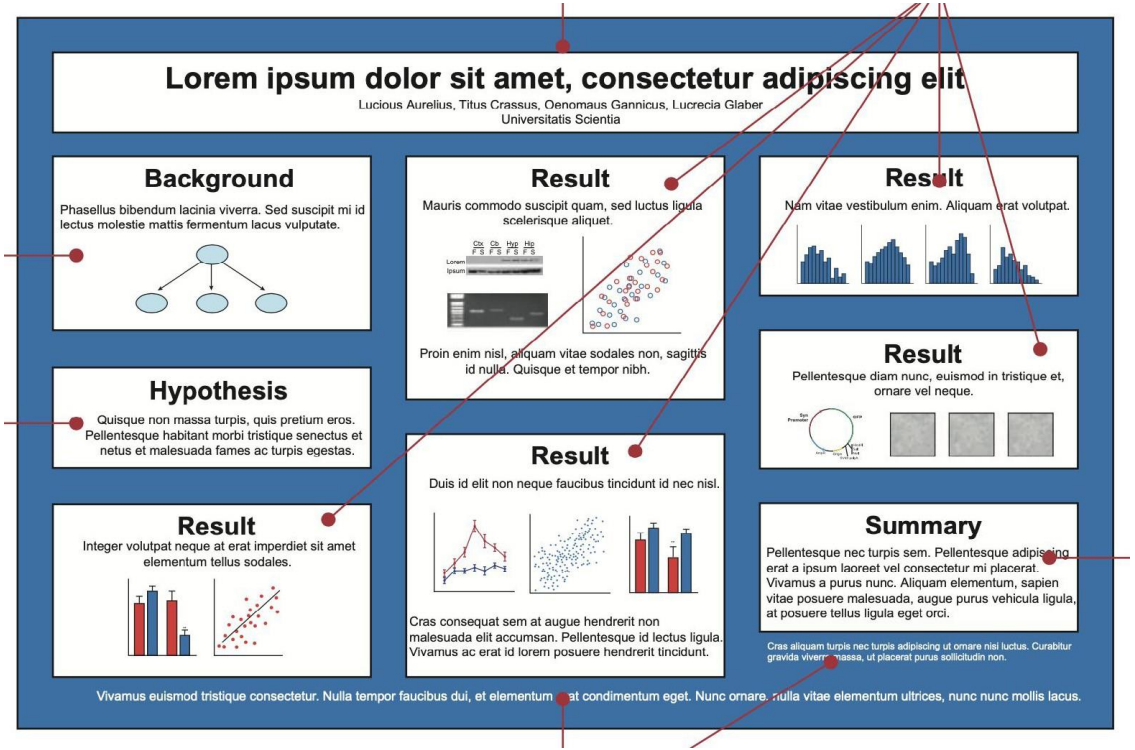
Summary/Conclusion

Acknowledgments and References

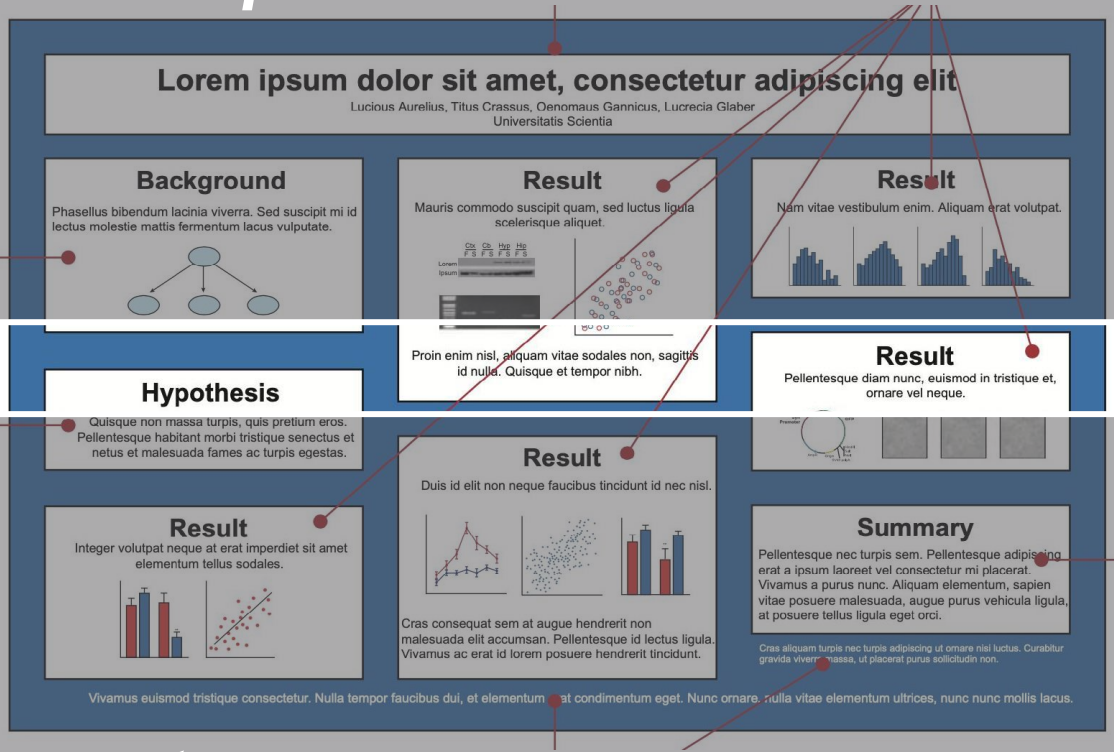
Everything on your poster needs to be visible from 10 feet away!

Title: 80 pt

Headings: 32 pt
Text: 24 pt
Figure Details: 18 pt



Premium poster space



Less visible poster space

Poster Sections

- Title, Authors, Logos
- Abstract
- Introduction/Background
- Hypothesis
- Methods
- Results
- Summary
- References
- Acknowledgements

Title

- Informative
- < 2 lines

Author(s)

- Presenter’s name
- Anyone else who contributed significantly
- PI

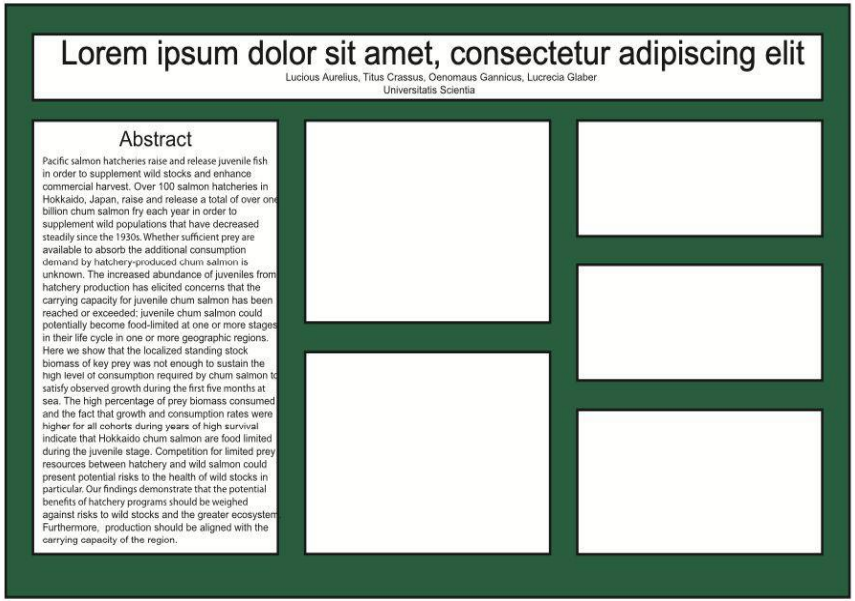
Affiliations

Logos

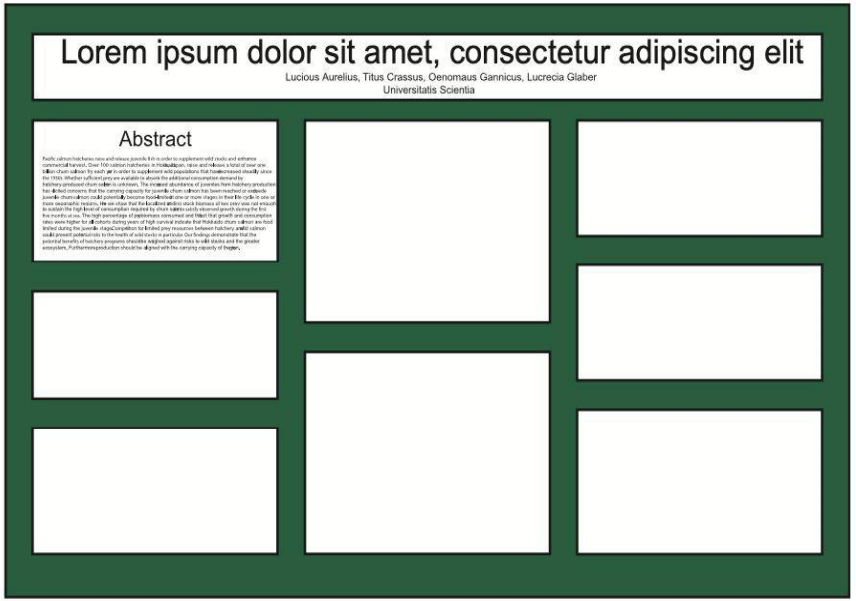
- University
- Program
- Grant

Abstract

- Can take up valuable space
- Consider leaving off if you need the space



Full abstract, legible font.



Full abstract, tiny font.

Introduction/Background

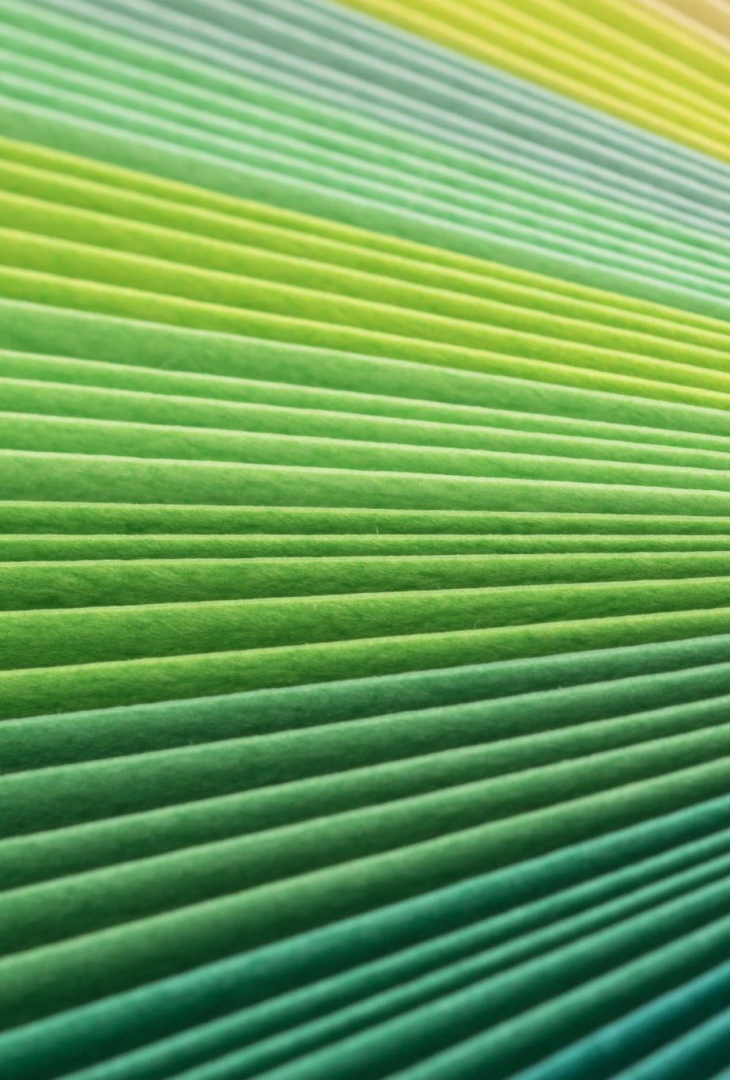
- Brief
- Hook your audience

Hypothesis

- Brief
- Possibly use diagrams

Methods

- Details depend on audience, complexity, and importance



Results/Findings

- 3-4 main findings
- Group/organize logically
- Easily understood figures
  - Simple, clear labels
- Complex figures
  - Reconsider using
  - Separate caption
- You will present the findings, don’t need to explain via text

Summary

- Restate main takeaways

References

- Don’t forget!
- Font size can be a little smaller

Acknowledgments

- PI/Mentor
- Significant People
- Funding
- Program

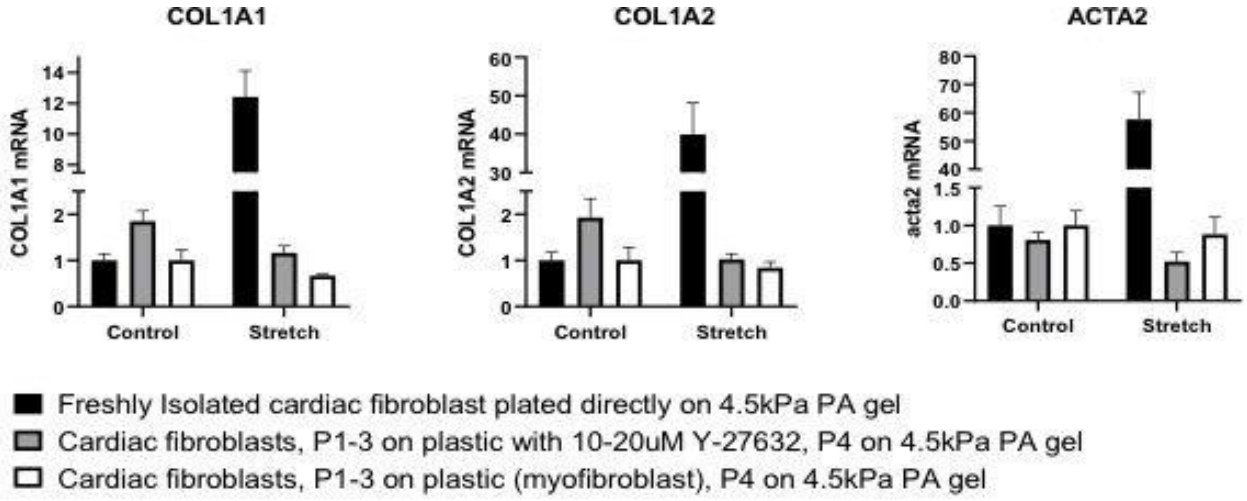


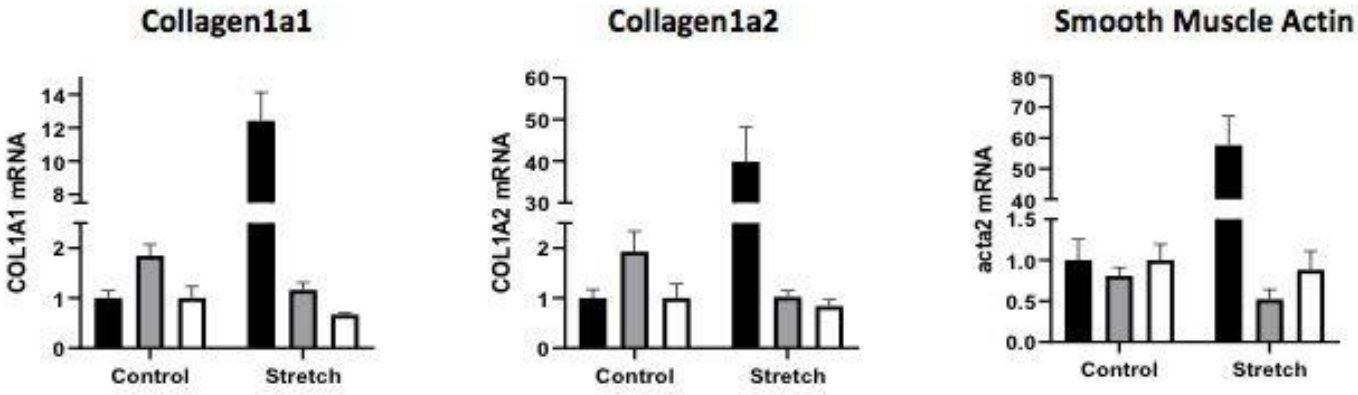
Figure 2. ROCK inhibition during cardiac fibroblasts to prevent myofibroblast differentiation and preserve pro-fibrotic responses to stretch.
Cardiac fibroblasts were expanded by pre-culturing on plastic for passage (P) 1-3 with and without the Rho kinase (ROCK) inhibitor Y-27632 (10-20uM). The culturing conditions were: 1) Freshly isolated cardiac fibroblasts plated directly onto gels (black bars); 2) Cardiac fibroblasts pre-cultured on plastic for P1-3 with Y-27632 (grey bars) and 3) without Y-27632 (white bars) before plating on 4.5kPa PA gels. All fibroblasts were left on PA gels for 3 days without Y-27632 before analysis. Fibroblasts were subjected to 30-minute stretch whereafter mRNA for collagen 1a1 (col1a1), collagen 1a2 (col1a2), and smooth muscle actin (acta2) were determined by real-time PCR. mRNA was normalized to 18S ribosomal RNA.

Result title is hidden

Too much text

ROCK inhibition during cardiac fibroblasts prevents myofibroblast differentiation and preserves pro-fibrotic responses to stretch.

Result title is clear



Freshly Isolated cardiac fibroblast plated directly on 4.5kPa PA gel
Cardiac fibroblasts, P1-3 on plastic with 10-20uM Y-27632, P4 on 4.5kPa PA gel
Cardiac fibroblasts, P1-3 on plastic (myofibroblast), P4 on 4.5kPa PA gel

Cardiac fibroblasts were expanded by pre-culturing on plastic for passage (P) 1-3 with and without the Rho kinase (ROCK) inhibitor Y-27632 (10-20uM).
All fibroblasts were left on PA gels for 3 days without Y-27632.

Simplified

O⁶-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Resensitizes Breast Cancer Cells to Anti-Estrogen Therapy

Joshua Smith[1], George C Bobustuc[1], Rafael Madero-Visbal[1], Jimmie Colon[1], Beth Isley[1], Jonathan Ticku[1], Kalkunte S. Srivenugopal and Santhi Konduri[1]

[1]Cancer Research Institute of M.D Anderson Cancer Center Orlando [2]Texas Tech University Health Sciences Center, Amarillo, TX

MD ANDERSON CANCER CENTER ORLANDO

SUPPORTED BY THE CHARLES LEWIS INSTITUTE

Abstract

Endocrine therapies using anti-estrogens are least toxic and very effective for breast cancers, however, tumor resistance to tamoxifen remains a stumbling block for successful therapy. Based on our recent study on the involvement of the DNA repair protein MGMT in pancreatic cancer (Clin Cancer Res. 15, 6087, 2009), here, we investigated whether MGMT overexpression mediates tamoxifen resistance. Specifically, we determined whether administration of MGMT inhibitor O^6-benzylguanine (BG)] at a non-toxic dose alone or in combination with the anti-estrogens (tamoxifen/fulvestrant) curtails human tamoxifen resistant breast cancer cell growth. Further, we also determined whether BG sensitizes breast cancers to tamoxifen using tamoxifen resistant cells.

MGMT expression was found to be increased in breast cancer cells relative to normal breast epithelial cells. Also, MGMT levels were significantly higher in tamoxifen resistant MCF-7 compared to the parent cells. Silencing of the ER-α expression using a specific siRNA resulted in augmentation of MGMT mRNA and protein levels by 2 fold. We also observed an inverse correlation between MGMT and p53 levels in breast cancer cell lines; moreover, p53 downregulation was accompanied by increased MGMT expression. Other experiments showed that BG alone or BG in combination with tamoxifen or fulvestrant decreased ER-α expression, whereas BG alone and fulvestrant alone increased and decreased the same respectively. However, all these treatments increased the $p21^{cip1}$ mRNA and protein expression significantly. BG inhibited tamoxifen resistant breast cancer growth in a dose-dependent manner and it also resensitized resistant breast cancer cells to anti-estrogen therapy (TAM/ICI). These combinations also enhanced the cytochrome C release and the PARP cleavage, indicative of apoptosis. In breast cancer xenografts, BG alone or a combination of BG with tamoxifen or fulvestrant caused significant tumor growth delay and immunohistochemistry revealed that BG inhibited the expression of MGMT, ER- α, ki-67 and increased $p21^{cip1}$ staining. These findings suggest that MGMT inhibition may provide a novel and effective approach for overcoming tamoxifen resistance.

Introduction

Recent advances in breast cancer research have identified key pathways involved in the repair of DNA damage induced by chemotherapeutic agents. The ability of cancer cells to recognize DNA damage and initiate DNA repair is an important mechanism for therapeutic resistance and has a negative impact on therapeutic efficacy. A number of DNA-damaging alkylating agents attack the nucleophilic O^6 position on guanine, forming mutagenic and highly cytotoxic interstrand DNA crosslinks. The DNA repair enzyme O^6-alkylguanine DNA alkyltransferase (AGT), encoded by the gene MGMT, repairs alkylation at this site and is responsible for protecting both tumor and normal cells from alkylating agents. MGMT is expressed constitutively in normal cells and tissues. In breast tumors, MGMT gene expression is elevated and levels are up to 4-fold higher than in the normal breast. Interestingly, it has been shown that tamoxifen accelerates proteasomal degradation of MGMT in human cancer cells. In 1991, Pegg, Moschel, and Dolan observed that O^6 benzylguanine (BG) inhibited AGT and potentiated the cytotoxicity of both chloroethylating agents and methylating agents. In a series of important observations, they fully characterized the interaction between BG and AGT and its therapeutic impact. They showed that BG binds AGT, transferring the benzyl moiety to the active-site cysteine [29]. The reaction is very rapid and more potent than any other previously known AGT inhibitor. BG is not incorporated into DNA in living cells and reacts directly with both cytoplasmic and nuclear AGT. Because BG is a pseudosubstrate for MGMT which results in the covalent transfer of benzyl group to the active site cysteine, the MGMT protein is degraded after each reaction. This stoichiometric reaction mechanism effectively depletes the AGT content in tumors and the associated repair of alkylation damage. BG is currently undergoing clinical trials in various cancers to increase the efficacy of alkylating agents.

Interestingly, several observations suggest an inverse correlation between the levels of MGMT and p53 tumor suppressor proteins where wild-type p53 suppresses transcription of human MGMT expression. Unfortunately, p53 function is often inactivated or suppressed in human cancers; therefore, restoration of wt-p53 activity is essential for the success of some treatments. However, whether or not this is mediated by suppression of MGMT expression has yet to be determined. To date, the cross-talk between MGMT and ER-alpha (and the link to p53 expression) has not been explored in drug (i.e., tamoxifen) resistant breast tumors. The anti-estrogen tamoxifen is the most commonly used treatment for patients with estrogen receptor positive breast cancers. Although many patients benefit from tamoxifen in the adjuvant and metastatic settings, resistance to this endocrine therapeutic agent is an important clinical problem. The primary goal of present study was to investigate the mechanisms of anti-estrogen drug resistance and to design new therapeutic strategies for circumventing this resistance. The results show that MGMT expression is increased in TAM-resistant breast cancers and inhibition of MGMT by BG significantly improves TAM-sensitivity.

Results

Prolonged Treatment of Tamoxifen Increases MGMT Expression: We developed a tamoxifen resistant MCF-7 cell line by using prolonged treatment of tamoxifen on the parental ER-positive breast cancer cell line, MCF-7. Tamoxifen-resistant MCF-7 cells proliferate at rates similar to the parental MCF-7. Prolonged treatment of tamoxifen onto MCF-7 cells increased MGMT expression compared to parental MCF-7 cells by 2 fold (Fig.1).

Knocking Down ERα Enhances MGMT Expression in Tamoxifen Resistant Breast Cancer Cells: It is not known whether ERα and MGMT transcriptionally regulate each other in tamoxifen resistant breast cancer cells. We therefore investigated whether down regulation of ERα has any effect on endogenous MGMT expression in these cells. As expected, downregulation of ERα using specific siRNA significantly reduced ERα protein levels in these cells. Western blot analysis was performed and the results in the left panel (Fig. 2A) shows that silencing of ERα increases MGMT expression in these cells, and interestingly, the results in the right panel (Fig.2B) show increased MGMT mRNA levels were increased as assessed by qRT-PCR. These data suggest that ERα–mediated signaling functions to repress MGMT gene expression in breast cancer cells.

Transcriptional Regulation Between MGMT and p53: Previously, it was reported that p53 negatively regulates MGMT in breast cancer cells. Therefore, we addressed whether or not silencing the p53 enhances endogenous MGMT transcription. Tamoxifen resistant MCF-7 cells were transfected with either p53 siRNA (p53-KD) (Fig.2C) or MGMT siRNA (MGMT-KD) (Fig.2D) along with Non-specific siRNA (NS). MGMT expression was consistently increased in p53 knock down cells, with different experiments showing a ~ fold augmentation (Fig. 2A) and as expected, knocking down MGMT decreased MGMT transcription where as p53 mRNA levels were unaffected in MGMT knockdown cells (Fig.2D). These results confirm that p53 can regulate MGMT at the transcriptional level.

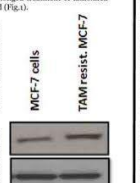


Figure 1. MCF-7 parental and tamoxifen resistant MCF-7 cell pellets were prepared, proteins were isolated and MGMT expression was detected by western blot analysis. Tamoxifen resistant MCF-7 breast cancer cells significantly increased MGMT expression compared to MCF-7 parental cells.

O^6-Benzylguanine Plays a Dual Role in Tamoxifen Resistant MCF-7 Cells: Contrasting with the experiments above, next, we studied whether or not knocking down MGMT has any effect on ERα transcription. As expected, knocking down MGMT decreased MGMT gene transcripts. However, it was interesting to find that ERα gene transcription was also reduced after MGMT silencing. (Fig.2E). These data demonstrate that BG has the ability to attenuate the not only the MGMT, but also the ERα transcription, indicating a possible dual role for MGMT blockers in these breast cancer cells.

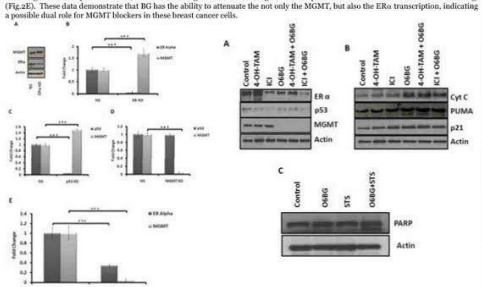


Figure 2. (A) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100nM) (NS), and cells were harvested 72h post transfection. Total proteins were isolated and ERα and MGMT expression was determined by western blot analysis. MGMT protein was significantly increased in ERα knock down cells (B) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100nM) (NS), and cells were harvested 72h post transfection. Total RNA was isolated and MGMT and ERα transcription was determined by qRT-PCR. MGMT transcription was significantly increased in ERα knock down cells. (C) Total RNA was isolated from non-specific siRNA (NS) (100nM) and p53 siRNA (90 nM) knock down tamoxifen resistant MCF-7 breast cancer cells. MGMT and p53 transcription was determined by qRT-PCR. (D) Total RNA was isolated from non-specific siRNA (NS) (100nM) and MGMT siRNA (100nM) knock down tamoxifen resistant MCF-7 breast cancer cells. MGMT and p53 transcription was determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (C & D).

Figure 3. (A) Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 μg) and 48h post treatment 4-OH-TAM (1μM), ICI (1μM) either alone or in combination with BG. 24h post treatment cells were harvested and proteins were isolated and western blot analysis was performed. (A) ERα, p53 and MGMT expressions (B) Cytochrome C, PUMA and p21 was determined by western blot analysis (C) tamoxifen resistant MCF-7 cells were treated with or without BG for 48h and later treated with staurosporin (1 μM/L) for 6 hrs PARP cleavage was determined by western blot analysis.

O6-Benzylguanine Modulates p53 Down-Stream Targeted Protein Expressions: Encouraged by the results reported, we investigated the effect of combination therapy on endogenous MGMT, p53, and ERα protein expressions. As expected, BG decreased MGMT expression, while combination therapy (4-OH-TAM or ICI combined with BG) significantly decreased both MGMT and ERα expressions. BG alone or in combination with tamoxifen or ICI decreased ER-α expression, whereas tamoxifen alone and ICI alone increased and decreased the same respectively (Fig.3A). p53 expression was slightly altered after ICI treatment. The reduction in p53 expression by ICI alone was reversed when BG was combined (Fig.3A). We investigated the effect of BG on proteins which are involved in cell cycle regulation, apoptosis in tamoxifen resistant breast cancer cells. All these treatments significantly increased the $p21^{cip1}$ protein expression (Fig.3B). PUMA expression was also increased with these treatments. Hence, PUMA may have translocated to the mitochondria, cytochrome C is released (Fig.3B), and apoptosis was triggered in these cells in presence of combination therapy. PARP cleavage is seen in BG treated cells in presence of staurosporin as an indicative of apoptosis (Fig.3C). Therefore, this data suggest that BG promotes cell cycle arrest and can induce apoptosis by modulating p53 function.

O6-Benzylguanine Modulated Transcriptional Targets in Tamoxifen Resistant Breast Cancer Cells: The effect of combination therapy on endogenous MGMT mRNA levels we aso stuid. Quantitative real-time PCR (qRT-PCR) resulted that anti-estrogens (TAM/ICI) increased the MGMT expression while the combination therapy decreased it compared to control levels. ERα transcription was decreased compared to controls with all these treatments (Fig.4A). Surprisingly, p21 and PUMA mRNA was significantly increased in the presence of combination treatments (Fig.4B &C). These results suggests that p53 mediated target gene transcription was affected by the drug combinations in breast cancer cells (Fig. 3 & 4).

O6-Benzylguanine Enhances p21 Transcriptional Activity in Tamoxifen Resistant Breast Cancer Cells: In order to investigate the effect of BG on p53 function, we performed luciferase reporter assays. Tamoxifen resistant MCF-7 breast cancer cells were transfected with p21 luc promoter construct in presence or absence of BG (target gene of p53). These results clearly demonstrate that BG significantly enhanced p21 transcriptional activity by 4-5 fold in these cells (Fig.4D).

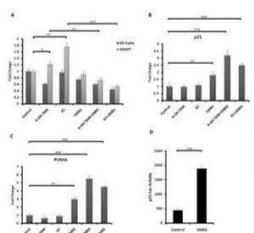


Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 μg) for 48h and later 4-OH tamoxifen and ICI (1μM) was either alone or in combination with BG and 24h later cells were harvested and total RNA was isolated. (A) MGMT and ERα (B) p21 transcription (C) PUMA transcription was determined by qRT-PCR. 4-OH tamoxifen and ICI induces MGMT transcription. BG induced PUMA and p21 transcription. (D) Tamoxifen resistant MCF-7 breast cancer cells were transfected with p21-luc construct and 6h later treated with BG and 24h later cells were harvested. p21 transcriptional activity was significantly increased by BG in these cells.

O6-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Increase Resistant Breast Cancer Cell Sensitivity to Anti-Estrogen Therapy (TAM/ICI): Detailed necropsy revealed that all the mice had tumors in the breast. The data summarized in Table 1 show the daily BG alone or in combination with twice weekly tamoxifen/ICI significantly decreased median tumor volume and weight as compared with that seen in tamoxifen/ICI treated and control mice. The combination of BG with tamoxifen or ICI produced the greatest decrease in median tumor volume as compared with control mice (83.99 mm³, 9.33 mm3 (TAM+BG), respectively; p< 0.0001); (83.99 mm³, 31.60 mm³ (ICI+BG), respectively; p<0.0100). Tumor weight was also significantly reduced in mice treated with combination therapy as compared with control mice (81.23 mg, 22.30 mg (TAM+BG), respectively, p<0.0005); (81.23 mg, 51.57 mg (ICI+BG), respectively, p<0.0005). (Table.1). Body weight was not changed among all treatment groups as compared with control mice. No visible liver metastases were present (enumerated with the aid of a dissecting microscope) in all treatment groups.

Histology and IHC Analysis: We next determined the *in vivo* effects of BG (alone or in combination) with tamoxifen/ICI. Tumors harvested from different treatment groups were processed for routine histological and IHC analysis. Tumors from mice treated with BG alone or in combination with tamoxifen/ICI exhibited a significant decrease in MGMT, ERα, ki-67 as compared with tumors treated with tamoxifen/ICI alone or control group. p53 expression was not much altered in these treatment groups. In sharp contrast, the expression of p21 was significantly increased in tumors from mice treated with BG either alone or in combination with tamoxifen/ICI. The images were analyzed by ImageJ (NIH) and MGMT, ERα, p53, p21 and ki-67 expressions were quantified by the ImmunoRatio plugin. (Fig.5).

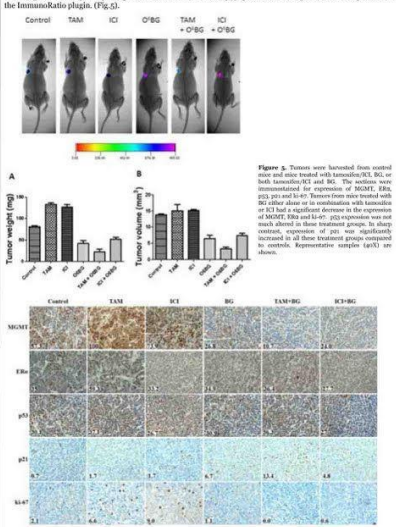


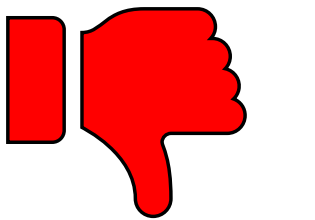
Figure 5. Tumors were harvested from control mice and mice treated with tamoxifen/ICI, BG, or both tamoxifen/ICI and BG. The sections were immunostained for expression of MGMT, ERα, p53, p21 and ki-67. Tumors from mice treated with BG either alone or in combination with tamoxifen or ICI had a significant decrease in the expression of MGMT, ERα and ki-67. p53 expression was not much altered in these treatment groups. In sharp contrast, expression of p21 was significantly increased in all these treatment groups compared to controls. Representative samples (40X) are shown.

Conclusions

1. In the present study, we observed that prolonged treatment with anti-estrogens causes drug resistance by inducing the DNA repair protein O⁶-methylguanine DNA methyltransferase (MGMT).
2. Decreasing the expression of MGMT by exposing breast cancer cells to BG sensitized these cells to anti-estrogen therapy (tamoxifen and ICI 182,780).
3. We also observed that combination therapy of anti-estrogens and MGMT blockers not only overcame the MGMT derived drug (tamoxifen and ICI) resistance but also increased the efficacy of anti-estrogen therapy by decreasing estrogen receptor expression and restoration of the functional activity of p53 in tamoxifen-resistant breast cancer cells.
4. Combination therapy inhibited tamoxifen resistant breast tumor growth *in vivo*.

Acknowledgements

We would like to thank the Florida Department of Health, Bankhead-Coley Cancer Research Program 09BN-01 for their funding of this project.



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Universitatis Scientia

Background

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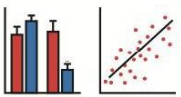

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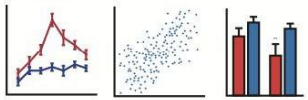

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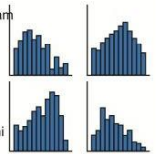

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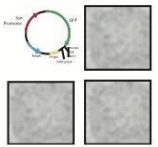

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Background

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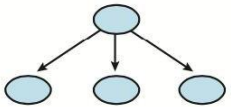

Hypothesis

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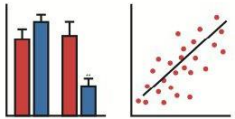

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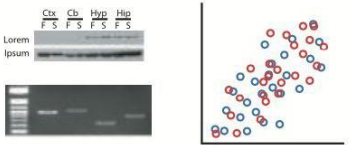

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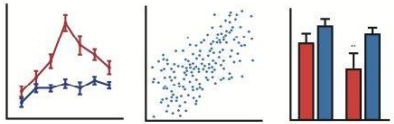

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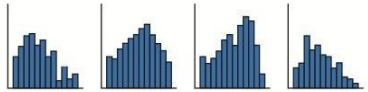

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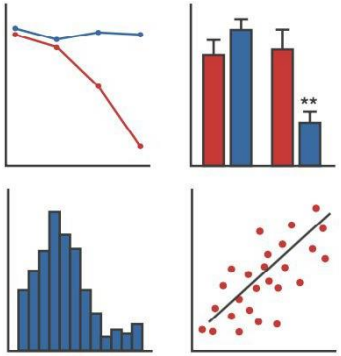
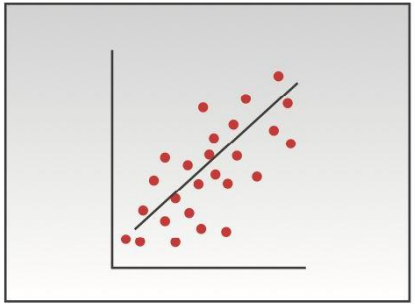
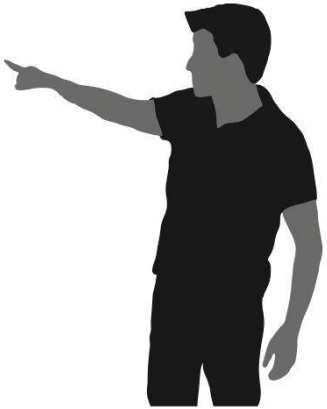
Software

- PowerPoint/Google Slides
- Adobe Illustrator
- Open-Source Alternatives
  - OpenOffice
  - Inkscape and Gimp
  - For charts and diagrams try Gliffy or Lovely Charts







Scientific Content

Your ideas, experiments, results, discussion, etc. Anything you want to communicate to an audience.

+

Visual Information

All of the visual aids you use to communicate information. In a paper, these are your figures; in a slide presentation, these are your slides; and in a poster presentation, this is your poster.

+

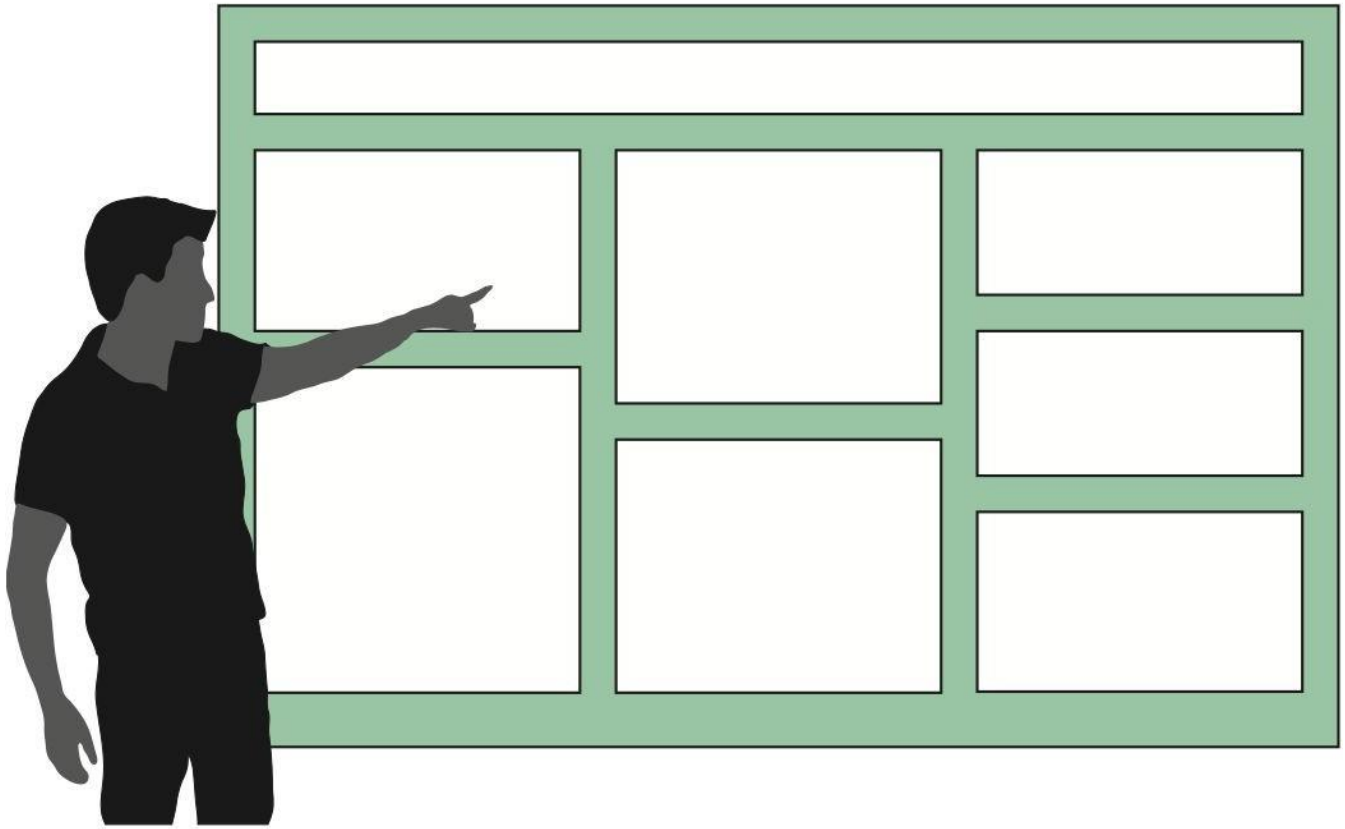
Delivery

Your narrative that leads the presentation of your visual information. In a paper, your narrative is written on the page. In a slide and poster presentation, you deliver your narrative orally and with nonverbal communication (body language).

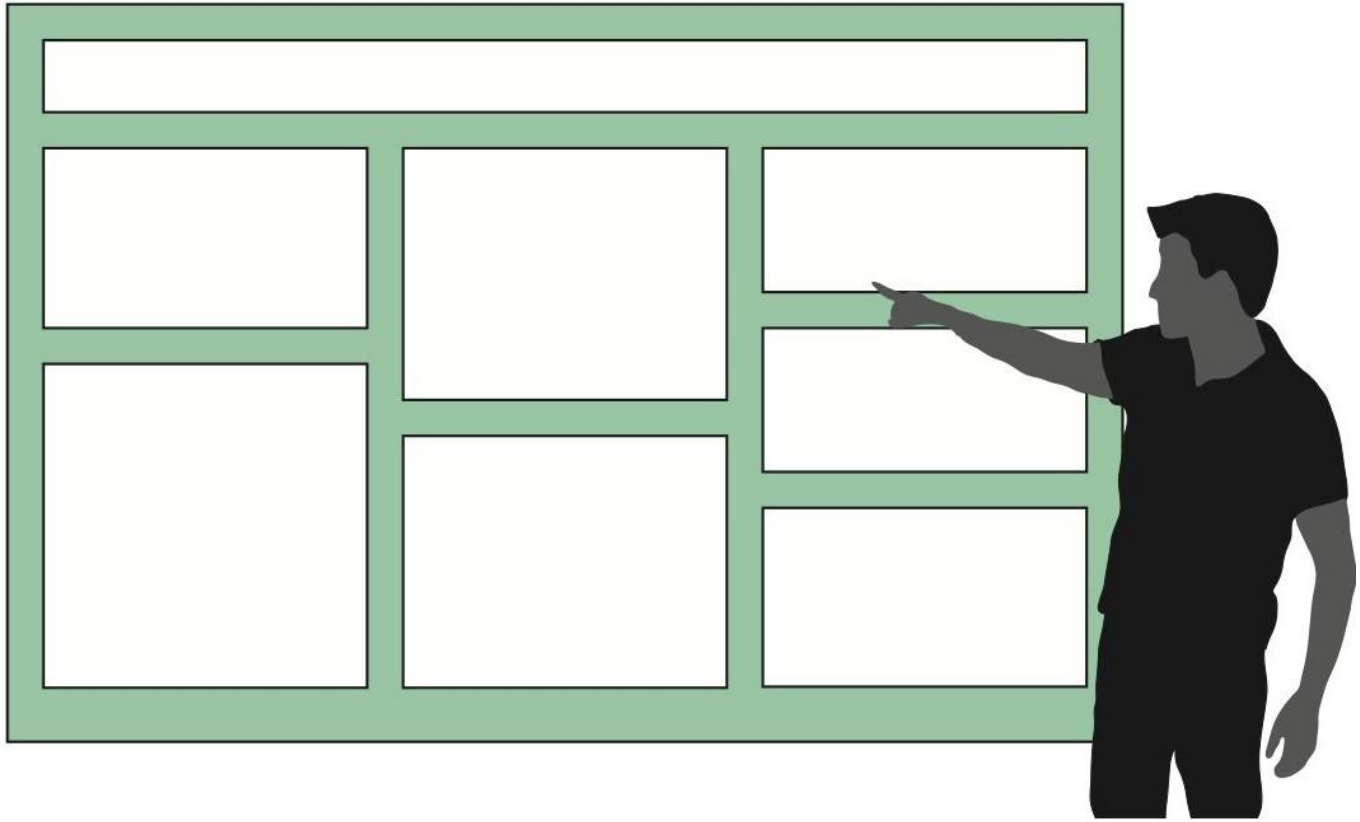
Presentation Tips

- Be present at your poster!
- Know your audience
- Point out visuals, but not text
- Consider supplementary information (e.g., a handout, tablet)
- Don’t block your poster
- Be professional
    - Dress, hygiene, body language

Always start a walkthrough by
standing just to the left of your poster.



When you are about halfway through,
completely cross to the other side.



Resources

Much of the material here from Matt Carter, *Designing Science Presentations* as well as Research Guides: How to Create a Research Poster: Design Tips

Designing conference posters —for advice & templates!

Powerpoint poster templates for research poster presentations

Scientific Poster PowerPoint Templates