Scientific Posters

(& Presenting Them)

Fall 2023

Presentation Macaulay TLC Fellow:

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With thanks to TLC Fellows Allison Cabana, Rakhee Kewada, Liza Shapiro & Frieda Benun for development of some included slides.

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MACAULAY HONORS COLLEGE

Agenda

1. Purpose of a poster
2. Technical aspects of making posters
3. Example Posters
4. How to present a poster
5. Questions (throughout)

What is the point of a poster?

- Short & concise explanation of your project and the conclusions
- Show your evidence/data/work in a clear and easy-to-read manner
- **Bring your work to a wider audience than those who would otherwise read it**

- *Bonus: Have an easily accessible example of your work if needed for future opportunities*

When & where you might present a poster?

- In your courses
- STEAM Festival
  - In-person /Online presentations
  - Website
- Professional Conference
  - Undergraduate/CUNY
  - Professional Organization
- Future Applications to your Careers

Different Things to Consider:

- Who is your audience?
- Are there any guidelines for your specific venue?
- Are you presenting it?
  - *If yes, how so (e.g. time, online, recorded, etc)?*

- What would YOU want to see in a poster?
  (This is part of your "style," *within guidelines*.)

Poster Types & Field Specificities:

- There are MANY ways you can make a poster, however - scientific posters generally fall into (or b/w) two main types:
  - **Conventional Scientific Poster** (Poster 1.0)
  - **Poster 2.0**

- For your professional field, there *may* be specific conventions that are widely used.
- Your professors may also have preferences.

Example of Conventional Poster:

Title, formatted in sentence case (Not Title Case and NOT ALL CAPS), that hints at an interesting issue and/or methodology, doesn't spill onto a third line (ideally), and isn't hot pink

Colin Purrington
666 Teipai Street, Posterville, PA 19801, USA

Introduction

Congratulations: a reader was mildly intrigued by your title. Now you have 2-3 sentences to hook him/her into reading more by describing what your question was and why the answer might be of general interest. Gratuitous background information will cause them to walk away (if you're standing next to your poster, that can be awkward).

Typography research has shown that body text is easier to read if you use a serif font such as Times. But non-serif fonts are great for title, headings, figure legends, etc. Research also shows that fully justified text (this paragraph) is slightly harder to read even though it looks really cool.

Figure 1. A photograph in your introduction can help lure people to your otherwise non-photogenic research. If it's not your image, ask photographer for permission to use, and cite him/her.

Materials and methods

Few people, if any, really want to know the gruesome details of what you've been up to, so be brief. Use lightly-annotated photographs, drawings, or flow charts to visually convey your general experimental approach. To better engage viewers in your protocol or system, try attaching actual objects such as study organism (dead specimen), research gizmo, photo flip book, or a short movie (attach an old smartphone with Velcro).

Figure 2. Hire an artist to illustrate the important step in your protocol. A photograph of you actually doing something might be nice, too. (Image by John Snow 1853)

Results

The overall layout in this areas should be visually compelling, with clear cues on how a reader should travel through the components. Be creative. You might want a large map with inset graphs, or have questions on left with answers and supporting graphs on right. Be sure to separate figures from other figures by generous use of white space. When figures are too cramped, viewers get confused about which figures to read first and which legend goes with which figure.

If you can add small drawings or icons to your figures, those visual cues can be priceless aids in orienting viewers. And use colored arrows or callouts to focus attention on important parts of graphs. You can even put text annotations next to arrows to tell reader what's going on that's interesting in relation to the how the hypothesis is being evaluated. E.g., "This outlier was most likely caused by contamination when I sneezed into tube." Also, don't be afraid of using colored connector lines to show how one part of a figure relates to another figure. These tips might induce gasps for published manuscript, but posters can be more personal and thus better guide viewers.

Figures are preferred but tables are sometimes unavoidable, like death. But go to great efforts to make it look professional. Look in a respected journal and emulate the layout, line types, line thickness, text alignment, etc., exactly. Again, use colored text or arrows to draw attention to important parts of the table.

Paragraph format is fine, but so are bullet lists of results:

- 9 out of 12 brainectomized rats survived
- (brainectomized rats are lost
- Control rats completed maze faster, on average, than rats without brains

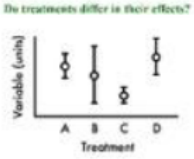

Figure 3. Legends can briefly describe the experiment, answer the question, and even include statistics if you so choose (unlike a manuscript figure legend).

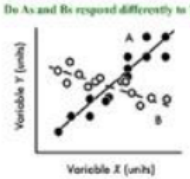

Figure 4. Label elements instead of relying on annoying keys that are default on most software. Add pictures of A and B if they are actually things (e.g., icons of rat with, without brain).

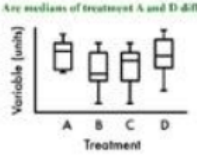

Figure 5. Don't be tempted to reduce font size in figure legends, axes labels, etc. This is because viewers are probably most interested in reading your figures and legends.

Conclusions

Conclusions should not be dry restatements of your results. You want to guide the reader through what you have concluded from results, and you need to state why those conclusions are interesting (i.e., don't assume reader will guess). These first several sentences should refer back to the burning issue mentioned in the introduction. If you didn't mention a burning issue in the introduction, go back and fix that.

A good conclusion will also explain how your conclusions fit into the literature on the topic. E.g., how exactly does your research add to what is already published on the topic? It's important to be humble and generous in this section, partly because authors of previous literature may still be alive and even attending the conference. You can also display your appreciation of others' input by citing conversations you have had (with pers comms).

Finally, you want to tell readers who have lasted this long what might be done next and who should do it. E.g., are you currently taking the next logical step, or should another person with different skills follow up on your amazing result? It's OK to put a bit of personality into this ending because viewers expect posters to be personal (and if you're not actually standing there to convey your enthusiasm, your poster text should be doing that *for* you).

If you have a graphical way to express the next step of your hypothesis, by all means include it in this section. For example, you might make a graph with hypothetical data that shows an expected result in a future experiment. That's something you normally don't show in a traditional manuscript, but it's totally fine for a poster.

If you're curious, this poster has 683 words. Aim for 500 words. If you are above 1000 words, your poster will be annoyingly long to everyone except your mentor or colleague.

A well designed poster retains plenty of white space separating edges of text boxes, graphics, and tables. You also want space between your text and edge of box. Without white space a poster will looked cramped and uninviting.

Literature cited

Bender, D.J., E.M. Brayne, and R.M. Brigham. 1996. Lunar condition influences coyote (*Canis latrans*) howling. *American Midland Naturalist* 136:413-417.

Brooks, L.D. 1988. The evolution of recombination rates. Pages 87-105 in *The Evolution of Sex*, edited by R.E. Michod and B.R. Levin. Sinauer, Sunderland, MA.

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Acknowledgments

We thank J. Giez for laboratory assistance, Mary Jones for seeds, and Herb Iunde for greenhouse care. Funding for this project was provided by the Department of Thinkology. Note that people's titles are omitted (titles are TMI).

Further information

More tips (and templates) can be found at "Designing conference posters":

http://colinpurrington.com/tips/poster-design

Example of Poster 2.0:

https://www.insidehighered.com/news/2019/06/24/theres-movement-better-scientific-posters-are-they-really-better

Technical Aspects of Making a Poster

YOUR POSTER SHOULD ANSWER…

My name
My place

The Effect of X on Y in NYC

Why?

What are we adding?

What did we do?

What did we find?

What did we learn?

What do we recommend?

What to Include:

- FULL Name & Contact Info (i.e., email)
- University and/or Organizational Affiliation
  - Logos, if Applicable

SCIENTIFIC CONTENT:

- Intro (&/or Abstract)
- Methods
- Data
- Conclusions
- References & Acknowledgments*

REQUIREMENTS

- Posters can be made with **Keynote**, **PowerPoint** or **Google Slides**.
  - Beware of conversion between PowerPoint and Google Slides!

- Poster dimensions must be 48” x 36” (or vice versa)
  - In PowerPoint/Gslides:
    - File > Page setup > Width= 48, Height=36

- You can also find templates in PowerPoint for scientific posters.

DESIGN GUIDELINES

- Fonts: legible, accessible, and simple
- Headings: 40-70 pt size
- Body: 24+ pt size
- Simple color scheme, minimal text, avoid paragraphs Graphics
  - Bullet points and indentation
    - As you can tell from reading this, no one likes to read large swaths of text in a bright color. Even a sentence or two in the same same bullet point can be visually unappealing, so when in doubt: split it into 2 bullets. And/or condense!

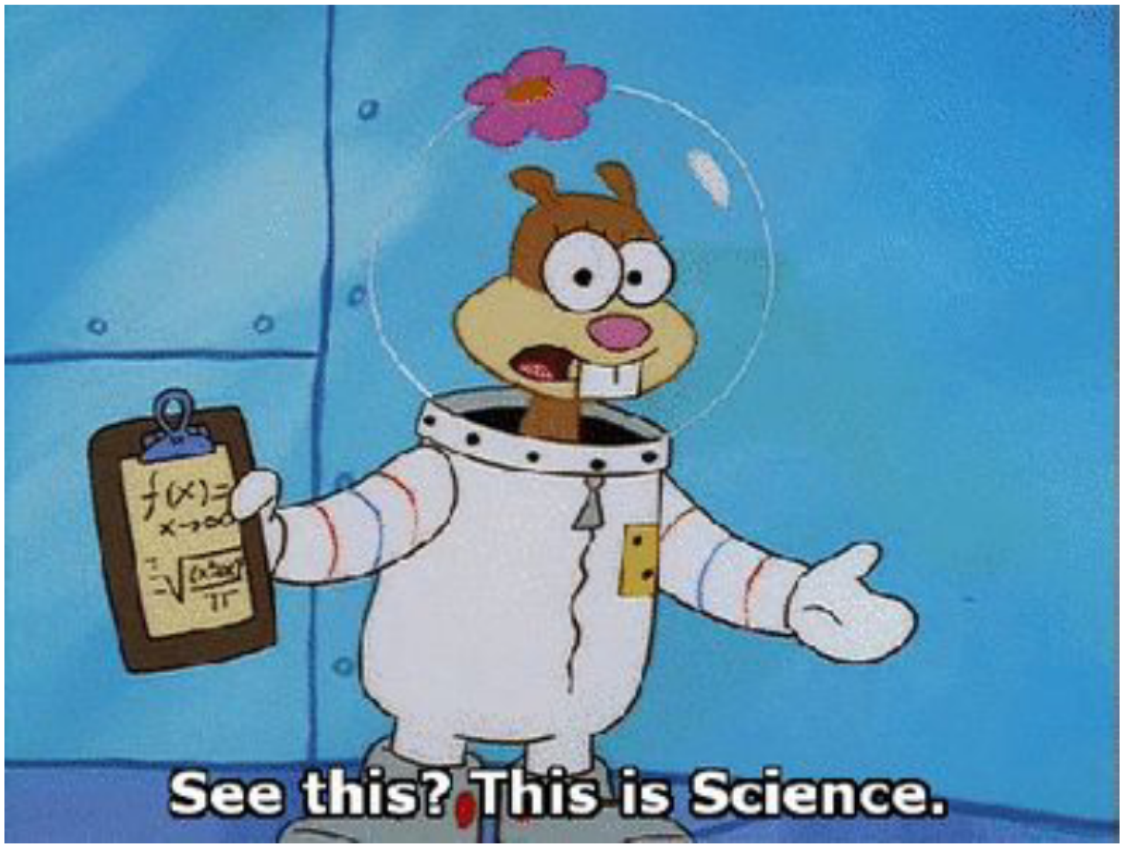
DESIGN GUIDELINES - NOTE on POSTER 2.0

- These might change depending on if you're going with a Conventional Scientific Poster *OR* Poster 2.0!

- **For Poster 2.0:**
  - Main finding is the largest, most focal piece.
  - Solid colors, bold statement.
  - Inclusion of a QR code to link to details about the study.
  - *Note that you are still including the scientific evidence on the poster, it is just less focal than in the conventional poster.*

WHAT MAKES A GOOD POSTER?

Regardless of 1.0 or 2.0



WHAT MAKES A GOOD POSTER?

Regardless of 1.0 or 2.0

- Clarity and legibility
- Succinct statement of your claim
- Evidence of your claim: Method, Data
- SIMPLE graphics and/or organization

- A way to seek more info

Examples of Posters:
Pros & cons?

SAMPLE POSTER 1:
HTTP://WEBVISION.MED.UTAH.EDU/2014/05/THE-ROLE-OF-NMDA-RECEPTOR-ACTIVITY-IN-RETINAL-GANGLION-CELL-DENDRITE-DEVELOPMENT/

3517 · D0117

The Role of NMDA Receptor Activity in Retinal Ganglion Cell Dendrite Development

Eerik M. Elias[1, 2], Ping Wang[2], and Ning Tian[1, 2]

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2. Department of Ophthalmology and Visual Science, University of Utah School of Medicine, Salt Lake City, UT, United States.

INTERDEPARTMENTAL PROGRAM IN NEUROSCIENCE, THE UNIVERSITY OF UTAH

RPB

UNIVERSITY OF UTAH HEALTH CARE, Moran Eye Center

Introduction

Background- *Retinal ganglion cell dendrites undergo dramatic structural changes during development as they grow out into the inner plexiform layer, stratify, and form synapses with bipolar and amacrine cells*

Problem- *Conflicting studies cannot agree whether or not retinal ganglion cell dendritic development is activity dependent*

Hypothesis- *Activity-dependent dendritic development depends on ganglion cell subtype. We predict that the refining RGC subtype, JamB, requires glutamatergic activity for dendrite development*

Methods

- *Model: JamB-CreER:YFP mice label OFF DS ganglion cell*
- *Imaging: confocal microscopy of WT and Grin1$^{-/-}$ JamB-CreER:YFP retinal whole-mounts*
- *Experimental treatments: 400 uM AP5 and 40 uM CNQX were injected intraocularly to block spontaneous glutamatergic activity before eye-opening. Dark reared mice were housed in a dark box from P6 to P30.*
- *Analysis: Individual neurons were traced using Neurolucida and analyzed with NeuroExplorer to measure dendrite length, protrusion number, and dendritic field area.*

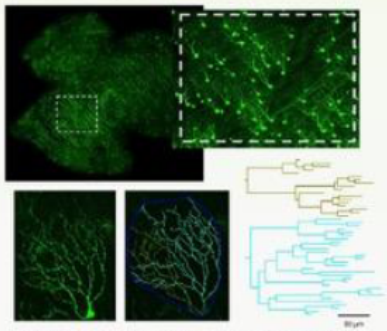

Figure 1. Imaging and dendrite tracing procedure of JamB RGCs. Dendrites are tracing in cyan and olive, dendritic field in blue.

Results

JamB RGC dendritic development occurs in two phases

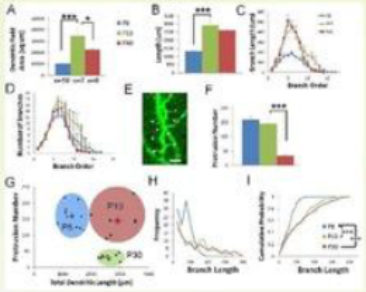

Figure 2. JamB RGC dendrites lengthen and eliminate protrusions during two distinct postnatal time periods. (A) Average dendritic field (DF) area of JamB RGCs at P5, P13, and P30. (B) Average dendritic length of the same three groups of JamB RGCs. (C) Branch length as a function of branch order of the same three groups of JamB RGCs. (D) Number of branches as a function of branch order. (E) Confocal image of a P5 GFP-labeled JamB RGC showing numerous protrusions/filopodia along dendrites. Scale 6.5 um. (F) Number of protrusions per neuron. (G) Scatter plot of protrusion number versus total dendritic length. (H) Histogram of dendritic branch lengths. (I) Branch length cumulative probabilities of the three groups of JamB RGCs. Values are mean ± s.e.m.

Dendritic expansion requires glutamatergic activity

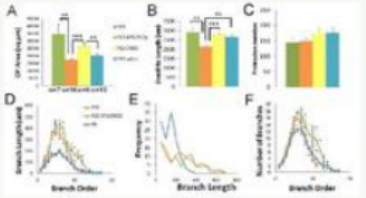

Figure 3. Blockade of synaptic activity mediated by glutamate receptor (GluR) selectively impairs the dendritic elongation of JamB RGCs. (A) Average DF area of P13 JamB RGCs treated and untreated with GluR blockers. (B) Average dendritic length of the same four groups of P13 JamB RGCs. (C) Average number of protrusions per neuron. (D) Branch length as a function of dendrite branch order. (E) Histogram of individual branch lengths. (F) Branch number as a function of branch order.

Results

NMDA receptors drive dendritic expansion

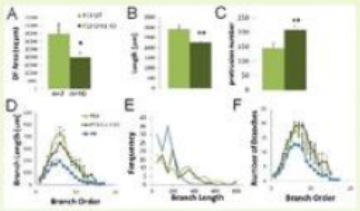

Figure 4. NMDA receptors are required for JamB dendritic expansion. (A) Average DF area of JamB RGCs of wild type (WT) mice and mice with conditional knockout of Nf12 expression by JamB RGCs (Grin1 KO) at the age of P13. (B) Average total length of dendrites of WT and Grin1$^{-/-}$ JamB RGCs. (C) Average protrusion number per neuron. (D) Average branch length as a function of branch order. (E) Histogram of individual branch lengths. (F) Branch number as a function of branch order.

Dendritic field consolidation is absent in NMDAR KO JamB RGCs

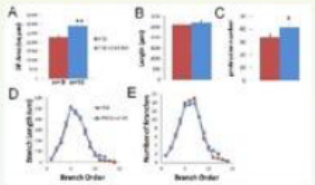

Figure 5. NMDA KO JamB RGCs lack dendritic field consolidation. (A) Average DF area of JamB RGCs of WT and Grin1 KO mice at the age of P30. (B) Average total length of dendrites of the same two groups of JamB RGCs. (C) Average total protrusion number per neuron. (D) Average branch length as a function of branch order. (E) Average number of branches as a function of branch order.

JamB CreER:YFP mice were kindly provided by Dr. Joshua Sanes at Harvard University

National Eye Institute, National Institutes of Health

Funding Source: NIH NEI 2R01EY012345

Visual inputs drive dendritic field consolidation

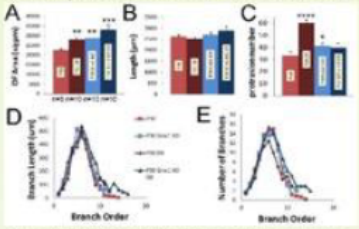

Figure 6. JamB-CreER:YFP and Grin1$^{-/-}$;JamB-CreER:YFP mice were raised in constant darkness from P6 to P30 and the dendritic structure of JamB RGCs of these mice were characterized and compared with the same two strains of mice raised under cyclic light/dark conditions. (A) Average DF area of JamB RGCs of four different groups. (B) Average total dendritic length of the same four groups of RGCs. (C) Number of protrusions per neuron. (D) Branch length as a function of branch order. (E) Number of branches as a function of branch order. Asterisks are comparison to P30 control.

Summary

- *JamB RGC dendritic development occurs in two phases, (1) dendrite elongation and field expansion and (2) elimination of dendritic protrusions and DF consolidation*
- *Dendritic elongation requires glutamatergic activity in the retina*
- *NMDA receptors on JamB RGCs are required for protrusion elimination, dendrite elongation, and DF expansion before eye-opening*
- *NMDA receptors may be required for DF consolidation*
- *Visual input drives DF consolidation*

References

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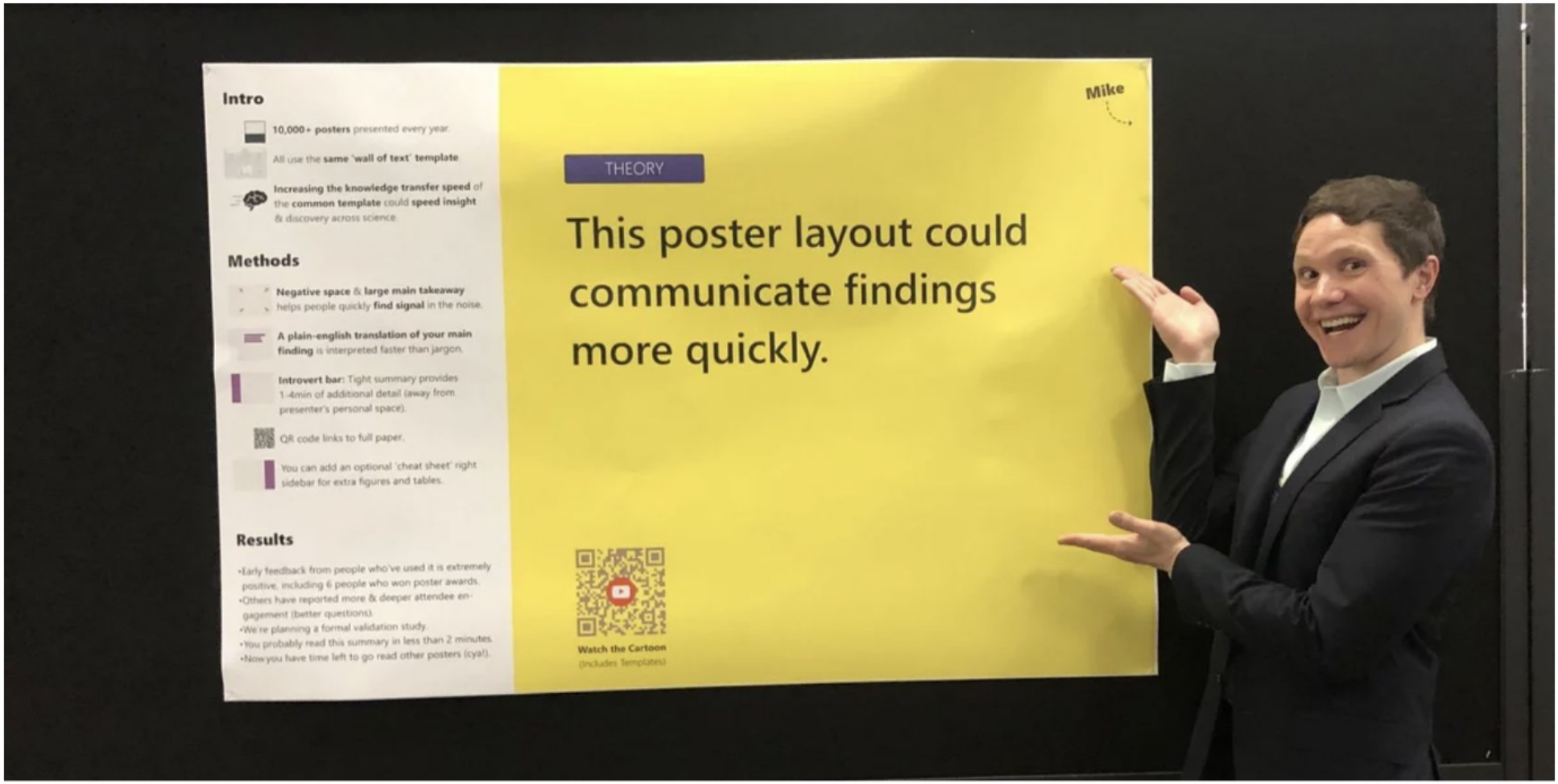
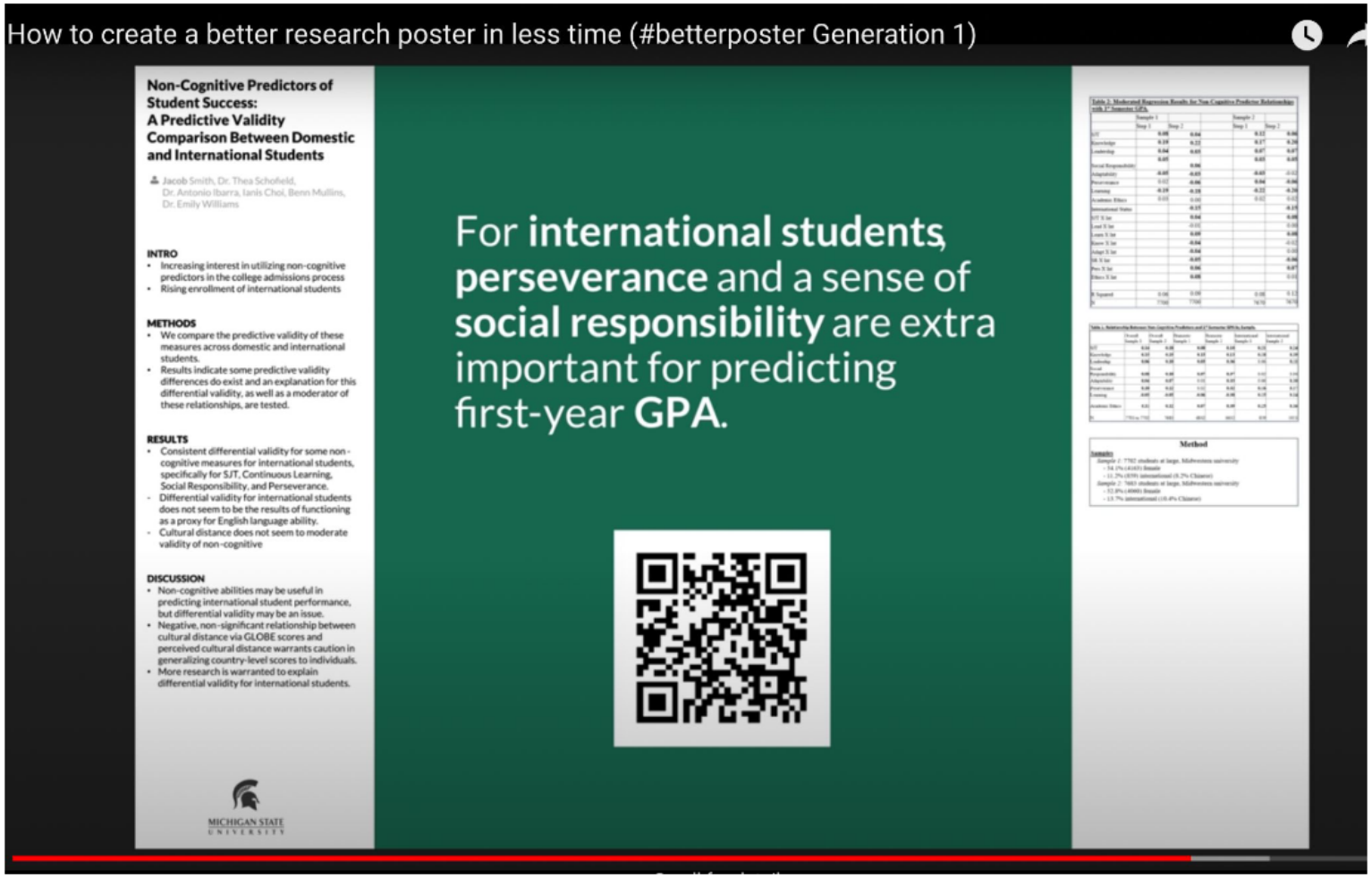
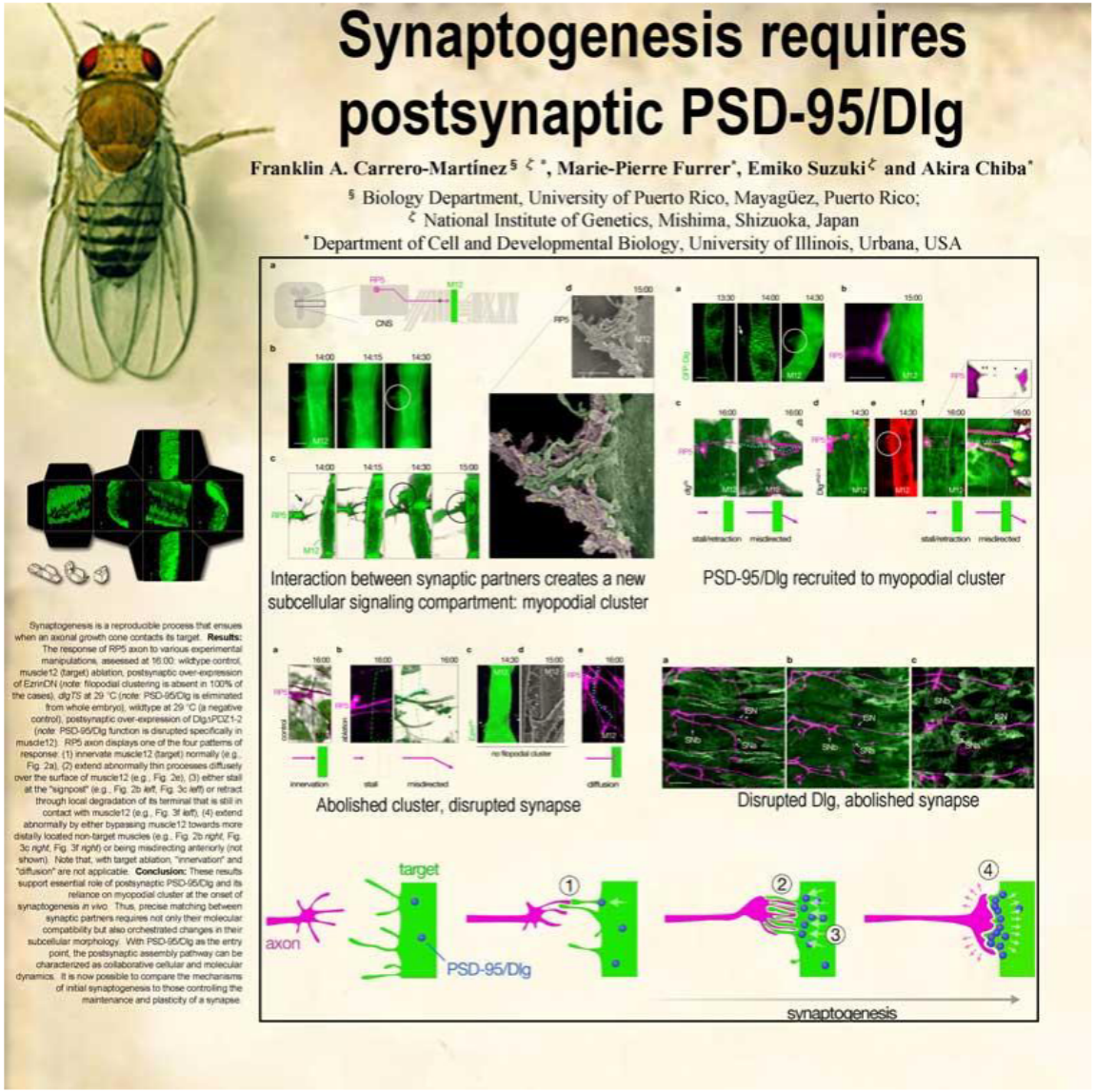
SAMPLE POSTER 2:
https://www.npr.org/sections/health-shots/2019/06/11/729314248/to-save-the-science-poster-researchers-want-to-kill-it-and-start-over



SAMPLE POSTER 3:
Still from - https://www.youtube.com/watch?v=1RwJbhkCA58



SAMPLE POSTER 4



How much of an Impact do Ginkgo Trees have on Air Quality?

BioBlitz Poster
Vadim Allayev, Kristina Andrade, Zae Jackson, Shelly Koppel, Lily Negroponte

Background

Ginkgo trees are impressively resilient plants. Research shows that ginkgo trees have a long lifespan of more than a thousand years, as well as strong resistances to drought, diseases, pollution, and poor soil. In the past there have been several programs to plant ginkgo trees in an effort to improve air quality. Curious about the effectiveness of ginkgo trees in this respect, we have done research to see if they really do effect.

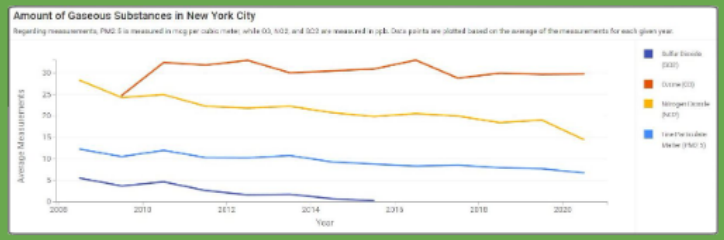
Research Question

How much of an impact do ginkgo trees have on air quality?

Methods

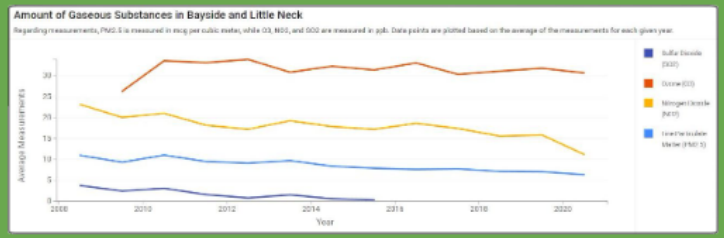
We compared the measurements of average levels of sulfur dioxide, nitrogen dioxide, and fine particulate matter in the air over time in three separate areas (taken from NYC Open Data) to the number of ginkgo trees in those areas (recorded by NYC Street Tree Map). Since sulfur dioxide and fine particulate matter are particularly good indicators of air pollution, we used these measurements as proxies to determine the relation between ginkgo trees and air pollution.

Results



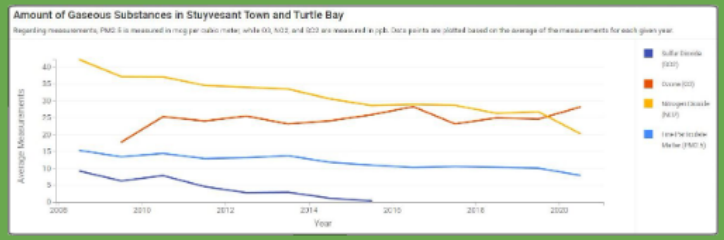
NYC
691,600 Ginkgo Trees

Fig.1



Bayside & Little Neck
22,947 Ginkgo Trees

Fig.2



Stuyvesant Town & Turtle Bay
2,585 Ginkgo Trees

Fig. 3

Analysis

Despite having about 9 times the ginkgo trees in the regions of Fig. 3, the regions of Fig. 2 does not see considerably steeper decreases in pollutants than that of Fig.3. Moreover, the trends of Fig. 2 and 3 follow those of Fig. 1, which represents all of NYC.

Conclusions

Our data suggests that ginkgo trees do not significantly impact NYC air quality. However, it is possible that ginkgo trees impacted the initial measurements of these gaseous substances. Many ginkgo trees in NYC were planted since the mid-1900s, and we can see a clear difference in the initial measurements at the start of Fig. 2 (in 2009) compared to Fig. 3. Still, proximity to various pollutants or green spaces and environmental/energy regulations likely play a more significant role in air quality.

References

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2. Hwang, Yook, S.-J., & Ahn, K.-H. (2011). **Experimental investigation of submicron and ultrafine soot particle removal by tree leaves.** *Atmospheric Environment (1994)*, 45(38), 6987–6994. (1994).
3. Kiyomizu, T., Yamagishi, S., Kume, A. et al. **Contrasting photosynthetic responses to ambient air pollution between the urban shrub Rhododendron × pulchrum and urban tall tree Ginkgo biloba in Kyoto city: stomatal and leaf mesophyll morpho-anatomies are key traits.** *Trees 33*, 63–77 (2019).
4. Bo Chen, Jingjing Xu, Donghuan Liu, Xinbing Yang, **Response of Ginkgo biloba growth and physiological traits to ozone stress**. *Global Ecology and Conservation, Volume 34, 2022*, e02020, ISSN 2351-9894, (2022).

This poster was made using the "Presenter's Poster" template from PeerRecognized.com

SAMPLE POSTER 5:
https://nabuy.deviantart.com/art/scientific-poster-186612994

SAMPLE Poster 6:

Source - "Better Posters"
http://betterposters.blogspot.com/2011/04/critique-breast-cancer-inhibition.html

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 Tieku[1], Kalkunte S. Srivenugopal and Santhi Konduri[1]

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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 [O⁶-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 tamoxifen alone and fulvestrant alone increased and decreased the same respectively. However, all these treatments increased the p21^{WAF1} 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^{WAF1} 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 O⁶ position on guanine, forming mutagenic and highly cytotoxic interstrand DNA crosslinks. The DNA repair enzyme O⁶-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 O⁶ 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 [25]. 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 cancer. 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 dodge 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 a 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.

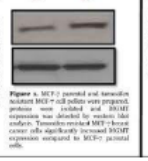

Figure 1. MCF-7 parental and tamoxifen resistant MCF-7 cell pellets were prepared, proteins were isolated and MGMT expression was detected by western blot analysis. Tamoxifen resistant MCF-7 breast cancer cells significantly increased MGMT expression compared to MCF-7 parental cells.

O⁶-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.

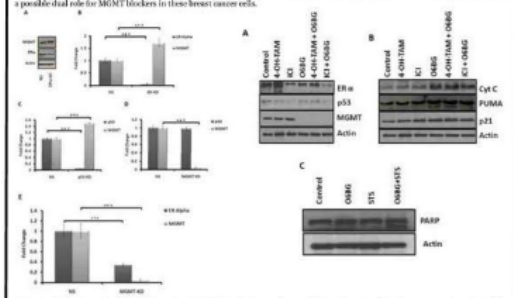

Figure 2. (A) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100nM) (NS), and cells were harvested 72h post transfection. Total proteins were isolated and ERα and MGMT expression was determined by western blot analysis. MGMT protein was significantly increased in ERα knock down cells (B) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100-nM) (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 (50 nM) 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 (Fig.2 D).

Figure 3. (A) Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 nM) and 4OH tamoxifen (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 expression (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). 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^{WAF1} 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 was also studied. 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).

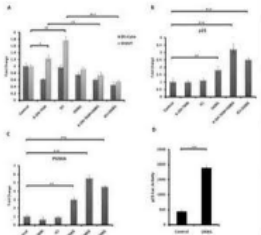

Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 nM) for 48h and later 4-OH tamoxifen and ICI (1μM) was either alone or in combination with BG and 24h later cells were harvested and total RNA was isolated. (A) MGMT and ERα (B) p21 transcription (C) PUMA transcription was determined by qRT-PCR. 4-OH tamoxifen and ICI induce MGMT transcription. BG enhanced 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 mm^3, 9.33 mm^3) (TAM+BG), respectively; p< 0.0000) (83.99 mm^3, 31.60 mm^3 (ICI+BG), respectively; p<0.0000). Tumor weight was also significantly reduced in mice treated with combination therapy as compared with control mice (81.33 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).

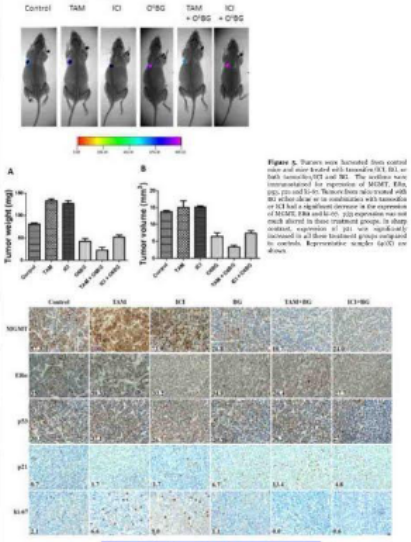

Figure 5. Tumors were harvested from control mice and mice treated with tamoxifen (0.1 ml, s.c. both tamoxifen/ICI and BG. The necrosis was immunostained for expression of MGMT, ERα, p53, p21 and Ki-67. Tumors from mice treated with BG either alone or in combination with tamoxifen or ICI had a significant 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 (n=3) 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

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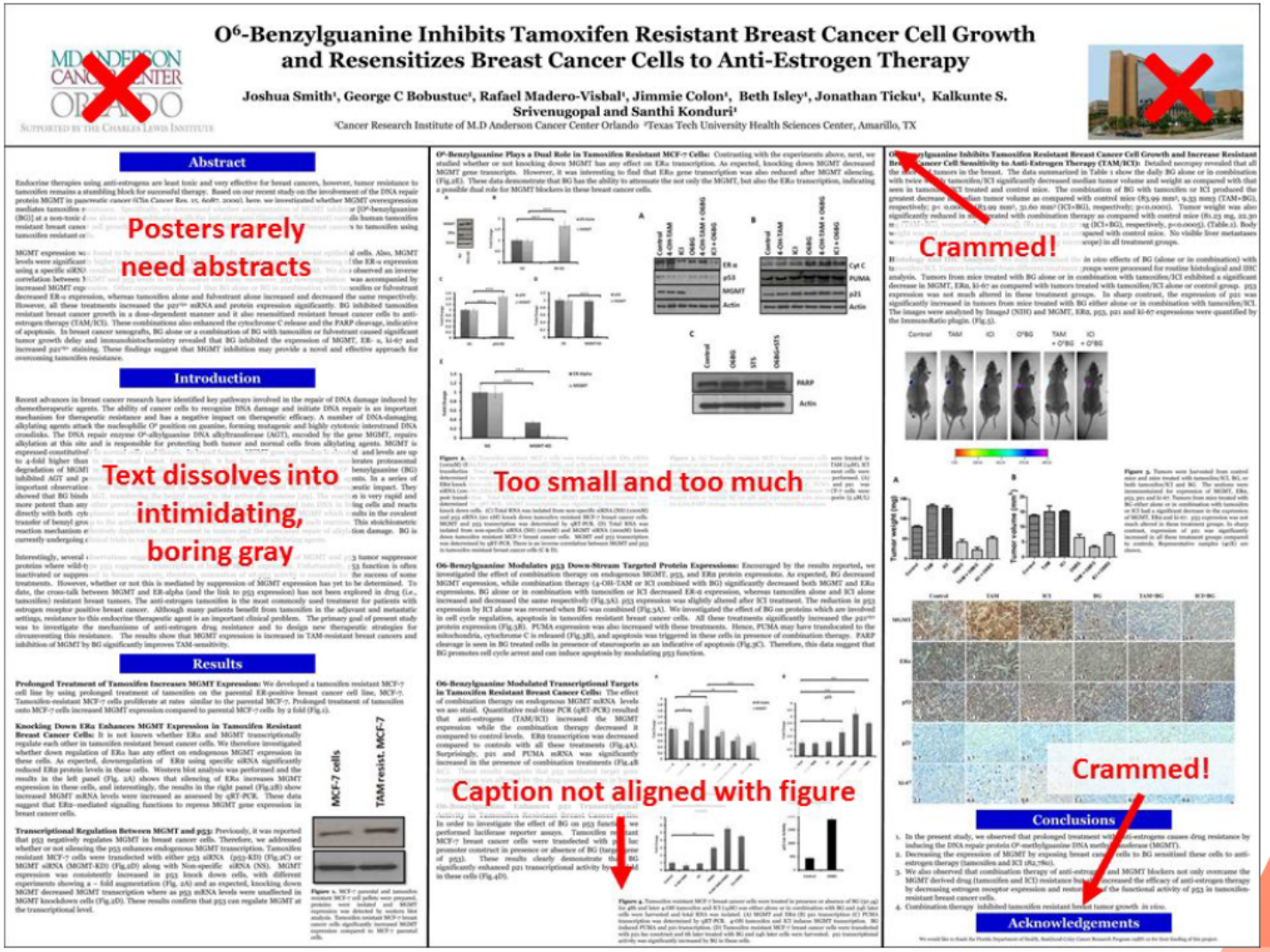
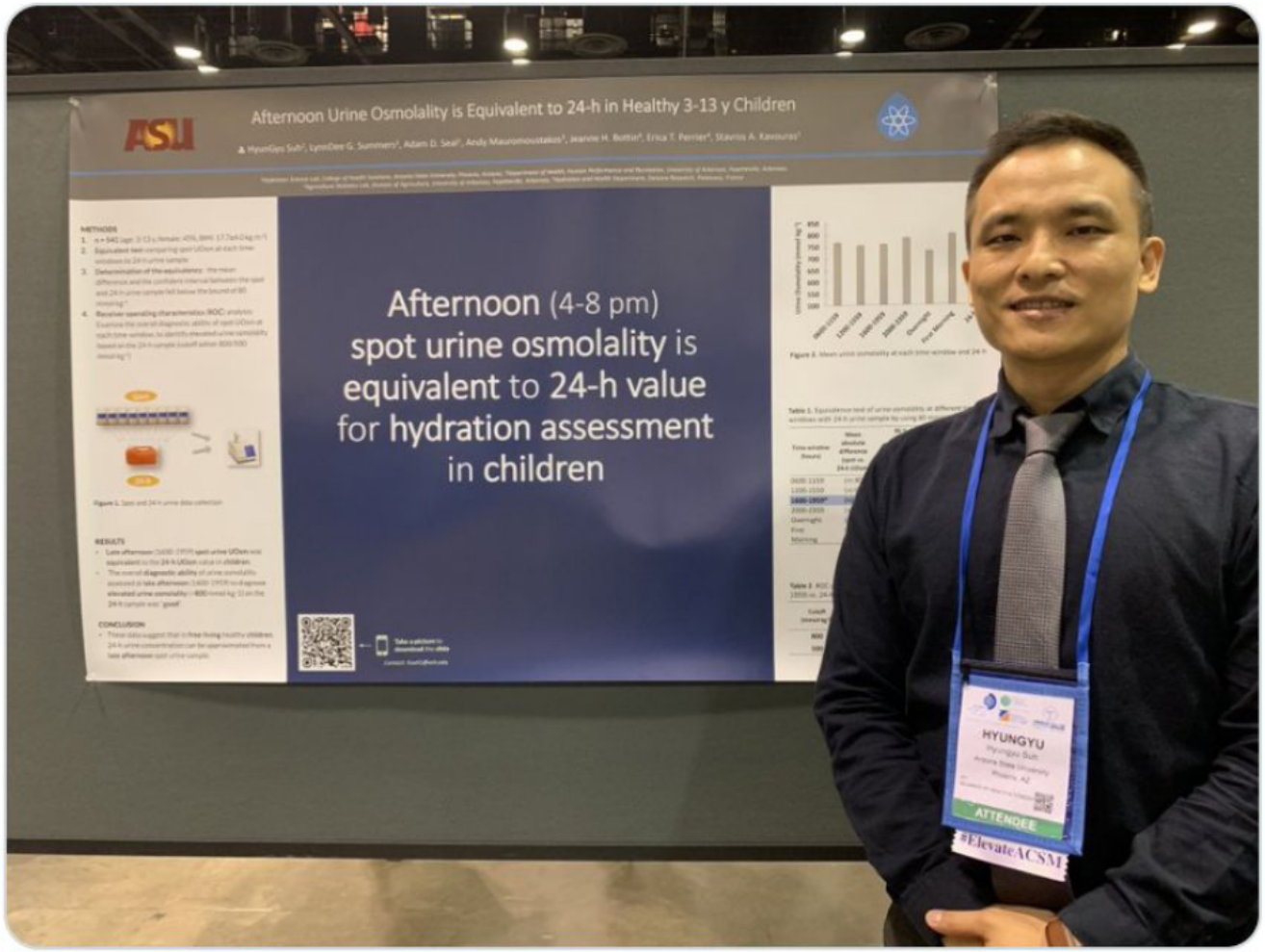
Source: http://betterposters.blogspot.com/2011/04/critique-breast-cancer-inhibition.html



Source: http://betterposters.blogspot.com/2011/04/critique-breast-cancer-inhibition.html

Poster Example 7:



https://twitter.com/DrHydration/status/1134193977572372480/photo/1

Additional Resources:

- NYU Guide to Posters: https://guides.nyu.edu/posters
- Article with Links and Examples from UC Davis: https://urc.ucdavis.edu/creating-effective-academic-posters
- Article Explaining Scientific Poster 2.0, with Examples: https://www.npr.org/sections/health-shots/2019/06/11/729314248/to-save-the-science-poster-researchers-want-to-kill-it-and-start-over
- Inside Higher Ed, Poster 2.0: https://www.insidehighered.com/news/2019/06/24/theres-movement-better-scientific-posters-are-they-really-better

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In-Person or Online

TIPS for Presenting:

- Plan
  - If this is with a group - be CLEAR on who does what. (note on STEAM)
- Practice!
- Slow down and **trust yourself**.
  - Your poster is there to back you up, but YOU know the research and the narrative behind the work.


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Hydration and Physical Health
Stories Beyond the Stones


EXIT


NO SMOKING
Access Ramp
MACAULAY
STEAM
Make your project

Things to Consider:

- Impact & Memorability
  - Storytelling
  - Emphasizing take-aways
- Audience
  - Consider questions an audience may ask
- Accessibility
  - **QR code / link to content audience can refer to in real time or later**

+ TLC Support

- Schedule an Appointment with a TLC Member: https://eportfolios.macaulay.cuny.edu/tlc/tlc-support-scheduler/