



RECOGNIZING OUTSTANDING UNDERGRADUATE RESEARCH

2019 Research Day at the Capitol

STUDENT ORIENTATION SESSION





2019 Research Day at the Capitol

**Congratulations for being selected
to represent your institution
at the**

**24th Annual
Research Day at the Capitol!**

Event Sponsors:

Oklahoma NSF EPSCoR

The National Science Foundation

Oklahoma State Regents for Higher Education





What is Research Day at the Capitol?

- ❖ Annual event, sponsored by:
 - Oklahoma State Regents for Higher Education
 - The National Science Foundation (NSF)
 - Oklahoma Established Program to Stimulate Competitive Research (OK NSF EPSCoR)

- ❖ To celebrate the excellent undergraduate student research being conducted on Oklahoma's college and university campuses

- ❖ A chance to inform Legislators and the public about undergraduate student research



2019 Research Day at the Capitol

YOUR STIPEND FUNDING

You can expect delivery of your \$250 stipend check within approximately 3-4 weeks. Contact me if you haven't received it by Jan. 7, so my office can put a trace on the check.

- Funds are to cover your travel to/from OKC and for costs related to your poster printing & display (easel, board, etc.).
- Your check must be mailed to your permanent address (as indicated on your nomination form) per State guidelines.
- Checks will be issued from “**OKLAHOMA STATE UNIVERSITY**,” NOT “EPSCoR.”
- OSU students' checks will be processed through the OSU Bursar's Office.



2019 Research Day at the Capitol

TWO DAYS OF ACTIVITIES – MARCH 25 & 26

March 25 (Hyatt Place Hotel)

- Formal judging: Poster & oral presentations

March 26 (State Capitol Building)

- Posters presented, 4th Fl. Capitol Rotunda
- Awards ceremony, 2nd Fl. Capitol Rotunda

Students must participate in all activities to retain the \$250 stipend and qualify for prizes.



2019 Research Day at the Capitol

CASH PRIZES: FOR THE TOP 7 PRESENTERS

The following prizes will be awarded:

Grand Prize: \$500 cash prize + \$4,000 summer research internship
\$2,500 award to the sponsoring college/university lab
to offset expenses of hosting the internship

1st, 2nd and 3rd Place Prizes will be awarded in each of two
categories: Regional/community colleges (3 awards/1st, 2nd, 3rd)
 Research-intensive campuses (3 awards/1st, 2nd, 3rd)

1st Place: \$500 cash prize (1 ea: regional & research-intensive)
2nd Place: \$250 cash prize (1 ea: regional & research-intensive)
3rd Place: \$250 cash prize (1 ea: regional & research-intensive)



2019 Research Day at the Capitol

MONDAY, MARCH 25: HOW YOU ARE JUDGED

- Panel of 4 judges
- WELL educated, but not necessarily experts in your field of study

You will be judged on the following:

1. Poster
2. Abstract
3. 3-min. oral presentation before judging panel
4. Oral responses to judges' follow-up questions



2019 Research Day at the Capitol

MONDAY, MARCH 25: HOW YOU ARE JUDGED

The following judging criteria are used, with a 1-10 point scale for each item:

- **Abstract**
Format, clarity, societal impact, objective of study, results, conclusions.
- **Scientific presentation**
Purpose, hypothesis, background information, results, impact, further study expected
- **Student's ability to explain the project**
- **Visual appearance**
- **Clarity for general audiences**
- **Societal impact statement**
- **Overall**



2019 Research Day at the Capitol

ABSTRACT: REVISION DEADLINE FEBRUARY 4, 8 A.M.

Judges will score your abstract as part of your cumulative score. If you wish to alter or edit the abstract that was originally submitted, you must submit your final, revised abstract in MS Word format prior to February 4th at 8 a.m.

- MS Word format, no PDFs accepted (template provided)
- Maximum 350 words
- If images are used they will detract from the available word count; image files must be submitted separately (not only embedded in the document).
- **INCLUDE RESULTS + SOCIETAL IMPACT**
- Avoid excessive scientific jargon, but don't oversimplify
- Must be the work of the student
- See the provided sample judging sheet for scoring criteria
- Be sure you receive a confirmation of receipt from me

Abstract of a Past Winner

Introduction: Cisplatin is widely used as a chemotherapy drug that induces DNA damage and ultimately causes cell death. However, therapeutic resistance and tumor relapse remains a significant clinical problem. Recently, our laboratory developed an assay called PADDA (primer anchored DNA damage detection assay) that screens genomic areas for DNA damage. We hypothesized that PADDA will measure the ability of cancer cells to repair damage induced by cisplatin, and therefore predict cancer response to cisplatin.

Aims: To define the levels of DNA damage in a human gene (p53) induced by cisplatin treatment, and to measure the ability of cancer cells to repair DNA damage induced by cisplatin.

Methods: Squamous cell carcinoma cells (SCC-1) were exposed to 50 μ M, 10 μ M, 1 μ M, and 0.1 μ M concentrations of cisplatin for 3 hours, and allowed to repair DNA damage for 0, 3, 9, 18, 24 and 48 hours. PADDA was used to quantify DNA damage in the p53 gene at diverse time points. Cell viability was determined by MTT. Data was analyzed by Student's t-test.

Results: We observed high levels of DNA damage in both the transcribed and non-transcribed strands of p53 for all concentrations of cisplatin tested. DNA damage induced by low doses of cisplatin was quickly repaired and cell viability was preserved. DNA damage induced by high doses of cisplatin was not significantly repaired and caused significant cell death. Cells that survived high doses of cisplatin recovered within 24 hours and continued to grow despite retaining high levels of DNA damage.

Conclusions: We established for the first time that PADDA is highly sensitive in detecting cisplatin-induced DNA damage. By measuring DNA damage and cell viability, we found that the SCC-1 cell line is resistant to cisplatin because it is able to repair DNA damage induced by low doses of cisplatin and remain viable even in the presence of high levels of DNA damage. Studies with patient samples are on-going in our laboratory. This data shows that PADDA can be used as a screening method to identify patients resistant to cisplatin treatment.

Societal Impact: This observation has significant clinical importance as it can be used to predict treatment response and direct treatment selection in cancer patients. The use of this assay in cancer patients will reduce health care expenses and ultimately save the lives of patients.

This work was supported by the Oklahoma Tobacco Research Center (LQ), the OUHSC Vice President for Research Fund (LQ) & the Oklahoma Center for the Advancement of Science and Technology (LQ).

Abstract of a Past Winner

Introduction: The P27Kip1 tumor suppressor is commonly disrupted in aggressive human cancers, which is thought to cause inappropriate proliferation. However, recent evidence comparing mouse fibroblast cells with (p27+/+) and without (p27-/-) the p27 gene suggested it might also play a role in cancer cell metabolism. A time course of cell metabolism indicated p27+/+ cells relied mainly on glucose, while p27-/- cells switched to another source of energy after a short time period. Since the amino acid glutamine is a major potential source of carbon in the cell media, our goal was to determine if p27-/- cells were switching from glucose to glutamine.

Methods: P27+/+ and p27-/- cells were cultured in standard DMEM media containing 10% fetal bovine serum and allowed to reach confluency. Cells were re-fed with fresh media containing glucose and glutamine. Media samples were collected over time and then analyzed for glutamine content. The two part assay involved deaminating L-glutamine to L-glutamate, then L-glutamate conversion to α -ketoglutarate in a reaction that also converts NAD⁺ to NADH which can be measured with a spectrophotometer at 340nm, and is proportional to the amount of L-glutamine.

Results: Glutamine levels initially slowly decreased at similar rates in the p27+/+ and p27-/- cells. However, at later times p27+/+ cells used less glutamine while its levels continued to decrease dramatically in the p27-/- cells.

Conclusion: Data was consistent with the existence of a metabolic change occurring in p27-/- cells involving switching from glucose to glutamine. These results suggest p27 plays a novel role in regulating nutrient usage.

Societal Impact: P27 down regulation is strongly associated with high histopathologic tumor grade as well as an indicator of poor patient outcome in a majority of breast cancer studies. Our results have potential clinical significance because it might be possible to develop new cancer treatments using glutaminolysis inhibitors that specifically target tumors with the p27 gene disrupted.

This project was supported by the National Institute of General Medical Sciences of the National Institutes of Health through Grant Number 8P20GM103447.

Near-death experiences (NDEs) can be considered an altered state of consciousness and are becoming a more common occurrence in today's society. The increase can be attributed to the advances in emergency medicine and technology, which allows people to approach the threshold of death - be declared clinically dead and then resuscitated. What is intriguing is their recollection of seemingly mystical, transcendent phenomena. These reports typically involve auditory and visual experiences with cognitive, affective, paranormal, and transcendental features. Religion, psychology, and neurobiology perspectives all provide different theories for what causes NDEs and what the experience means. The religious perspective discusses how NDEs have influenced certain religions and how NDEs could be used as evidence to support certain religious tenants. The psychological view discusses how NDEs could be a response to stress and what psychological effects NDEs have on the experiencer. The neurobiological angle provides possible biological explanations elucidating brain function in response to near-death experiences. This study examines each perspective to arrive at a unified explanatory model. **METHODS? RESULTS? CONCLUSION? IMPACT?**



2019 Research Day at the Capitol

MONDAY, MARCH 25 * HYATT PLACE HOTEL, OKC

9:00 a.m – 4:00 p.m. Poster & Oral Presentation Judging

- Individual timeslots will be provided in advance
 - Arrive at designated time
 - Bring your poster with you, mounted on a firm board
 - Check in at the EPSCoR table
 - Poster number will be provided at check-in
 - Place the number in the top/right corner of your poster
Leave the number on your poster throughout the event/both days
 - Wait outside the judging room for your turn to present
 - Students will enter the judging room one-at-a-time
 - Take your poster in with you
(Demonstration materials are okay if they are small and safe)
- **IMPORTANT!!** An easel will be provided in the judging room. However, YOU must bring your own easel to the Capitol the following day.



2019 Research Day at the Capitol

POSTER AND ORAL PRESENTATION JUDGING

Oral Presentation: 3 minutes (timed)

- Walk in- SMILE, introduce yourself, be confident, and take them through what you have done - using your poster as a guide.

Poster Review + Q & A by Judges: 5 minutes maximum (timed)

- Anything on your poster is eligible for questioning so BE FAMILIAR with all components.
- Questions are usually to re-affirm or clarify something about your presentation.
- Kinds of questions - Procedural, social impacts, future aspirations.
- The entire 5 minutes may not necessarily be used.

-
- ❖ After Q&A: Exit the room with your poster (& demonstration materials if you brought them)
 - ❖ Leave the number on your poster for Tuesday
 - ❖ You are free for the rest of the day



2019 Research Day at the Capitol

PRESENTATION PREP & SUGGESTIONS

- The best way to improve your presentation skills is to present.
- Record yourself presenting and play back your recording to notice and fix your mistakes.
- Practice presenting to a friend who doesn't have a science background and listen to their feedback on your presentation.
- Practice presenting in an empty room using the volume you plan to speak at and hand gestures (pointing to figures/text on poster).
- Maintain natural eye contact with your audience in order to keep them engaged.
- Emphasize the importance of your societal impact. Explain why your scientific findings are important.
- What if you're asked a questions that you don't know the answer to? Do NOT make up an answer—it's better to say you don't know.



2019 Research Day at the Capitol

ADDITIONAL PRESENTATION SUGGESTIONS

Special suggestions from past participants:

- Don't underestimate the difficulty of synthesizing your project into a mini 3-minute presentation.
 - Time yourself over and over again to make sure you can present your material in the 3-minute timeframe.
- Also practice how you will talk to any Capitol guests who may not have experience in your area or who are non-scientists (this will be different than your more technical/judged presentation).
- Know ahead of time what you want to share with your Legislators.



2019 Research Day at the Capitol

SUGGESTIONS FROM THE JUDGES

- Review sample judging criteria sheet
- Review your abstract and make sure it's accurate; use the space that you have been allotted & revise if necessary
- Talk loud and project your voice
- Pay close attention to societal impact and research objective
- Answer, "What have you accomplished with your research?"
- Statistics are good—provide proof of outcomes



2019 Research Day at the Capitol

SUGGESTIONS FROM THE JUDGES

- Focus on what you contributed in regards to the research. Don't claim to have done it all if that's not the case. Toot your horn if it's applicable!
"With assistance I...."
"In collaboration with my faculty mentor I....."
"I explored _____ with the grad assistant on the project."
"I independently performed....."
- Avoid jargon in oral presentations; clarity for general audiences should be considered
- Societal impact statement should be included on the poster and also in the oral presentation



2019 Research Day at the Capitol

SOME COMMENTS DIRECTLY FROM THE JUDGES' BOOKS

- **Grand Prize: No weak spots**
Student showed great understanding of the chemistry; really fascinating and relevant research.
- **1st Place Winner: “6s” on Clarity for General Audiences**
Knew subject matter, but didn’t do a very good job of making it approachable to everyday language. Very polished presentation.
- **2nd Place Winner**
Used lots of jargon, but started strong with a clear statement. Clear on what work he did.
- **3rd Place Winner:**
Didn’t convey why we should care about this research (i.e. societal impact).
- **Non-Winning Student: Scored 8-9 on everything except Visual Appearance (2-4)**
The poster is all words.



2019 Research Day at the Capitol

HOTEL ACCOMMODATIONS (REQUEST DEADLINE FEB. 4)

EPSCoR will provide lodging on the evening of Monday, March 25 for student participants who live outside the OKC metro area and who have requested lodging prior to the February 4th deadline.

- Conference hotel: Hyatt Place Hotel
The EPSCoR office will book the room for you and pay the hotel directly for the room charge.
- Hotel will require a credit/debit card from students at check-in to cover any incurred incidental charges.
- A guest may stay in the room with you at no additional charge.
- Email me no later than Feb. 4 to secure a room; a signup sheet is available today (indicate one bed or two in the room).
- Confirmation numbers will be issued to you in February.
- If a room is booked on your behalf and is not used, you/your institution will be responsible for the charges.



2019 Research Day at the Capitol

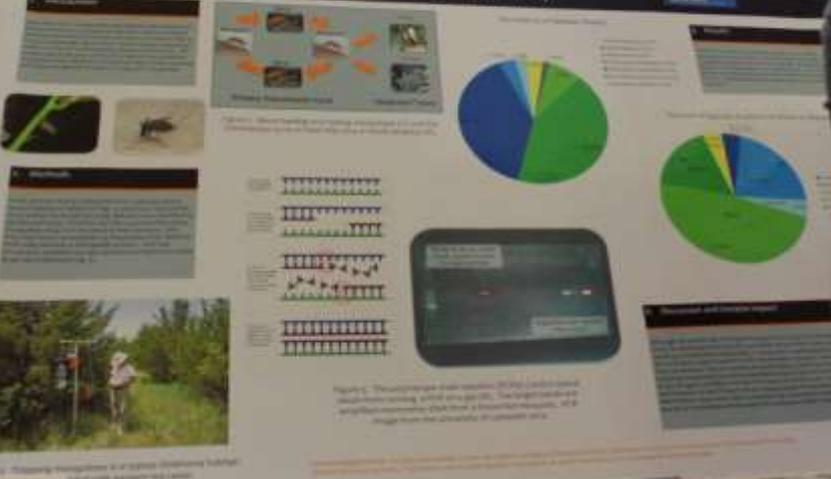
TUES., MARCH 26 CAPITOL POSTER SESSION



Bloodmeal Identification of Mosquitoes in Oklahoma

Megan Prouty, Valerie O'Brien, Tyler Ward, and Michael Reiskind
Department of Entomology and Plant Pathology, Oklahoma State University

21



21 Megan Prouty
Oklahoma State University



2019 Research Day at the Capitol





2019 Research Day at the Capitol

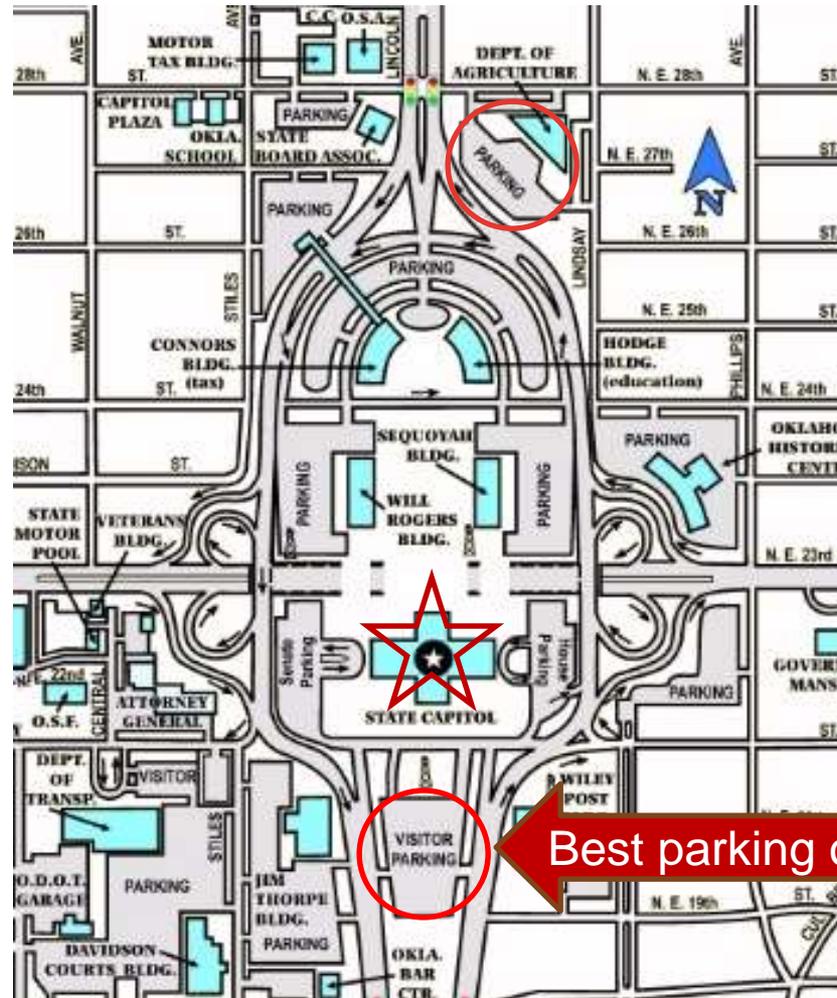
TUES., MARCH 26 TIMELINE: POSTER SESSION & AWARDS

- 6:30-7:00 a.m. Breakfast for students staying at the Hyatt
(Free for guests, Hotel lobby)
- 7:30-8:00 a.m. Students arrive at Capitol, 4th Floor Rotunda
(Setup posters)
- 8:25 a.m. All poster set up & ready to present
- 8:30-11:15 a.m. Posters on exhibit, 4th Floor Rotunda
(Students greet Legislators & Capitol guests)
- 11:30 a.m. Awards Ceremony, 2nd Floor Rotunda
- 12:30 p.m. Adjourn & take down posters



2019 Research Day at the Capitol

CAPITOL COMPLEX: IT'S A MAZE + LIMITED GOOD PARKING



Best parking option - arrive early!





2019 Research Day at the Capitol

TUES., MARCH 26 ARRIVAL INFORMATION

- Give yourself plenty of time—it's hard to navigate around the Capitol streets & parking lots in the dark
- Park in an approved visitor parking area
- Go through security (no knives, etc.) & take elevator to the 4th floor
- Your program book, survey form, and a contact sheet will be on your table.
- Wear your name badge from the previous day.
- **Everyone must be set up and ready to go by 8:30 a.m.**
- Survey forms/contact sheets should be turned in to the EPSCoR table prior to the awards ceremony (~11 am).



2019 Research Day at the Capitol

MARCH 26 CAPITOL POSTER SESSION

A six-foot table covered with a white, floor-length tablecloth will be provided for you.

You are required to bring:

- Photo ID
(May be requested by security at the Capitol entrance)
- Your poster
- Firm board backing for your poster
- Easel
- Tacks, Velcro or other attachment materials
- Your poster number that was provided the previous day

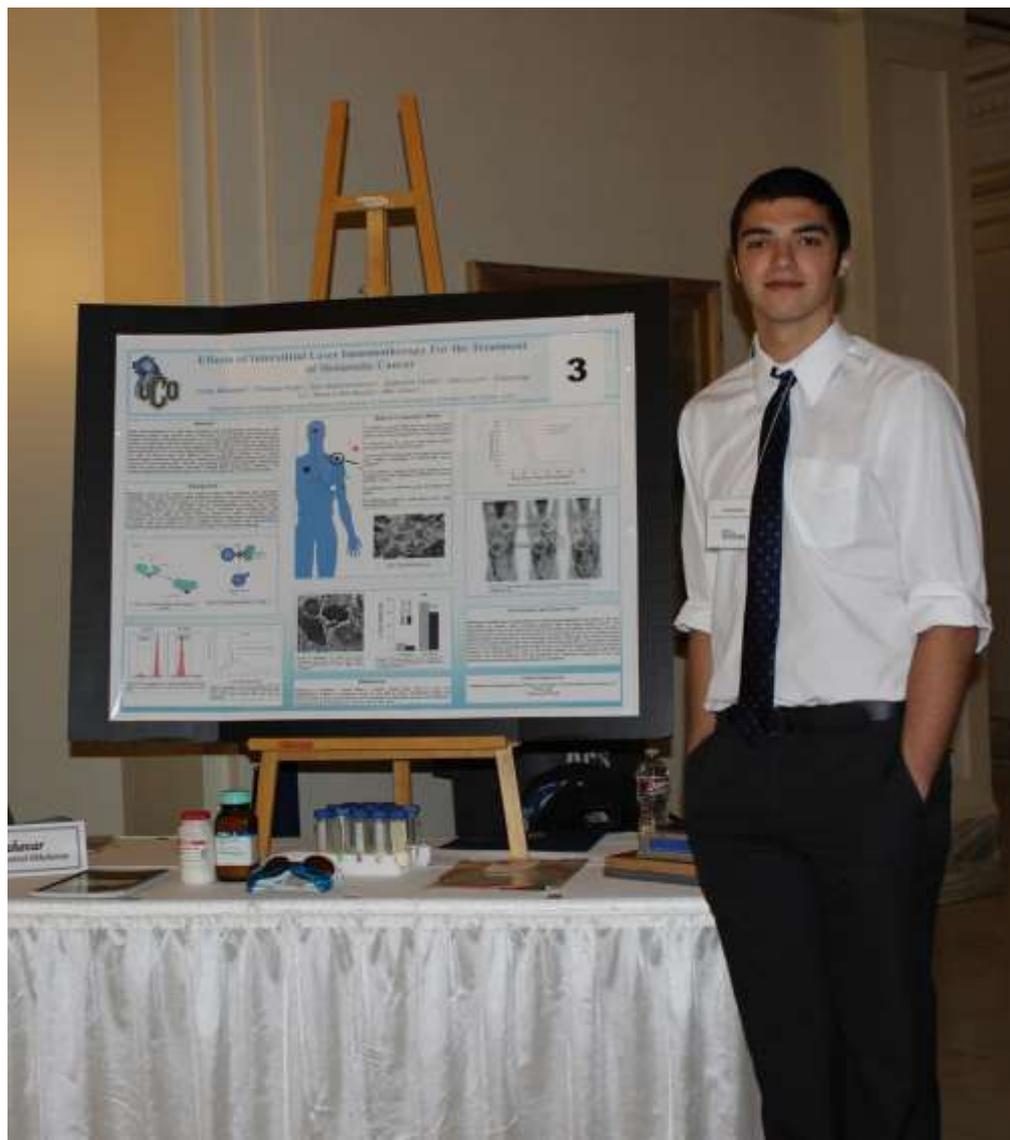
You are highly encouraged to:

- Bring hands-on demonstration materials



March 26 * At the Capitol

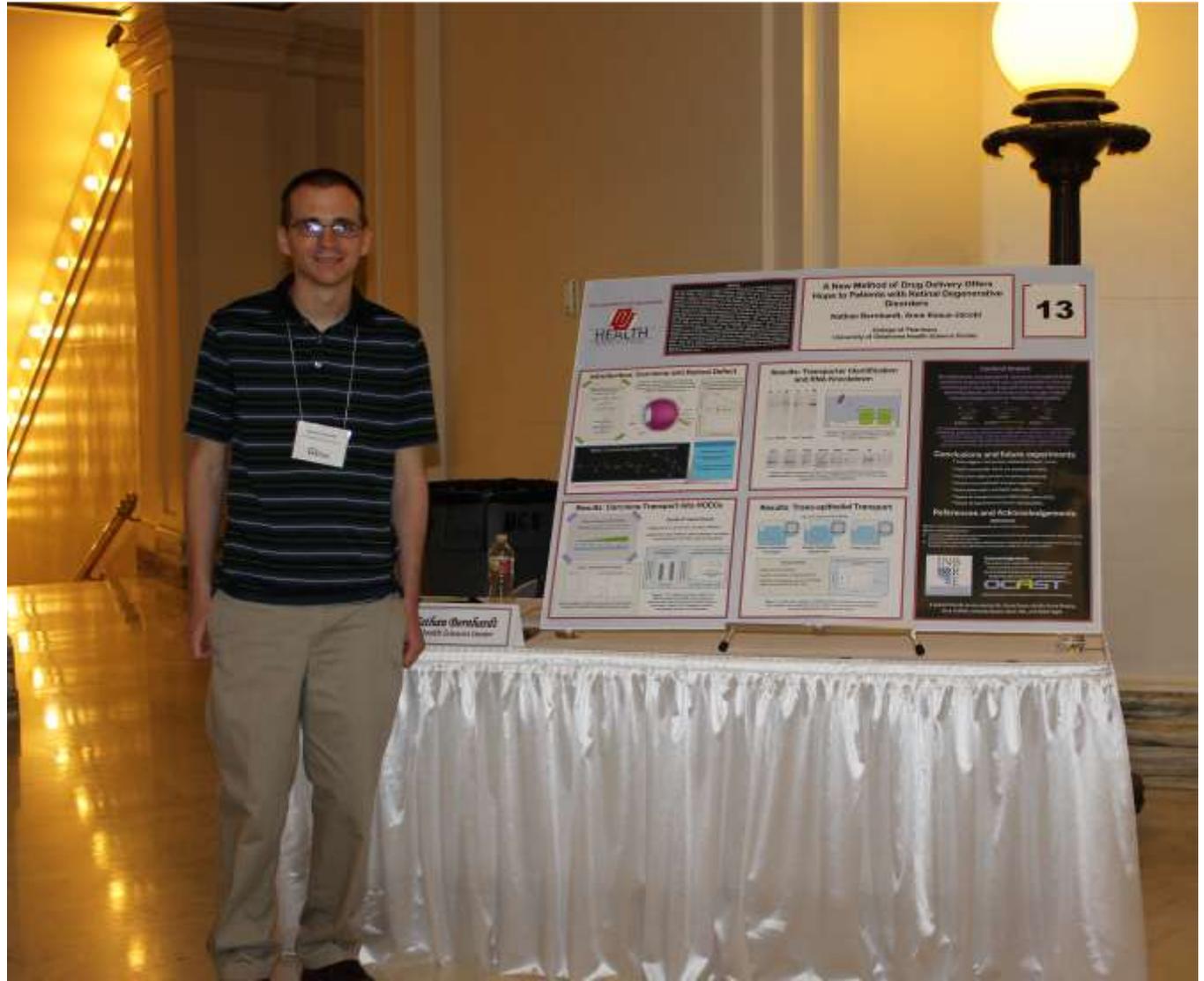
BRING HANDS-ON DEMONSTRATION MATERIALS





March 26 * At the Capitol

DRESS PROFESSIONALLY; A NEW SUIT ISN'T NECESSARY





2019 Research Day at the Capitol

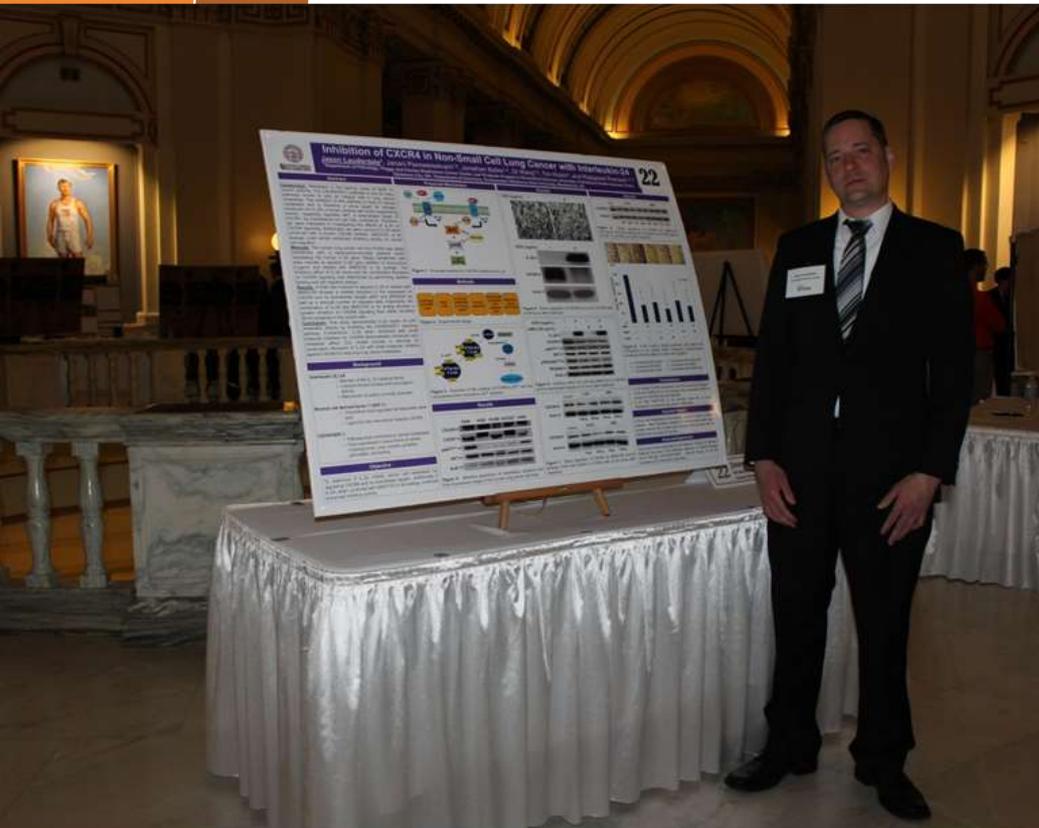
APPROPRIATE DRESS – BOTH DAYS





March 26 * At the Capitol

DO NOT FORGET YOUR EASEL AND FIRM BOARD!





March 26 * Day of the Event

SHARE YOUR WORK: RDC JUDGES, LEGISLATORS & CAPITOL VISITORS





March 26 * At the Capitol

THE LEGISLATORS

- Identify your home and school Representatives and Senators (may be different)
www.oklegislature.gov
- Remember: Use layman's terms & outline how your research affects and/or benefits his/her constituents!
- Not everyone will receive a citation, but we make a recommendation and provide details to encourage it.
- Grab a photographer.





2019 Research Day at the Capitol

TIMELINE OF IMPORTANT DATES

Dec. 2, 2018 – March 24, 2019

Students prepare scientific posters & oral presentations

Monday, February 4, 2019

Students' revised abstracts and lodging requests are due

Monday, March 11, 2019

Online registration closes

Monday, March 25, 2019

Poster/oral presentation judging
9 a.m. – 4 p.m.
Hyatt Place Hotel, Oklahoma City
Student Participation Mandatory

Tuesday, March 26, 2019

Posters on Exhibit
8:30 – 11:15 a.m.
4th Floor Rotunda, State Capitol, OKC
Student Participation Mandatory

Tuesday, March 26, 2019

Awards Ceremony
11:30 a.m. – noon
2nd Floor Rotunda, State Capitol, OKC
Student Participation Mandatory

All March 25-26 activities are mandatory for student researchers; registered guests are invited to participate in all Capitol activities on the 26th.



2019 Research Day at the Capitol

POSTER PREP: A QUICK REFERENCE GUIDE

Purpose of your Research Poster: Disseminate research findings and progress to Legislators, the public, and your peers

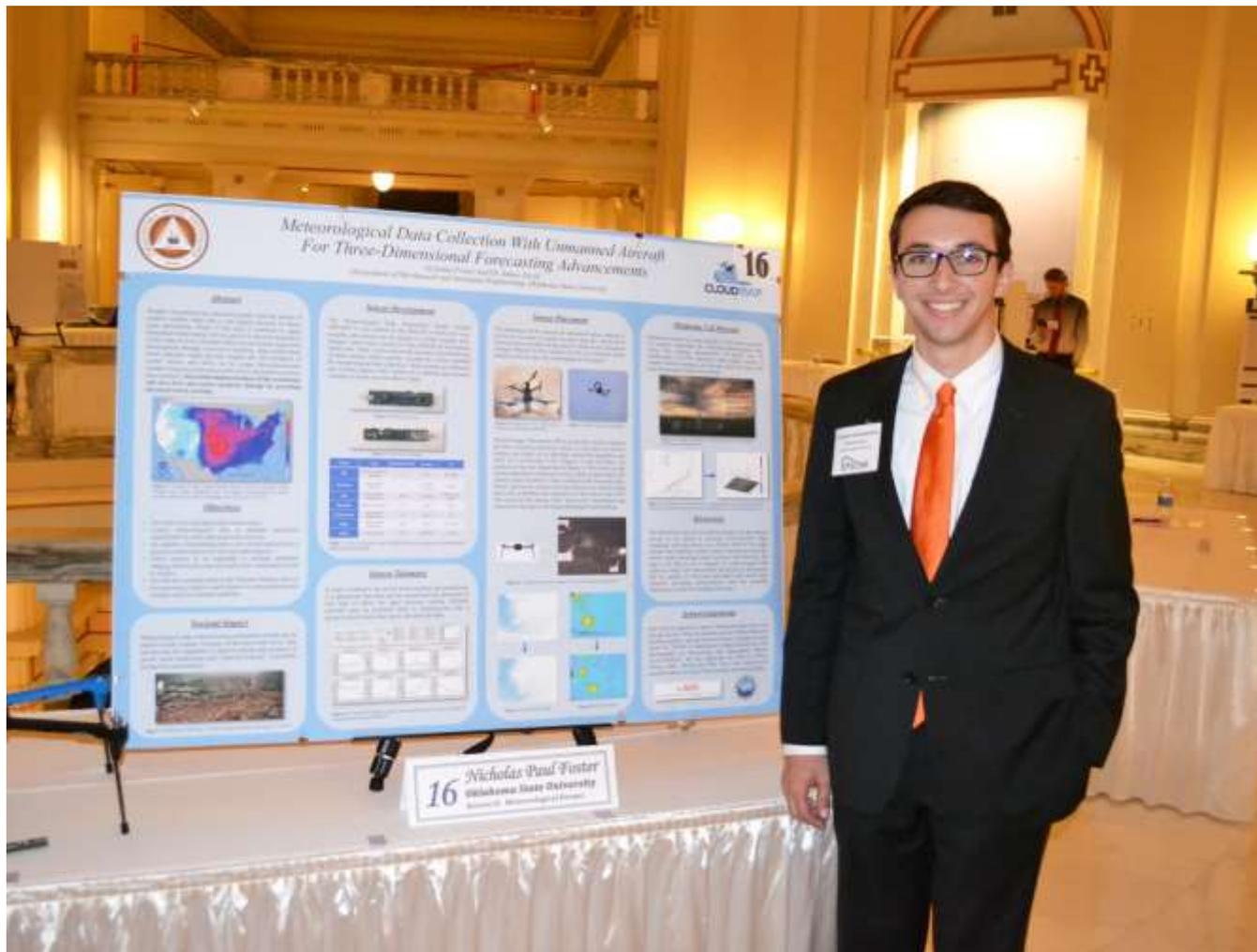
- Will not be a cut-and-paste version of your abstract
- Visually communicates a “take-away message”
- Spotlights your most important ideas, points, findings
- Serves as an interface between your research results and your oral presentation

❖ *Posters must be the work of the student researcher.*



2019 RESEARCH DAY AT THE CAPITOL

Poster Prep: A Quick Reference Guide





2019 Research Day at the Capitol

POSTER PREP: A QUICK REFERENCE GUIDE

❖ PowerPoint is recommended for your poster design

❖ Before you start

- Check with your print shop regarding size and color constraints that may apply
- Average size 48"x36"
- Set the page size (in your program) to match the final print size
- Ask your mentor for advice regarding where to print your poster



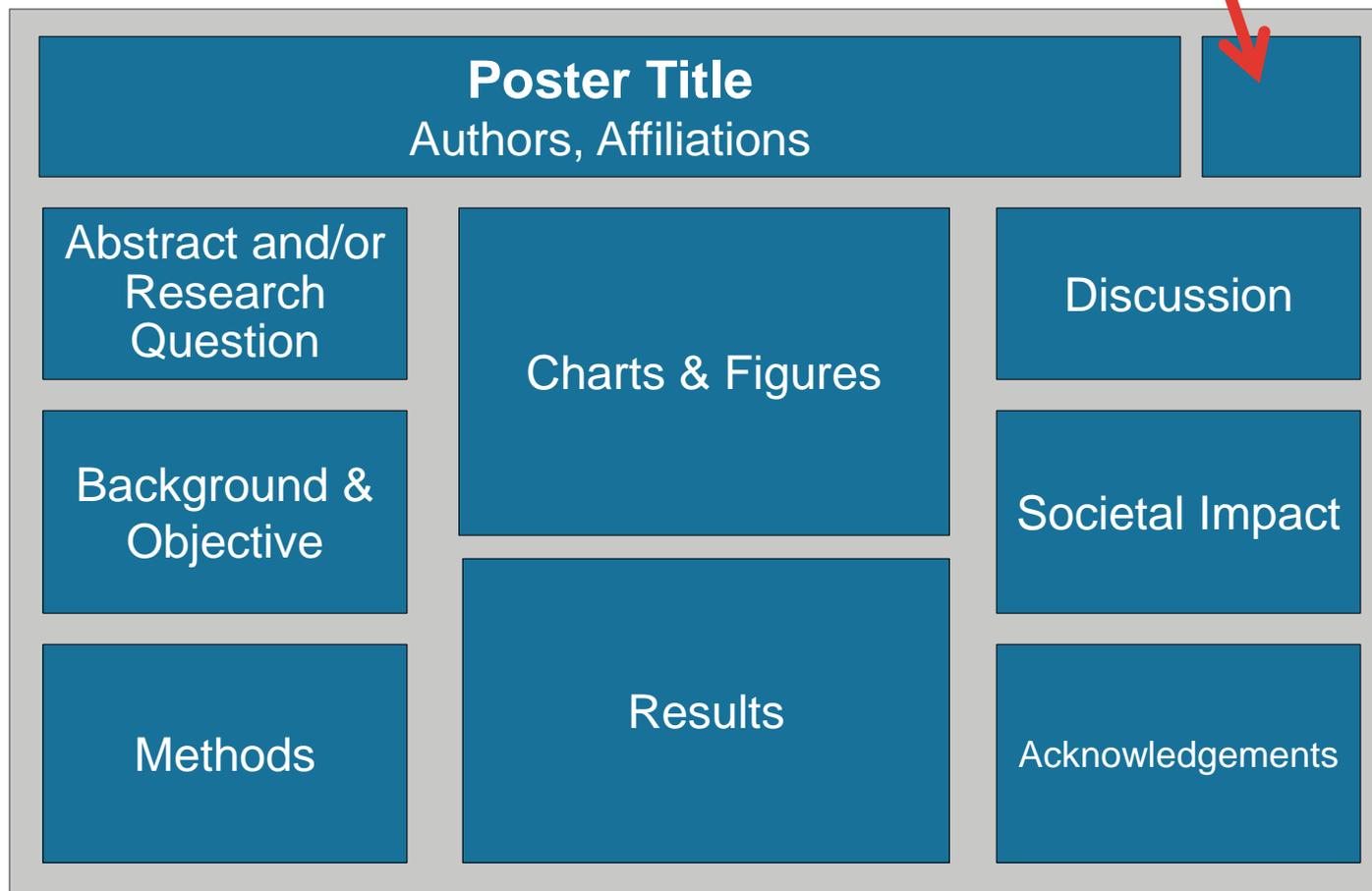
➤ Your poster for this event may deviate slightly from the standard/traditional 48"x36" poster dimensions



2019 Research Day at the Capitol

POSTER PREP: GENERAL POSTER FORMAT

Leave 4.5x5.5" blank space here for poster number that will be provided to you





2019 Research Day at the Capitol

POSTER PREP: A QUICK REFERENCE GUIDE

- Every poster should be custom made/tailored to the event you are preparing it for
- Your Research Day at the Capitol poster is being judged by highly educated individuals from various STEM fields but it IS ALSO being viewed by important Legislators who may not have a science background.

SO:

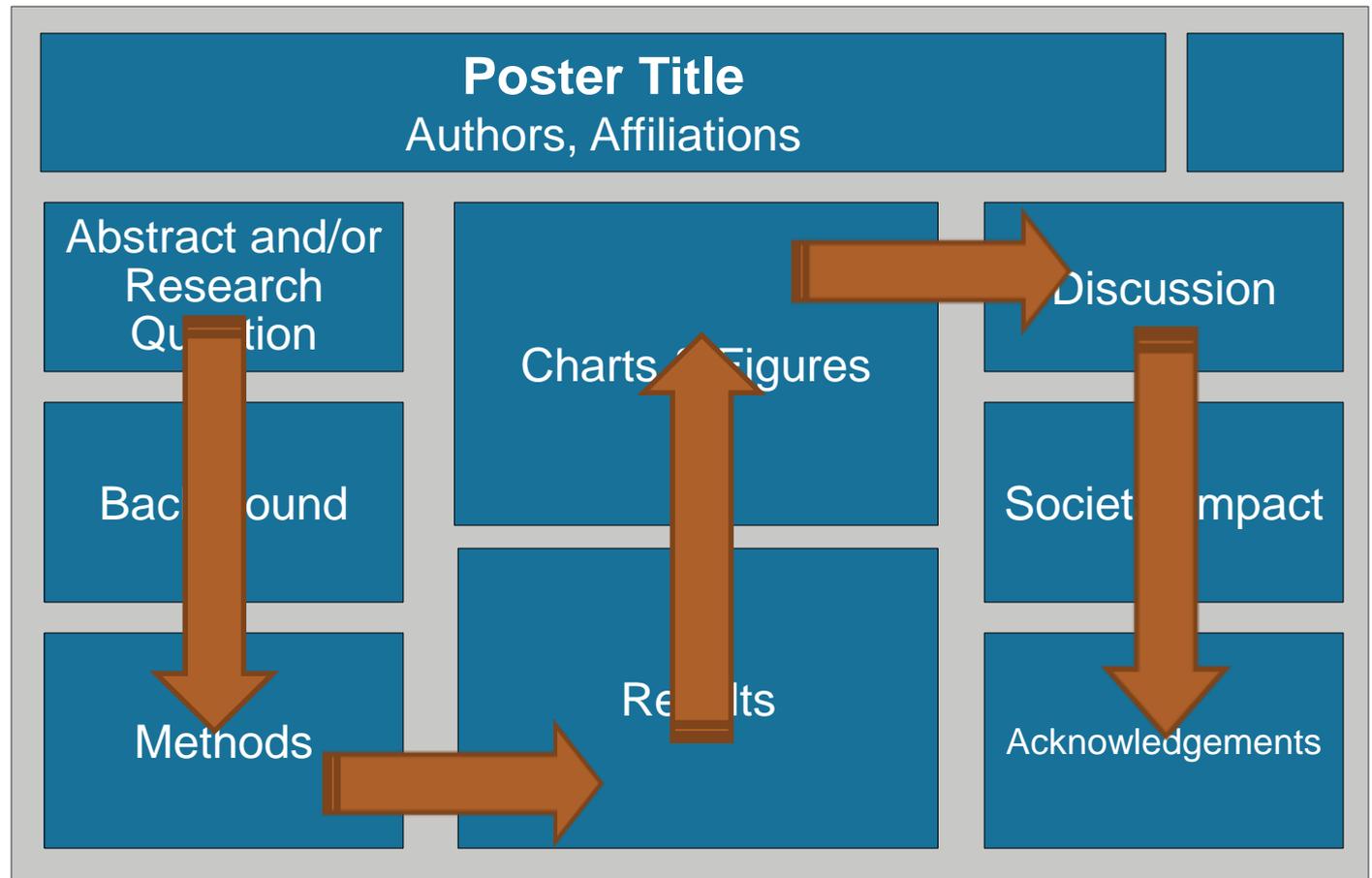
- Succinctly and clearly express your scientific research and findings
- Include all essential information; keep writing concise
- Avoid field-specific jargon
- DON'T dumb-down your research!
- Use your Societal Impact Statement to reach the audience members who may not understand the deeper science.



2019 Research Day at the Capitol

POSTER PREP: GENERAL POSTER FORMAT

Use logical flow between sections: top to bottom and left to right

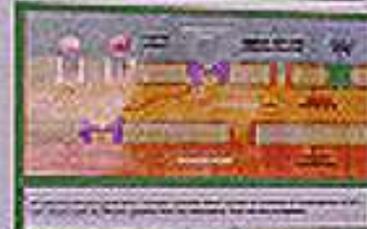


Ca²⁺-Dependent Cl⁻ Action Potentials in Chick Different Points of View

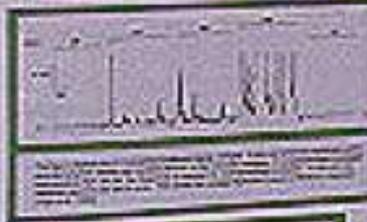
Roberta Elisei, Daniela Anarado, Sarah Orla, Kyong Kim

The chick retina is a well-studied model system for the study of the development of the visual system. The chick retina is a well-studied model system for the study of the development of the visual system. The chick retina is a well-studied model system for the study of the development of the visual system.

All external stimuli cause a depolarization of the membrane. Subsequently, Cl⁻ channels open causing hyperpolarization.

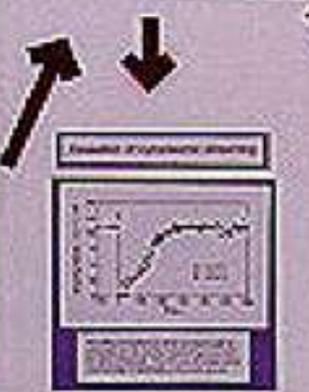


Cl⁻-dependent Cl⁻ channels are involved in the generation of action potentials.



Cl⁻ channels are involved in the generation of action potentials. The presence of Cl⁻ channels is essential for the generation of action potentials in the chick retina.

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Cl⁻ channels are involved in the generation of action potentials. The presence of Cl⁻ channels is essential for the generation of action potentials in the chick retina.

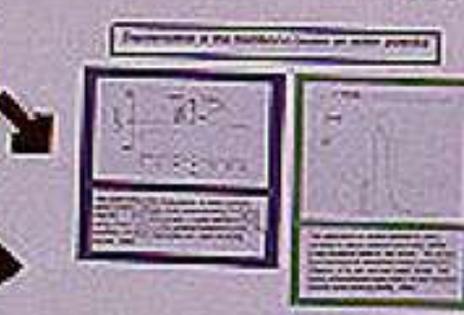
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Cl⁻ channels are involved in the generation of action potentials. The presence of Cl⁻ channels is essential for the generation of action potentials in the chick retina.

Abstract

Introduction: Ras oncogene activations are present in approximately 30% of human malignancies including colon, pancreas, thyroid and hematopoietic cancers. Our earlier studies reveal that oncogenic K-Ras-transformed cells are highly sensitive to inhibition by phorbol 12-myristate 13-acetate (PMA). In this study, we utilized a human fibrosarcoma cell line (HT-1080) with a mutated N-Ras allele to investigate further the effects of PMA on Ras-transformed cells.

Methods: The entire coding region of N-Ras was amplified from HT-1080 cell cDNA by PCR and sequenced. HT-1080 cells were cultured in Dulbecco's Modified Eagle Medium (DMEM) supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO₂, and humidified conditions in the presence or absence of PMA. Cell counts were obtained on a 2/10 mm hemocytometer and phase contrast microscope. Cell density and morphology were observed with Wright-Giemsa and immunofluorescence staining. Activation of Erk1/2 was assessed using Western blot analysis and immunofluorescence staining.

Results: Heterozygous N-RasQ61K mutation was found in HT-1080 cells. Cultures treated with a high dose of PMA (10uM) consistently showed a significant (p<0.05) decrease in cell number compared to the respective control culture. Results for HT-1080 cell cultures treated with a low dose of PMA (0.02uM) were less consistent and the decrease was not always significant (p>0.05). PMA-treated cells have a stretched appearance with prominent actin reorganization and appear differentiated.

Conclusions: PMA induces extensive cell growth inhibition and morphology changes in HT-1080 fibrosarcoma cells.

Introduction

The Ras proto-oncogenes encode small GTPases (N-Ras, H-Ras, and K-Ras) which act as molecular switches in regulating cellular proliferation, differentiation, and survival.¹ Non-transformed Ras proteins are only transiently active while oncogenic mutations create constitutively active Ras proteins.² This state results in constitutive activation of downstream effectors, including the Ras-Raf-Mek-Erk(p42/p44 MAPK) pathway, which is involved in cellular proliferation.³ Oncogenic Ras mutations occur with a 30% frequency in cancers of the highest mortality.¹ We investigated the effect of PMA on the HT-1080 human fibrosarcoma cell line containing an endogenous mutated N-Ras allele. PMA mimics the endogenous activator diacylglycerol (DAG) to activate proteins across many different classes including novel and classical protein kinase C isozymes, protein kinase D isozymes, and Ras guanine nucleotide exchange factors which activate Ras proteins.² PMA is perhaps best known for its tumor promoting properties in the mouse skin carcinogenesis model. Prolonged topical application of PMA promotes skin tumors on mice previously exposed to a mutagenic carcinogen.² Our earlier studies, however, revealed that oncogenic K-Ras-transformed cells are highly sensitive to inhibition by PMA. Depending on the cell type, PMA is capable of promoting mitogenic responses or initiating growth arrest. Our goal was to investigate further the effects of PMA on N-Ras-transformed cells.

Acknowledgments

This project was supported by the National Institute of General Medical Sciences of the National Institutes of Health through Grant Number 8P20GM103447 and by Oklahoma EPSCoR.

Results

Heterozygous N-RasQ61K mutation in HT-1080

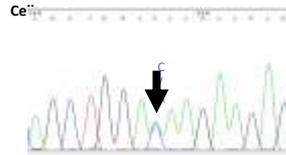


Figure 1. The entire coding region of the N-Ras gene was amplified from HT-1080 cell cDNA by PCR and sequenced. HT-1080 cells show heterozygosity for the N-Ras mutation with a single amino acid substitution at position 61, from a glutamine (Q) to a lysine (K).

Comparative Morphology

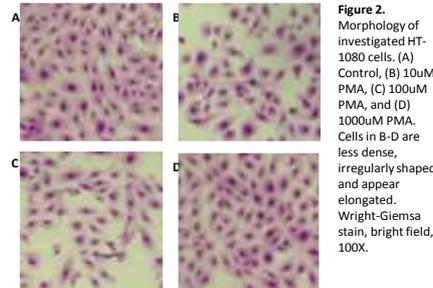


Figure 2. Morphology of investigated HT-1080 cells. (A) Control, (B) 10uM PMA, (C) 100uM PMA, and (D) 1000uM PMA. Cells in B-D are less dense, irregularly shaped and appear elongated. Wright-Giemsa stain, bright field, 100X.

Viable cell count

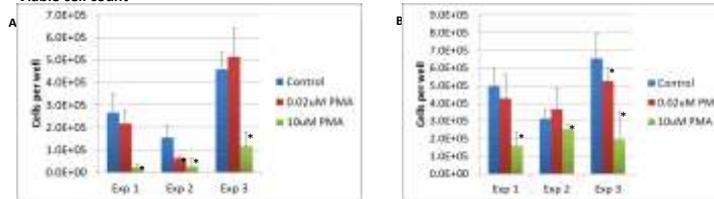


Figure 3. Experiments in set (A) were seeded with half the uL HT-1080 cells seeded in set (B). Four viable cell counts were taken per slide, error bars indicate standard deviation. *P<0.05 indicates PMA-treated cells are significantly different from the corresponding control cells.

Morphological changes, actin reorganization and Erk 1/2 activation by PMA in HT-1080 cells

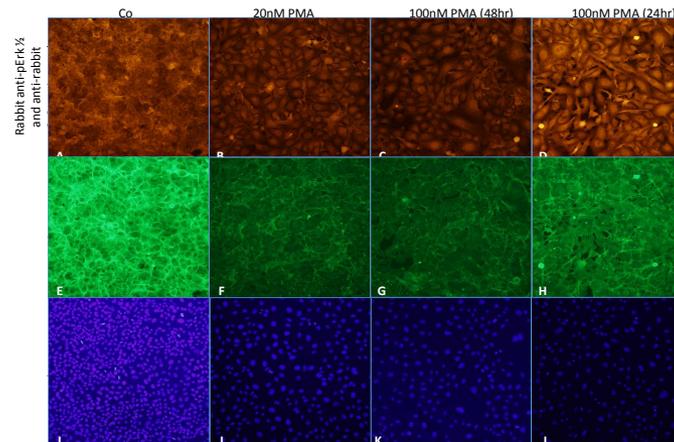


Figure 4. With the addition of PMA, A-D highlight increasing Erk 1/2 activation, E-H actin reorganization, and I-L increasing nucleus size and decreasing cell numbers. Brightly stained mitotic cells are visible in image I which are lacking in J-L after PMA treatment. Immunofluorescence microscopy, 200X.

Materials & Methods

Cell culture. Stock cultures of HT-1080 cells were maintained in DMEM supplemented with 10% FBS at 37°C, 5% CO₂, and humidified conditions. PMA dissolved in DMSO was added to cultures at a concentration of 0uM, 0.02uM, or 10uM. For each experiment to determine cell count, a high and low volume of HT-1080 cells were seeded from the stock culture; the low volume was always equivalent to half of the high volume. DMSO alone at the final concentration used in our experiments (<1%) is assumed to have no effect on cell growth. Cell numbers were determined on a 2/10 mm hemocytometer and phase contrast microscope (1X) after trypsinization in the presence of 0.05% EDTA.

Cell staining. HT-1080 cells were treated with 10uM, 100uM, 1000uM PMA or DMEM alone (control) for three days, fixed with methanol and stained with a Wright-Giemsa stain. For immunofluorescence microscopy, adherent HT-1080 cells were grown on glass coverslips. Cultures were treated with 20nM PMA for 48 hours, 100nM PMA for 48 hours, 100nM PMA for 24 hours or DMEM alone (control). Cells were fixed with 4% formaldehyde, permeabilized with 0.2% Triton X-100, and blocked with 50mM Tris-HCl to minimize nonspecific binding. Samples were then labeled with rabbit monoclonal antibody recognizing pERK 1/2 followed by goat anti-rabbit polyclonal secondary antibody Cy3-conjugated. Actin were stained with FITC-phalloidin dye and nuclei were stained with Hoechst 33258 dye.

Western blotting. Proteins were separated by SDS-PAGE, transferred to a PVDF membrane and blocked with 1% BSA. The membrane was probed with a rabbit monoclonal antibody recognizing pERK 1/2, washed and then probed with a goat anti-rabbit-HRP conjugated polyclonal secondary antibody. Chemiluminescent detection was performed and images were

Conclusions

- HT-1080 cells treated with PMA exhibited extensive growth inhibition as determined by cell counts three days after treatment. Growth inhibition was most effective at higher doses of PMA (10uM) with fewer HT-1080 cells seeded from the stock culture. The high dose of PMA (10uM) showed a significant (p<0.05) decrease in cell number in six out of six experiments, regardless of the initial concentration of HT-1080 cells seeded.
- In the presence of PMA, HT-1080 cells tend to be less dense in patches across the culture whereas the density is more uniform throughout control cultures.
- After PMA treatment, HT-1080 cells become stretched in appearance with prominent actin reorganization, more stress fibers are visible and the cells and nucleus appear larger. Single giant cells are also visible.
- Based on nuclear staining, the number of mitotic cells appear to decrease with the addition of PMA.
- Erk 1/2 becomes more active in the nucleus of PMA-treated cells.

Societal Impact

Both K-Ras and N-Ras transformed cells are sensitive to PMA treatment which may have implications for development of anti-cancer drugs targeting oncogenic RAS or its downstream effectors.

References

- Takashima A, Faller D. Targeting the RAS oncogene. *Expert opinion on therapeutic targets*. 2013;17:507-531.
- Griner E, Kazanietz M. Protein kinase C and other diacylglycerol effectors in cancer. *Nature*. 2007;7(4):281-294.



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POSTER PREP: A QUICK REFERENCE GUIDE

Font Suggestions

- Use clear, simple fonts
e.g. Times New Roman, Garamond, Arial, Century Gothic
- Title, 60-72 pt
- Authors & Institution, 38 pt
- Headings of boxes/sections , 42 pt
- Text of boxes/section, 26-32 pt
(each column of text should have 11-12 words per line)
- Figure legends, 32 pt
- Acknowledgements, 26-32 pt
- Adjust font size as needed to fill your poster, but be consistent throughout the poster



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POSTER PREP: A QUICK REFERENCE GUIDE

Graphics & Photos

- Use visual aids to tell your story (images, charts, diagrams, timelines)
- Minimal text to supplement the graphics
- Use titles, legends, consistent color (X and Y-axes should be labeled!)
- Be concise in your wording
- Text and graphics should be legible from three feet away
- Careful use of color (2-3 colors maximum)
- Photos must be min. 300 ppi
- Credit photos when appropriate

A Novel Polysulfide Synthesized Entirely From Waste and Its Use In Water Remediation

Austin M. Evans, Michael P. Crockett, Prof. Justin M. Chalker
The University of Tulsa Department of Chemistry and Biochemistry
Tulsa, Oklahoma, USA 74104

Abstract

Many functional materials today are prepared from non-renewable feedstocks. Addressing this issue, our research team has developed a novel polysulfide material synthesized entirely from the industrial waste products sulfur and limonene. This material is easy to synthesize on a large scale and is effective in removing toxic metals from water.

Background

Many chemical products are synthesized from non-renewable petroleum sources. Addressing this issue, our goal was to use abundant and renewable compounds as starting materials. Specifically, we reacted limonene and sulfur directly to form a polysulfide. 70,000 tons of limonene are produced as waste each year by the citrus industry. Sulfur is produced in the excess of 70,000,000 tons per year by the petroleum industry. Their wide availability has prompted exploration of these materials as chemical feedstocks.



Figure 1. Production of sulfur and Limonene

Because of the high sulfur content of our limonene-sulfur polysulfide, we hypothesized that it would bind to toxic metals and therefore be useful in removing toxic metals from water. This is particularly pertinent to Oklahoma because many of our waterways exhibit some form of toxic metal pollution.

Materials and Synthesis

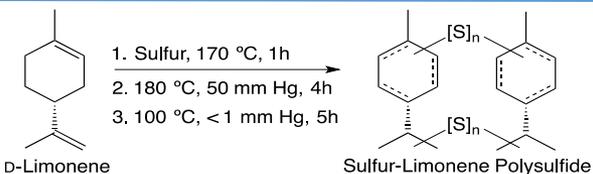


Figure 2. Reaction Scheme with proposed structure

Reaction Outline

- 1) Melt Sulfur (124 °C)
- 2) Heat to 170 °C (Radical Formation)
- 3) Add equal mass of limonene (b.p. = 176 °C)
- 4) Heat 1-5 hours at 170 °C
- 5) Process directly (mold, coat, etc)



Figure 3. Products of reaction

Reaction Features

- 1) No exogenous solvents or reagents
- 2) Completely atom economical
- 3) Operationally simple
- 4) Easily Scalable, 100 gram syntheses are routine

Water Remediation

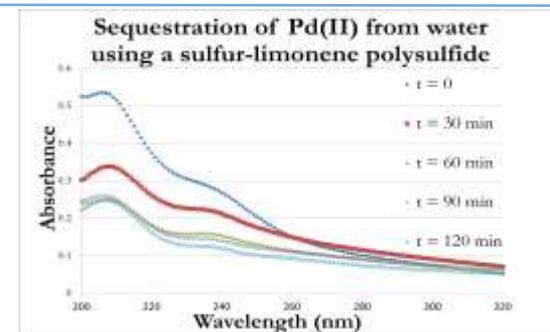


Figure 4. Palladium Catalyst Sequestration from Water Using UV-Vis Spectroscopy, we monitored a time course of the sequestration of a valuable palladium metal catalyst.

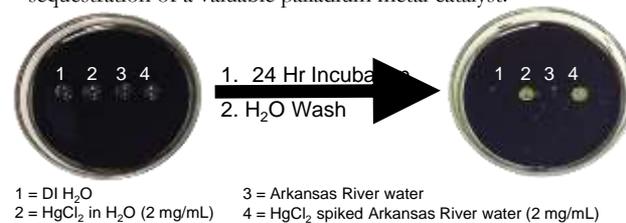


Figure 5. Mercury sensing by a chromogenic response

Societal Impact

We have synthesized a novel polysulfide material entirely from industrial waste. The limonene-sulfur polysulfide is useful in removing metals from water, including mercury salts. We are currently investigating commercialization of this technology for on-site purification of natural waterways.

References

1. Chung, et al. *Nature Chemistry* **2013**, 5, 518-524.
2. Polymers from Renewable Resources Gandini, A. *Macromolecules* **2008**
3. Crockett, M. P.; Evans, A. M.; Chalker, J. M. Unpublished
4. Sulfur-Limonene Polysulfide. Crockett, M. P.; Evans, A. M.; Chalker, J. M. Provisional patent filed Oct 24, 2014. No. 62068074.

REAL WORLD SYNTHETIC BIOLOGY:

Production of Arsenic Biosensors and Aspirin Producing Bacteria

Authors: Madison J. Duckwall and Lori Gwyn Southwestern Oklahoma State University

Abstract: Synthetic biology is an emerging field that applies engineering principles to biological systems to solve problems. BioBricks, a molecular toolbox of genes with varied functions, have compatible enzyme sites that function as cut and paste locations so that one can "build" bacteria to accomplish a specified purpose. Arsenic is toxic when present in high quantities (>10 ppb) in water. Medications are complicated and occasionally dangerous to synthesize. Utilizing synthetic biology to modify bacteria to become sensors and synthesizers will address these problems. Arsenic biosensors capable of detecting micro quantities of arsenic in drinking water was goal one. Three parts were used to build an arsenic sensor: an arsenic promoter, a red reporter, and destination. The promoter is the on switch activated in the presence of micro to nano arsenic concentrations, which then signals the reporter to make the bacteria grow red/pink. The destination is a place to seal the promoter and reporter together in a predicted manner as well as provide antibiotic resistance to further select only desired bacteria. BioBricks were also used to develop aspirin synthesizing bacteria. Proposed parts for this purpose include an isochromatase synthase and acetyltransferase. Experiments are underway to test the activity of proteins produced by these parts. It is expected that the bacteria will utilize their own metabolites (salicylic acid) to make aspirin to be secreted into its surroundings. This project is relevant as a proof of concept that drugs can be synthesized easily with bacteria. We have successfully made an arsenic biosensor and plan to make it more sensitive and selective. Preliminary experiments of the aspirin project show that the E. coli can grow in $\leq 0.1\%$ salicylic acid and up to 0.1% acetic acid. This indicates that the completed aspirin synthesizer should not be inhibited by the acidic environment it will create.

Arsenic Biosensor

Intro:

The EPA accepted level of arsenic in drinking water is 10 parts per billion (ppb). This is a worldwide problem as well as local.

EPA



In Weatherford, OK approximately \$750,000 were spent as one method of decreasing arsenic concentration in the drinking water by 1ppb.



In Texas, Nepal 25% of the wells show arsenic concentrations up to 50 ppb.

EPA

Arsenic biosensors can be useful in determining if there are dangerous levels of arsenic present in the water for consumption.



Methods:

Refer to figures 2 and 3 for detailed methods. The genes used for the arsenic biosensors are promoter J23201 to bind the arsenic and "turn on" the reporter E1059 which causes the red/pink color of the bacteria. Both genes are enclosed in the destination pSB1A2 which accounts for the Ampicillin resistance allowing for confirmation of the desired bacteria.

Results: We successfully constructed the arsenic biosensor that detects arsenate (AsO_4^-) in the media. See figure 1 for the difference in growth between the control exposed to arsenic and the one not exposed. The arsenic is currently very sensitive and can detect down to 1ppb concentrations of arsenic in the media. The hope for future directions is to increase specificity of the promoter leading to the arsenic so that we can get a better picture of whether or not the water is safe for drinking.

Figure 1. The arsenic biosensor exposed to arsenic grew pink while the arsenic biosensor not exposed to arsenic grew the normal white/yellow of E.coli. The clocks signify the rapid multiplication rate of bacteria.

Reaction Scheme

Figure 2. The genes used for the arsenic biosensors are promoter J23201 to bind the arsenic and "turn on" the reporter E1059 which causes the red/pink color of the bacteria.

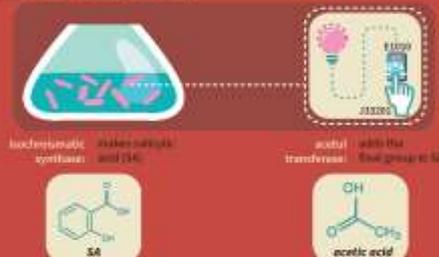
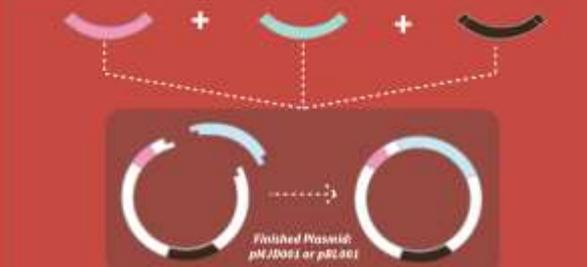


Figure 3.

Step 1: Enzym Digest



Step 2: Ligation



SOCIETAL IMPACT STATEMENT:

BioBricks can be utilized to build an infinite number of biological tools. Production of arsenic biosensors for identifying arsenic contamination will determine drinking water safety. Construction of aspirin producing bacteria is a potential alternative method to costly manufacturing procedures.

Aspirin Synthesizer

Intro:

Many medications are difficult and expensive to produce. The BioBricks Aspirin Synthesizer will use salicylic acid (SA), a normal bacterial waste product, and turn it into aspirin as the final waste product accepted by the E. coli. The idea behind the aspirin synthesizer is that if a simple small molecule drug can be made via E. coli then it is proof of concept of a possible route of drug manufacturing. Using biological systems could cut down on the complexity and cost of synthesizing a wide variety of medications. At this point the plan is to expand on that of the aspirin molecule to other medications that derive their structure from SA.



Aspirin is mostly made of fresh and then synthesized.



Methods:

Figure 3 and 4 show the detailed method of making the plasmid. The production of the plasmid is based off of the same protocol as the arsenic biosensor with the only difference being the plasmid gene parts used. Here an isochromatase synthase is used to make the starting material SA and an acetyltransferase will be used to put the finishing touch of an acetyl group on SA to make and secrete the aspirin.

Table 1. E. coli Growth under various Acidity Conditions

Conditions	No SA/ No AA	SA/ No AA	No SA/ AA	SA/ AA	Control SA
Growth (OD)	Yes	Yes	Yes	Yes	No

Figure 4. An isochromatase synthase is used to make the starting material SA and an acetyltransferase to put the finishing touch of an acetyl group to the SA to make and secrete the aspirin.

Results: A system of producing aspirin in bacteria is making the environment less acidic for the E. coli to live in. To address this problem, we the lowest controls of the E. coli in acidic conditions from salicylic acid and acetic acid. As shown in Table 1, the bacteria grew normally in these environments confirming that we can proceed with further experiments to engineer the plasmid that will cause the E. coli to secrete aspirin. The goal of producing the Aspirin Synthesizer (pN1001) is a first proof of concept that we can manufacture drugs via bacteria. We are planning to take this technology and add more genes to add different molecular groups to make different drugs. Initially lower synthesis (100) drugs are the best type of drugs to pursue because these drugs are direct salicylate derivatives.

References:

- Roman Shrestha, Arsenic Contamination of Groundwater in Nepal: Good Public Health Intention Gene Bad. Inquiries Journal.
- Hareesh Arora, Sakib Pathan, and Saqib Kasori, Arsenic Pilot Plant Operation and Results-Weatherford, Oklahoma. Sandia Report. Printed May 2007.

Acknowledgements:

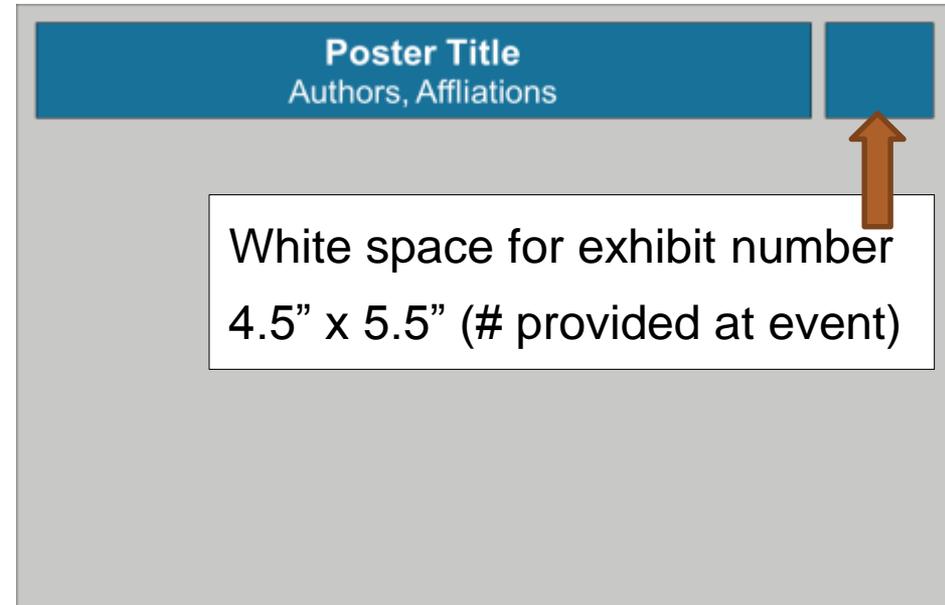
Thank you to the SWOSU Department of Chemistry and Physics, SWOSU College of Arts and Sciences, SWOSU Business Enterprise Center, and the US Department of Labor for providing funding and support. Thank you to Nicha Monahan for assistance with experiments.



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POSTER PREP: A QUICK REFERENCE GUIDE

- ❖ **Title** - Keep it simple & concise
- ❖ **Authors** – List all who were involved
- ❖ **Institution** –
Campus
you are
representing



Background

- Since the rise of processed food and readily available, high-fat commodities, obesity has drastically increased. Despite the correlation between poor diet and exercise, researchers have identified an innate biological element to obesity. This is a complex genetic disease not entirely understood, outside of the few genetic associations made thus far.

Obesity Rates for Adult Women (Ages 18-64 years) BYRS 2012



Figure 1. Obesity rates by state (Digital image: The State of Obesity, 2012 Web, 18 March 2017)

- MicroRNAs (miRNAs) are small, noncoding, RNAs 22 nucleotides in length. They bind to the 3' UTR untranslated region of their target mRNAs and repress protein production by destabilizing the mRNA, causing translational silencing. miRNAs play a role in various cellular processes such as development, differentiation, and growth. This study explores the roles of miRNA in adiposity by observing effects of miRNA in triglyceride concentration in the *Drosophila melanogaster* (fruit fly), in hopes of identifying a connection between miRNA regulation and obesity.

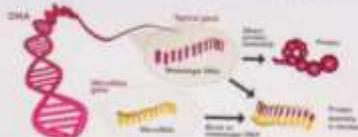


Figure 2. miRNAs, acting via miRNAs. (Digital image: Compendex one, Biochemical Reviews, 117, 2-4, Web, 6 March 2017)

Objectives of Study

- Develop a better physiological understanding of obesity by identifying miRNAs that affect fat.
- Discover which miRNAs significantly positively or negatively influence triglyceride content.

Societal Impact

- Obesity can generate countless long-term health implications including heart disease, diabetes, and stroke.
- It affects society as a whole; not only those who require treatment, but taxpayers who subsidize healthcare for the obese.
- Oklahoma in particular has one of the highest obesity rates in the nation.
- This disease has become an economic and health crisis.

Methods

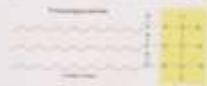
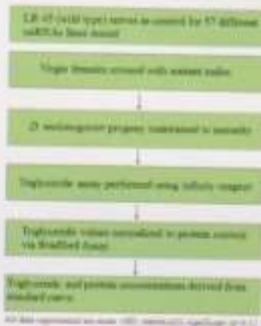


Figure 3. Triglyceride ("fat") molecule. Adding different miRNAs will determine what the various miRNAs control with proteins (Bradford of triglyceride). Concentration of protein fat is relative measured using spectrophotometry.

Mutant Fruit Flies

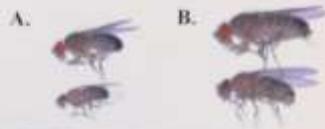


Figure 4. Mutant flies (Lipid 1) female and Lipid 2 female. A: Obese fly (Lipid 2) female and Lipid 1 female.

Results

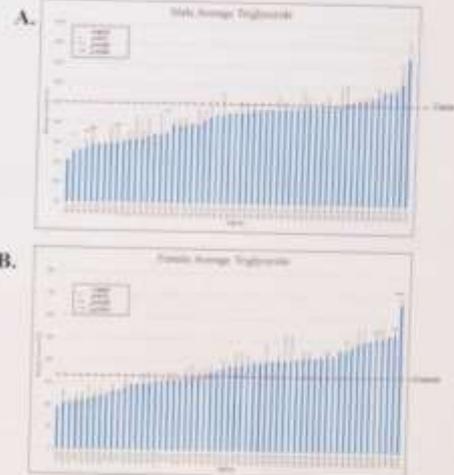


Figure 5. Fat (lipid) molecule average amount of 20 in a wild-type control fly, shown from three standard deviations. A: Obese male triglyceride level. B: Obese female triglyceride level.

Male v. Female

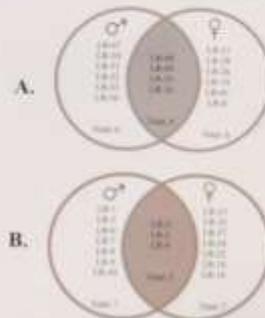


Figure 6. Overlapping genes control of sexual dimorphism on lipid metabolism. A: Obese (miR-93) high triglyceride miRNAs for male and female. B: Obese (miR-101) high triglyceride miRNAs for male and female.

Discussion

- Insulin functions in maintaining energy homeostasis, it controls levels of circulating glucose and regulates storage of resources in the form of glycogen and fat.
- Low fat as a result of miRNA downregulation of insulin signaling could mean that not enough glucose was transported into the cells, mimicking an insulin resistant state and causing cells to turn to the breakdown of fat for fuel.
- High fat is explainable by change in insulin pathway where too much glucose is being brought into the cell and excess stored as fat.
- Dysregulation of the insulin pathway could be a major cause of obesity. This disease may be modifiable by miRNA.

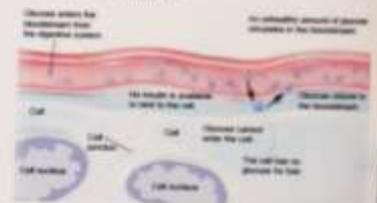


Figure 7. Insulin binds to receptor on the cell membrane. Signal transduction occurs resulting allowing glucose transport to bring glucose into the cell. When insulin receptor state, insulin causes signal the cell to transport glucose. Glucose enters in the blood into the cell to be used in glycogen and triglyceride storage for fuel. What is Insulin Resistance. (Digital image: Understanding, 2-6, Web, 27 March 2017)

Conclusions

- Knockout of specific miRNAs caused nearly 30% increase and decrease in fat compared to control fly.
- Many of the miRNAs that cause a significant change in fat play essential roles in the insulin pathway.
- This study shows that genetically altering the insulin pathway could affect obesity.
- The cure to obesity and even diabetes may be within reach and as simple as manipulating miRNAs that regulate the insulin pathway.

Future Research

- Explore other screening mechanisms to further test miRNA correlation to obesity.

Acknowledgements

This work was supported by RSI/CRU Grant





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POSTER PREP: GENERAL POSTER FORMAT

Abstract and/or
Research
Question

- Summary of your poster
- What is the problem or issue you are investigating/trying to solve?

Background &
Objective

- Objective of your research
- How does your work contribute to existing research on the topic?

Methods

- What did you do?
- Use visual aids when possible

Impact of Wastewater Treatment Plant Effluent on Antibiotic Resistance in Aeromonads



Maegan Dallis, Samantha Henderson, Chrystal Moore, Kelley Dixon, Cindy Cisar

Department of Natural Sciences, Northeastern State University

ABSTRACT

Aeromonads, gram-negative bacteria belonging to the genus *Aeromonas*, are ubiquitous freshwater organisms. Some species of aeromonads are opportunistic human pathogens which often have been linked to gastroenteritis in humans. Our objective of this study was to determine whether wastewater treatment plant (WWTP) effluent contributes to antibiotic resistance in aeromonads. Little is known about the impact of WWTP effluent on antibiotic resistance, one of the world's growing public health problems. In November 2007, Tallapoosa Creek water was analyzed for the presence of aerobics, and bacteria were isolated from creek sediments. Samples were taken upstream and downstream of the Tallapoosa wastewater treatment plant. No antibiotics were detected in the water sample taken upstream of the wastewater treatment plant, but four antibiotics were detected at subtherapeutic levels in the downstream water sample: erythromycin, ciprofloxacin, ofloxacin, and trimethoprim. Distinct isolates from the sediments were identified at least to genus by sequencing their 16S ribosomal RNA genes. Forty-five aeromonad strains were isolated from sediment samples upstream of the WWTP, and twenty-eight aeromonad strains were isolated from sediment samples downstream of the WWTP. These isolates were tested for susceptibility to the antibiotics tetracycline, trimethoprim, and ofloxacin. Seven aeromonads were resistant to trimethoprim (1 upstream, 6 downstream), 6 aeromonads were resistant to tetracycline (2 upstream, 4 downstream), and 4 aeromonads were resistant to ofloxacin (all downstream). Ofloxacin is a second generation *Fluoroquinolone* antibiotic that was approved by the Food and Drug Administration in 1996. We believe that this is the first report of ofloxacin resistance in aeromonads in the United States. Resistance to ofloxacin is of concern because *Fluoroquinolones* are a relatively new class of broad spectrum antibiotics that can be used to treat bacterial infections when older antibiotics fail. We also determined that four of the downstream aeromonad strains exhibited multidrug resistance while none of the upstream strains did. Although this sample size is small, the data indicates a statistically significant increase in the incidence of antibiotic resistance in aeromonads exposed to effluent from the wastewater treatment plant. The Environmental Protection Agency does not currently regulate levels of antibiotics or antibiotic resistant bacteria in effluent released from wastewater treatment plants. Our data indicates that these common components of WWTP effluent may have a significant impact on endemic bacterial populations in these ecosystems.

INTRODUCTION

Bacterial diseases are controlled through the use of antibiotics. Not surprisingly, antibiotics have been reported as the second most commonly prescribed class of drug in the United States. However, antibiotics are often overprescribed or taken inappropriately. Bacteria exposed to antibiotics are constantly evolving. Increased levels of antibiotic in water, the result of widespread use in humans and in agriculture, could lead to the development and spread of antibiotic resistance in bacteria. This would pose problems for infection control and increase healthcare costs. This project examines antibiotic resistance in aeromonads in a freshwater ecosystem that receives effluent from a wastewater treatment plant (WWTP), a potential source of both antibiotics and antibiotic resistant bacteria.

MATERIALS AND METHODS



Colony count - water



Colony count - sediment



Antibiotic susceptibility test

Table 1. Most Probable Number Data¹ for Total and Antibiotic Resistant Coliforms in Water Samples from November 2007

Date	Site ²	Total coliforms	Ampicillin resistant		Ofloxacin resistant		Tetracycline resistant	
			E. coli	Total coliforms	E. coli	Total coliforms	E. coli	Total coliforms
Nov 07	T	28.9 ± 8.1	2,950.0 ± 250	10.1 ± 4.3	4.2 ± 1.1	1,676.7 ± 868.1	23.0 ± 2.7	
	D ³	3,967.7 ± 440.1	2,133 ± 126.7	1,699.4 ± 245.1	89.9 ± 55.0	33.0 ± 2.0	24.0 ± 11.1	

MPNs were determined in water samples using the Colisure® quantitative system (IDEXX Laboratories). Values are MPN per 100 ml water ± SD.
²T is water from Tallapoosa Creek except approximately 0.1 miles upstream of the WWTP. D is the effluent from the Tallapoosa WWTP.
³No data available.
 Tallapoosa WWTP was undergoing repairs on the date the effluent was sampled.

Table 2. Aeromonads Isolated in November 2007¹

Location	Number	Identification ²
Upstream sediment	45	<i>Aeromonas</i> spp. (2), <i>Aeromonas hydrophila</i> (23)
Downstream sediment	28	<i>Aeromonas</i> spp. (15), <i>A. hydrophila</i> (23)
WWTP effluent	1	<i>A. hydrophila</i> (7)

¹MPN values in water of 100-1000 organisms. Numbers in parentheses indicate number of isolates.

Table 3. Antibiotic Susceptibility of Aeromonads Isolated in November 2007¹

Location	Antibiotic	Number	Susceptible / Resistant	Multidrug Resistance
Upstream sediment	Ofloxacin	45	145 of 45 susceptible — 100% 0 of 45 resistant — 0%	none
	Tetracycline	45	113 of 45 susceptible — 85.6% 22 of 45 resistant — 4.4%	
	Trimethoprim	45	146 of 45 susceptible — 87.8% 21 of 45 resistant — 2.2%	
Downstream sediment	Ofloxacin	28	24 of 28 susceptible — 85.7% 2 of 28 resistant — 14.3%	2 resistant to ofloxacin and trimethoprim
	Tetracycline	28	24 of 28 susceptible — 85.7% 2 of 28 resistant — 14.3%	1 resistant to tetracycline and trimethoprim
	Trimethoprim	28	27 of 27 susceptible — 100% 0 of 27 resistant — 0%	1 resistant to tetracycline, trimethoprim and ofloxacin

¹See above text for details regarding antibiotic resistance. Colony density, antibiotic resistance positive. The antibiotic is on the axis.

SOCIETAL IMPACT

Antibiotic resistant pathogens are a serious threat to human health. We have determined that wastewater treatment plant effluent, a source of antibiotics and antibiotic resistant bacteria, can contribute to antibiotic resistance in downstream bacterial populations. Development of best practices to reduce the amounts of antibiotics and antibiotic resistant bacteria released into the environment may help in preventing the spread of antibiotic resistance in bacteria.

RESULTS

In November 2007 four antibiotics were present in Tallapoosa Creek water samples collected downstream of the WWTP: erythromycin (0.000 µg/L), rifampin (0.006 µg/L), ofloxacin (0.039 µg/L), and trimethoprim (0.024 µg/L). No antibiotics were detected upstream of the WWTP. In addition, antibiotic resistant bacteria were present in Tallapoosa Creek water and in WWTP effluent (Table 1). Many bacteria isolated from Tallapoosa Creek sediments in November 2007 were identified as aeromonads (Table 2). Forty-five aeromonad strains were isolated from sediment samples upstream of the WWTP and 28 aeromonad strains were isolated from sediment samples downstream of the WWTP. Of these, 7 strains were resistant to trimethoprim, 5 strains were resistant to tetracycline and 4 strains were resistant to ofloxacin. Several of the downstream aeromonad isolates were resistant to more than one antibiotic and one downstream aeromonad was resistant to two additional antibiotics (Table 3). Numbers of antibiotic resistant aeromonads were compared using a chi-square contingency test with Yates correction for small sample size. There were significantly more antibiotic resistant aeromonads present in sediments downstream of the WWTP than upstream of the WWTP in November 2007 ($P = 0.011$).

DISCUSSION

- Antibiotics and antibiotic resistant bacteria were both present in this freshwater ecosystem. However, antibiotic resistant aeromonads were more likely to be found downstream than upstream of the WWTP suggesting that WWTP effluent contributes to antibiotic resistance in aeromonads.
- Roughly equal numbers of bacteria were isolated from sediments upstream and downstream of the WWTP, but the ratio of aeromonads to other bacteria was lower in the downstream bacterial population. Therefore, although more likely to be resistant to antibiotics the downstream aeromonad population appeared to be negatively impacted by the WWTP effluent.
- Four aeromonad isolates from downstream of the WWTP were resistant to ofloxacin. To our knowledge, this is the first report of ofloxacin resistance in aeromonads in the United States.

We are currently analyzing the genes responsible for antibiotic resistance in the aeromonad strains. Ultimately, we plan to quantify the rate of occurrence of horizontal transfer of antibiotic resistance in bacteria in the environment, identify the transfer mechanism(s) involved, and assess the impact of environmental reservoirs of antibiotic resistance on human pathogens and disease.

ACKNOWLEDGEMENTS

Funding was provided by the Oklahoma Center for the Advancement of Science and Technology, OKRS award IIR07-124, and by NSF-NERC grant F0200016478-04.



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POSTER PREP: GENERAL POSTER FORMAT

- Use legends that explain your figures

Charts & Figures

Discussion

- Use illustrations to support/explain results

Results

- Also called “conclusions”
- Explain what your results indicate
- Outline future project plans



A Novel Assay to Predict Cancer Resistance to Cisplatin

Lacy Brame¹, Vengatesh Ganapathy¹, Ilangovan Ramachandran¹, Lurdes Queimado¹⁻⁵

Departments of ¹Otorhinolaryngology, ²Cell Biology and ³Pediatrics; ⁴The Oklahoma Tobacco Research Center and ⁵The Peggy and Charles Stephenson Cancer Center, The University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104, USA.



Introduction

- Cisplatin is widely used as chemotherapy drug that induces DNA damage and ultimately triggers apoptosis. However, therapeutic resistance and tumor relapse remains a significant clinical problem.
- Recently, our laboratory developed an assay (Fig. 1) called primer anchored DNA damage detection assay (PADDA) that screens genomic areas for DNA damage¹. PADDA has been shown to detect a dose-dependent increase in DNA damage caused by genotoxic agent (Fig. 2).
- We hypothesized that PADDA will discriminate the ability of cancer cells to repair damage induced by cisplatin, and therefore predict cancer resistance to cisplatin.

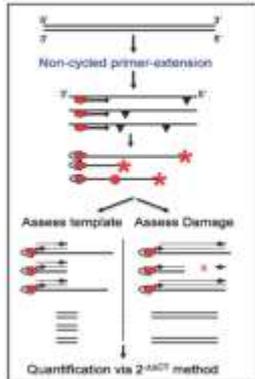


Figure 1. Diagram of PADDA. A single strand-specific non-cycled primer extension performed with a 5'-biotin-tagged primer and Vent exo- DNA polymerase identifies damaged nucleotides (inverted triangles), and generates a pool of highly specific biotin-tagged extended products, each of them derived from one strand of a single DNA molecule. Each extended product has a stop, which represents replicative arrest by a damaged nucleotide or nick. Some extended products will contain misincorporations that represent polymerase lesion-by-pass with misincorporation. After several purification steps, the strand-specific, biotin-bound extended products can be used for damage quantification on a high throughput setting q-PADDA.

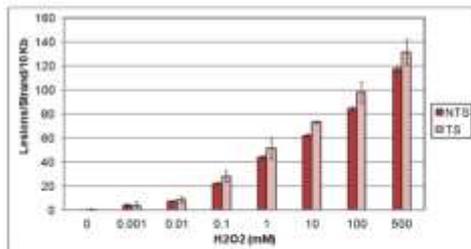


Figure 2. Quantification of induced DNA damage after in vitro exposure to a dose escalation of H₂O₂. Strand-specific DNA damage was quantified by q-PADDA. Lesion frequency was estimated via Poisson equation. NTS, non-transcribed strand; TS, transcribed strand; Data represents Mean \pm S.E.M.

Aim

To define the levels of DNA damage induced at p53 nucleotides by cisplatin treatment and to measure the ability of cancer cells to repair damage induced by cisplatin.

Materials & Methods

PADDA was used on a high-throughput setting to quantify DNA damage in human oral cancer cells (SCC-1) exposed to different doses of cisplatin. Cell viability was determined by 3-[4,5-dimethylthiazol-2-yl]-2,5 diphenyl tetrazolium bromide (MTT) assay. Data was analyzed by Student's *t*-test.

Results

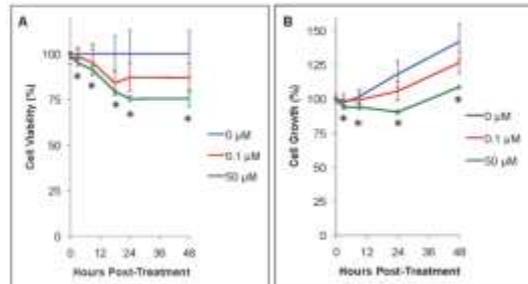


Figure 3. Cell viability assay and cell growth. SCC-1 cells were treated with 0 μ M, 0.1 μ M, 50 μ M concentrations of cisplatin and allowed to repair damage for 0, 3, 9, 18, 24 & 48 hour time intervals. Cell viability (A) and cell growth (B) were determined by MTT assay. Data shown as Mean \pm S.D. * $p < 0.01$.

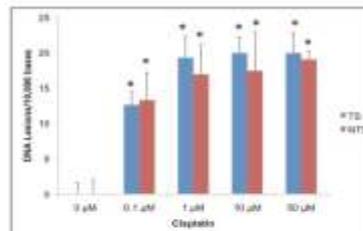


Figure 4. DNA damage measured by q-PADDA in SCC-1 cells exposed to cisplatin for 3 hours. Damage was quantified by q-PADDA in both transcribed (TS) and non-transcribed (NTS) strands. Data shown as Mean \pm S.E.M. * $p < 0.01$.

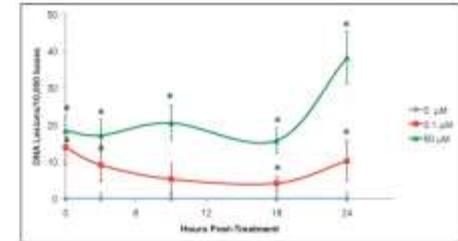


Figure 5. DNA damage measured by q-PADDA in SCC-1 cells after exposure to cisplatin. SCC-1 cells were treated with 0 μ M, 0.1 μ M, 50 μ M concentrations of cisplatin and allowed to repair damage for 0, 3, 9, 18 & 24 hour time intervals. Data shown as Mean \pm S.E.M. * $p < 0.01$.

Conclusion & Societal Impact

- PADDA was able to quantify DNA damage and repair after cisplatin treatment. This information will allow us to determine if resistance to cisplatin is due to effective damage removal or to damage tolerance. This data would facilitate the development of strategies targeting the mechanism of drug resistance.
- This observation has significant clinical importance as it can be used to predict treatment response and direct treatment selection in cancer patients.

Future Directions

- This assay has potential to elucidate the differential efficacy of cisplatin as a chemotherapy drug and act as a preliminary screening method to determine differential cisplatin resistance.
- This project can be extended to determine the genotoxicity and resistance of cisplatin in other head and neck cancer cell lines.
- q-PADDA can be used to determine if patients will respond or become resistant to not only platinum-based chemotherapy treatments, but also to other treatments that induce DNA damage.

Acknowledgement

Funding was provided by the Oklahoma Tobacco Research Center and the Oklahoma Center for the Advancement of Science and Technology. Dr. Queimado holds a Presbyterian Health Foundation Endowed Chair in Otorhinolaryngology.

References

- Reis AM, Mills VK, Ramachandran I, Friedberg EC, Thompson D and Queimado L. Targeted detection of in vivo endogenous DNA base damage reveals preferential base excision repair in the transcribed strand. *Nucleic Acids Res.* 40(1): 206-219, 2012.



2019 Research Day at the Capitol

POSTER PREP: GENERAL POSTER FORMAT

- DO NOT OVERLOOK THIS SECTION!!!
- Arguably one of the most important
- 2-3 concise sentences
- Explain the social benefits of your research in layman's terms

Societal Impact

Acknowledge your:

- Funding source(s)
- Collaborators (big and small)
- Journal articles used as references
- EPSCoR

Acknowledgements



Development in Potential Anti-HIV & Antimetastatic Drugs: C-Symmetric Tris-Linked Bridged Tetraazamacrocycles as Potential CXCR4 Antagonists

Courtney D. Garcia¹, B. N. Shockey¹, B. Gridley², S. J. Archibald¹, Dominique Schols³, T. J. Hubin³

1. Department of Chemistry, Southwestern Oklahoma State University, 100 Campus Drive, Weatherford, OK 73096 USA

2. Department of Chemistry, University of Hull, Cottingham Road, Hull, HU6 7RX, UK

3. University of Leuven, Belgium.

1. Societal Impact:

CXCR4 chemokine receptors are found on the surface of immune, and other, cells, and together with the specific natural ligand, CXCL12, have been revealed to play a role in a number of disease states. CXCR4 expression has also been reported in at least 23 different cancers. Target organs for breast metastases such as liver, lung, and bone have high levels of CXCL12. Due to the wide-ranging potential biomedical applications that might result, our aim is to develop new antagonists for the CXCR4 co-receptor.

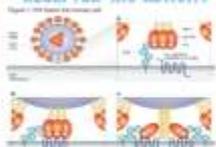
2. Objectives:

Our objectives were to synthesize C3-symmetric tris-linked analogues of our most effective bis-tetraazamacrocycles (metal complexes) and to characterize their chemical and physical properties in preparation for determining if the added macrocyclic enforces their antagonism of CXCR4.

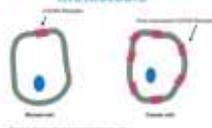
3. Methods:

Synthetic routes extending our bis-linked ligand syntheses to use the C3-symmetric linker 1,3,5-trisubstituted benzene were developed. Copper(II), nickel(II), cobalt(II), and zinc(II) complexes were made using our previous methods. Electropray mass spectra, UV-Visible spectra, cyclic voltammograms, magnetic moments, X-Ray crystal structures, and ¹H and ¹³C NMR spectra were collected to characterize the complexes.

RECEPTOR-HIV ACTIVITY

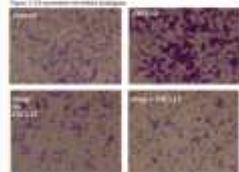


CXCR4 and Cancer Cell Metastasis



ANTI-CANCER ACTIVITY INVASION ASSAYS

- Cell invasion assays in response to chemokine gradient.
- Initially used U937 cells.
- Experiments run in presence and absence of antagonist.



Restrict to one configuration

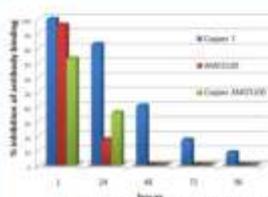
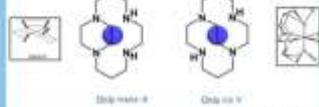
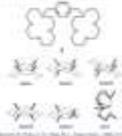
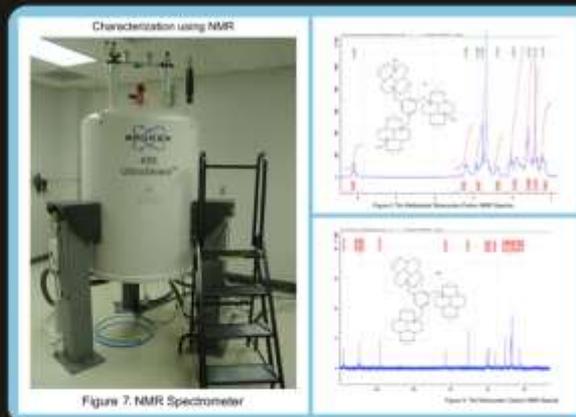
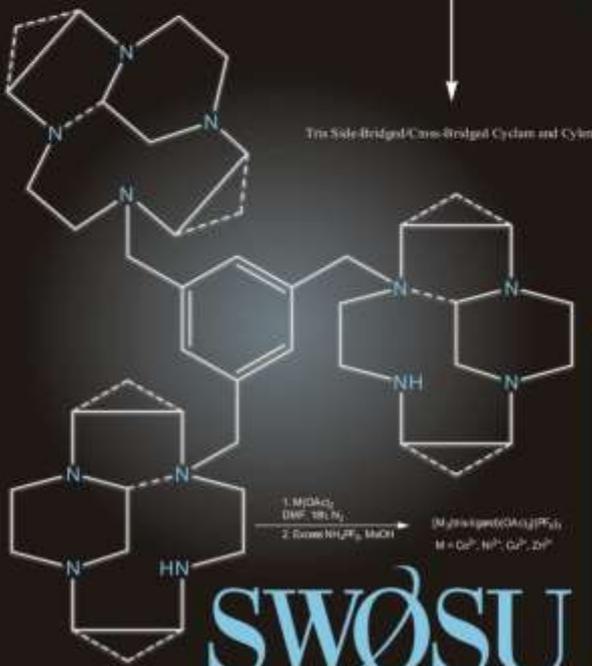


Figure 5. The inhibition of anti-CXCR4 antibody binding over time after exposure to SHM of the drug. A population of 100,000 cells was incubated for each data point and analyzed by flow cell cytometry using a secondary fluorescein tagged IgG antibody (negative values are not shown).

Synthesis of C3-symmetric tris-linked analogues



Experiment	log K CuCl ₂	log K CoCl ₂
Dly-1	0.05	1.19
Ni-5	0.22	16.3
Dly-3	0.57	6.7
Dly-3	3.93	15.74
Dly-4	0.12	15.99
Dly-5	0.26	16.84
Dly-6	0.44	17.79
AMD006	0.011	0.0000
AMD051	0.1	0.1

Figure 10. Binding Experiments: CXCR4 & CCR5

4. Results:

The ligand syntheses of the side-bridged and cross-bridged C₃-symmetric ligands proceeded similarly to the previously developed bis-linked routes. Complexation with the desired metal ions proceeded as expected. Characterization of the metal complexes resulted in distinguishable quality purity in each step of synthesis. Experiments investigating the Calcium release have shown that the C₃-symmetric compounds are highly potent as CXCR4 antagonists, but as in the bis-linked compounds. An unexpected benefit of this linking is CCR5 binding. CCR5 is another important chemokine receptor.

5. Conclusions:

C₃-symmetric tris-linked bridged tetraazamacrocycles are easily produced, using an appropriate linker and following synthetic methods adopted from the bis-linked analogues. Metal ion complexation proceeds smoothly following known procedures. Calcium ion release is observed when the natural ligand for CXCR4, CXCL12, binds. Preventing Calcium release is evidence of strong antagonism by the potential drug molecule. Also, several of the C₃-symmetric compounds have demonstrated excellent antagonism of a related chemokine receptor, CCR5, as well. This exciting result may lead to a new class of dual chemokine receptor antagonists.

6. Future plans:

Experimental data on the specific disease states of HIV infection and cancer with the resulting complexes will inform our understanding of the requirements for producing even more efficient CXCR4 antagonists of this class.

Poster Examples: “Do’s” and “Don’ts”

Strengths:

- Logical order
- Various visual aid types
- Acknowledgements

Weaknesses:

- Sections & images not aligned
- Distracting background
- Too many visual components

Parasite Spillover Effects on Native Communities in New Zealand Streams and Lakes

Richard Patterson, The University of Otago Department of Zoology, Bridging Disciplines Program, Evolutionary and Ecological Parasitology Group

The University of Texas at Austin, The Science Institute

Presented at the 2010, 11th International Parasitology Conference

What is parasite spillover?

Parasite spillover is a process that describes the transfer of native parasites from new host species to native hosts.

- First, native parasites infect introduced or invasive host species.
- With a new host, parasites flourish.
- Now, parasites return to native species with increased infection and disease rates.

Substrate shows that Salmo trutta originating from Europe and rainbow trout Oncorhynchus mykiss (North American) were first introduced to New Zealand waters in the late 19th century. Their effects on local and native stream communities as a non-indigenous species include lesser-studied effects such as parasite spillover and disease.

Invited by: J.M. Brown, A.A. Townsend, C.C. Meek, E.B. Squires, N.M. Williams, and J. Williams

Could parasite spillover be a cause of native species loss and local level extinction?

Fig. 1. Making clear use of a sub-diagram to show a sub-diagram.

My Experience

I spent five months learning with this project, conducting various lab and field tests. In the laboratory, I learned the importance of (lab: health) hygiene and safety. I also conducted field studies on growth, survival, and health. In the field, I helped to set nets and traps for fish. We also collected benthic invertebrate and zooplankton samples.

Discussion

Native species loss is a critical issue throughout the world in many different environments. This may stem from Conservation International's biodiversity hotspots which are at least 50 percent of native species are already lost. The most biodiverse regions, including New Zealand, are also the ones most at risk.

Competition and predation are the traditional impacts of invasive species on native species, but disease-driven impacts are becoming more widely recognized and researched. Whereas parasite spillover is already an accepted form of disease-driven impact, parasite spillover can potentially be more widely used as a tool for describing and understanding impacts of invasive species and native species loss.

A parallel study with similar methods is currently being conducted by the same team of researchers in Argentina. Other areas of the world where parasite spillover has been researched include a study of competing native and invasive grasshopper populations in California (Gittle and Wilson 2003) with more awareness of this issue, more research and studies will hopefully begin and consider parasite spillover as a potential cause for native species loss, potentially helping reverse the trends in global biodiversity.

Objectives

1. Test whether the presence of brown trout Salmo trutta and their parasite abundance is correlated to increased infection rates in four native species fish.
2. Identify for native fish and brown trout seasonal variations in infection intensity.
3. Understand the impact of parasites on host's condition, survival, and reproductive potential through-captivity experimentation for all four species. Parasite transmission, establishment, and mortality in different host species will also be identified.
4. Use multi-host and shared-parasite stochastic simulation models.
5. Consider global implications of this model by applying it to an Argentine system and conducting a literature survey of the abundance of shared parasites in native and exotic freshwater fish.

Methods

- Analyze freshwater fish communities in lakes and streams
- Field surveys
- Host-substrate infection trials
- Mathematical modeling

Lake Taupo, Lake Taumoko, Lake Taupo, Upper Taupo Stream

Presented by: J.M. Brown, A.A. Townsend, C.C. Meek, E.B. Squires, N.M. Williams, and J. Williams

2010, 11th International Parasitology Conference

Acknowledgements

Richard Patterson, The University of Otago Department of Zoology, Bridging Disciplines Program, Evolutionary and Ecological Parasitology Group

The University of Texas at Austin, The Science Institute

Presented at the 2010, 11th International Parasitology Conference

Systematic reviews of animal experiments demonstrate poor human clinical and toxicological utility

ATLA: Alternatives to Laboratory Animals, 2007; 35(6): 641-669



Andrew Knight BSc, PhD, DSc, FRCGS
Laboratory Director
Lancaster, UK, andrew.knight@lancaster.ac.uk



INTRODUCTION

What is a systematic review?

Systematic reviews of animal experiments, and usually human clinical trials, are generally considered to be the highest quality of evidence available for assessing a treatment's safety and efficacy.

Claims supporting laboratory animal use

Systematic reviews using laboratory animals in safety assessments of human regulatory claims have been used to support the safety of a wide range of drugs. However, the evidence base for these claims is poor. In fact, the majority of these reviews are of such low quality that the majority of laboratory animals used in safety assessments are unnecessary. This paper reports the results of a systematic review of the safety of human regulatory claims supported by laboratory animal safety assessments.

THE QUALITY OF EVIDENCE REVIEWED

The quality of laboratory animal studies is generally poor. The majority of human regulatory claims supported by laboratory animal studies are of such low quality that the majority of laboratory animals used in safety assessments are unnecessary. This paper reports the results of a systematic review of the safety of human regulatory claims supported by laboratory animal safety assessments.

METHODS

The 20 most frequently cited systematic reviews of the safety of human regulatory claims supported by laboratory animal studies were identified. The quality of the evidence reviewed in these reviews was assessed using a standardised quality assessment tool. The quality of the evidence reviewed in these reviews was assessed using a standardised quality assessment tool.

RESULTS & DISCUSSION

The 20 most frequently cited systematic reviews of the safety of human regulatory claims supported by laboratory animal studies were identified. The quality of the evidence reviewed in these reviews was assessed using a standardised quality assessment tool.

Causes for the poor human utility of animal evidence

There are a number of reasons why laboratory animal studies are of such low quality. These include: poor experimental design, poor quality of data, poor quality of analysis, and poor quality of reporting.

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Conclusions

The majority of laboratory animal studies are of such low quality that the majority of laboratory animals used in safety assessments are unnecessary. This paper reports the results of a systematic review of the safety of human regulatory claims supported by laboratory animal safety assessments.

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Systematic reviews of laboratory animal studies are of such low quality that the majority of laboratory animals used in safety assessments are unnecessary. This paper reports the results of a systematic review of the safety of human regulatory claims supported by laboratory animal safety assessments.

Methodological quality

The quality of the evidence reviewed in these reviews was assessed using a standardised quality assessment tool. The quality of the evidence reviewed in these reviews was assessed using a standardised quality assessment tool.

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Limitations

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Conclusions

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CONCLUSIONS

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REFERENCES

1. Knight A. (2007) Systematic reviews of laboratory animal studies are of such low quality that the majority of laboratory animals used in safety assessments are unnecessary. *ATLA*, 35(6): 641-669.



An example of why you should NOT use a photo or graphic as your poster background.

Text is impossible to read and potential observers would be too distracted by the image to sort through the information anyway.

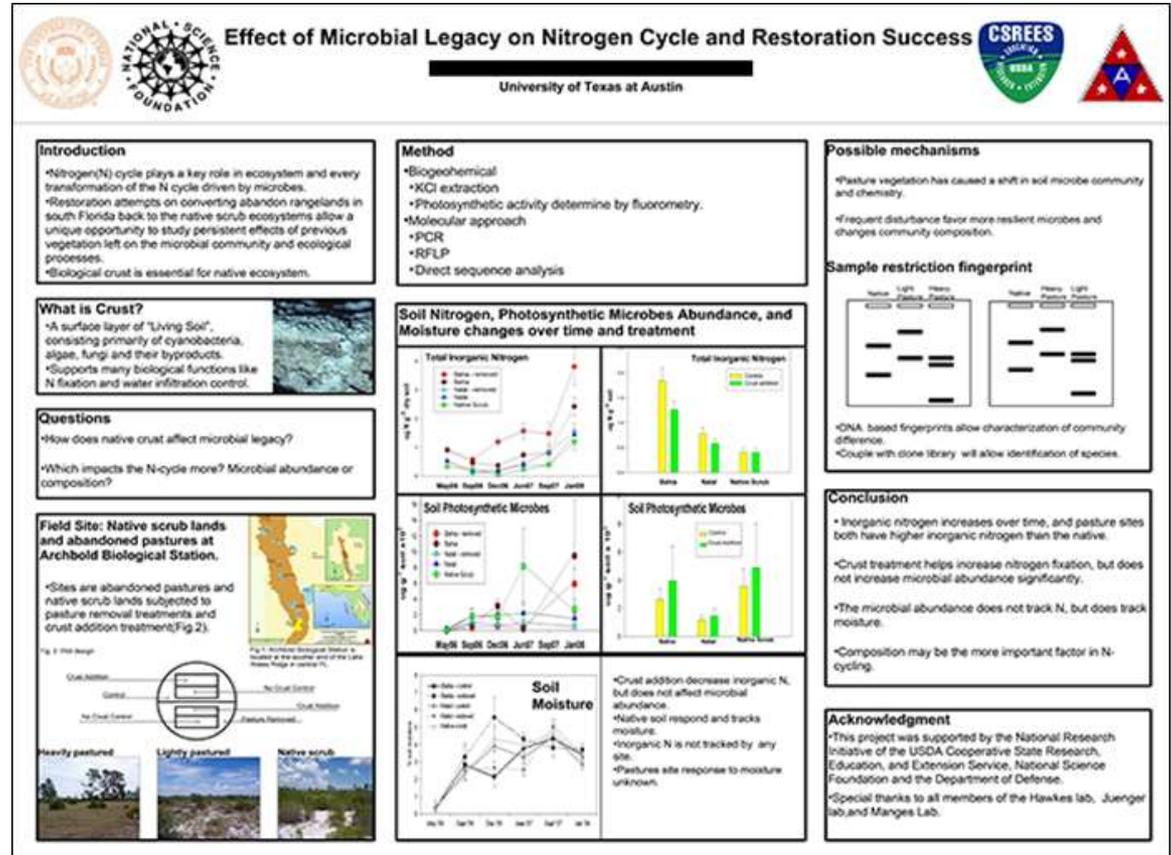
Poster Examples: “Do’s” and “Don’ts”

Strengths:

- Clearly defined research questions
- Effective use of visual aids
- Clear organizational structure
- Bullets break up text

Weaknesses:

- Technical language & undefined acronyms (limits audience)
- Narrow margins within text boxes
- Too many thick borders around boxes
- Uses incorrect logo for the institution



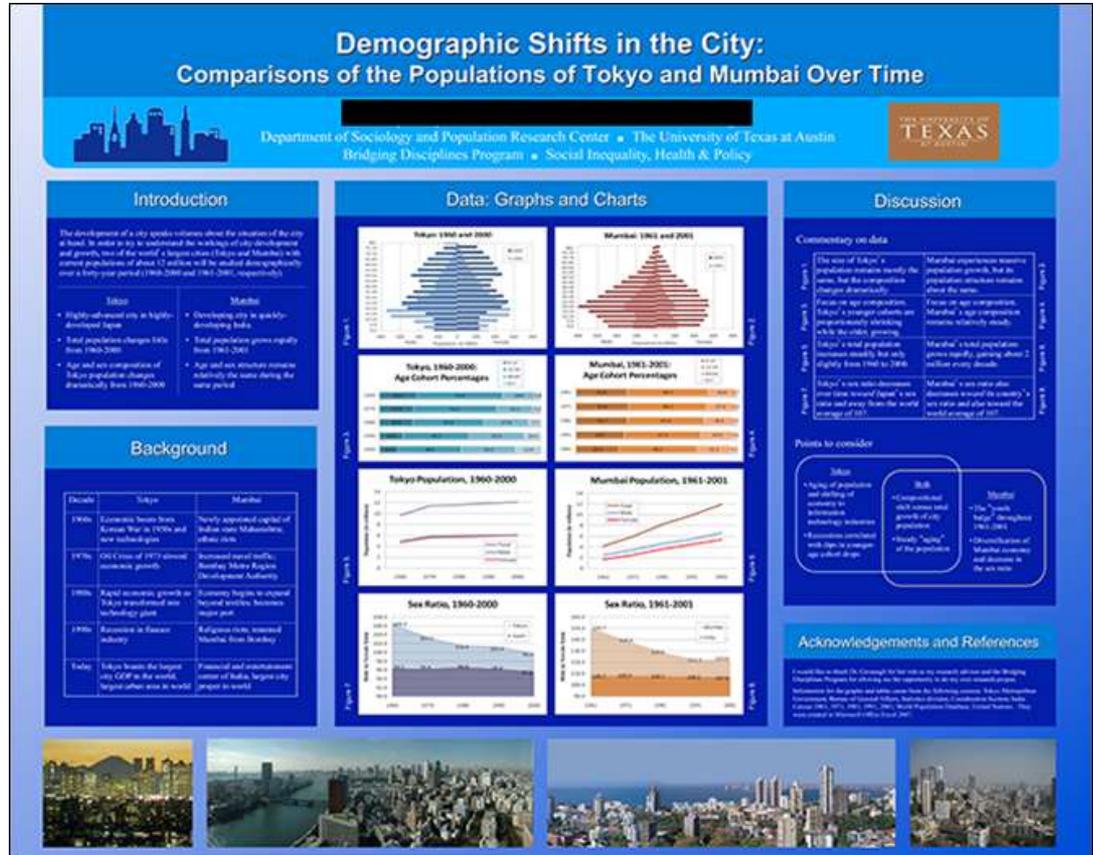
Poster Examples: “Do’s” and “Don’ts”

Strengths:

- Venn diagram in discussion
- Consistent graphics
- Multiple types of visual aids

Weaknesses:

- Light text on dark background
- Color backgrounds should be avoided, especially dark ones
- Unlabeled, non-credited photos



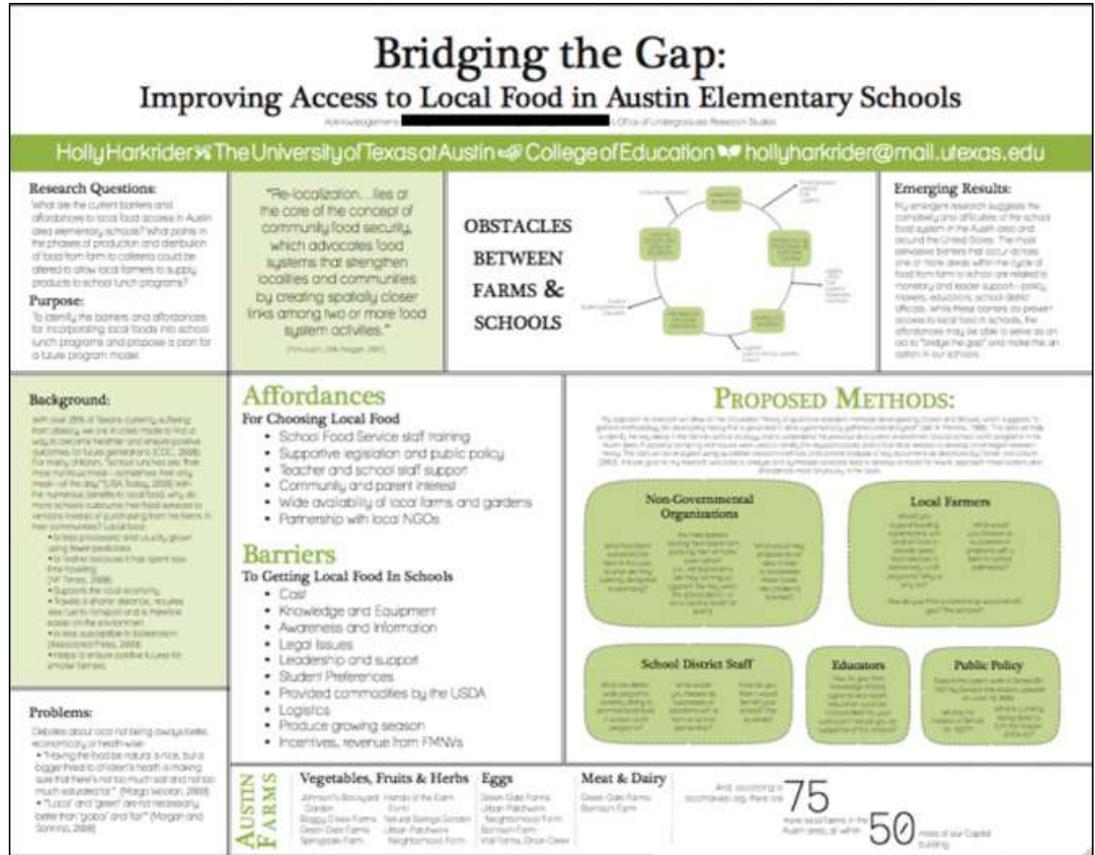
Poster Examples: "Do's" and "Don'ts"

Strengths:

- Easy to read
- Clearly defined research question
- Use of white space
- Simple color scheme
- Use of shapes, figures, bullets to break up text
- Compelling title (and title font size)
- Clean visual impression

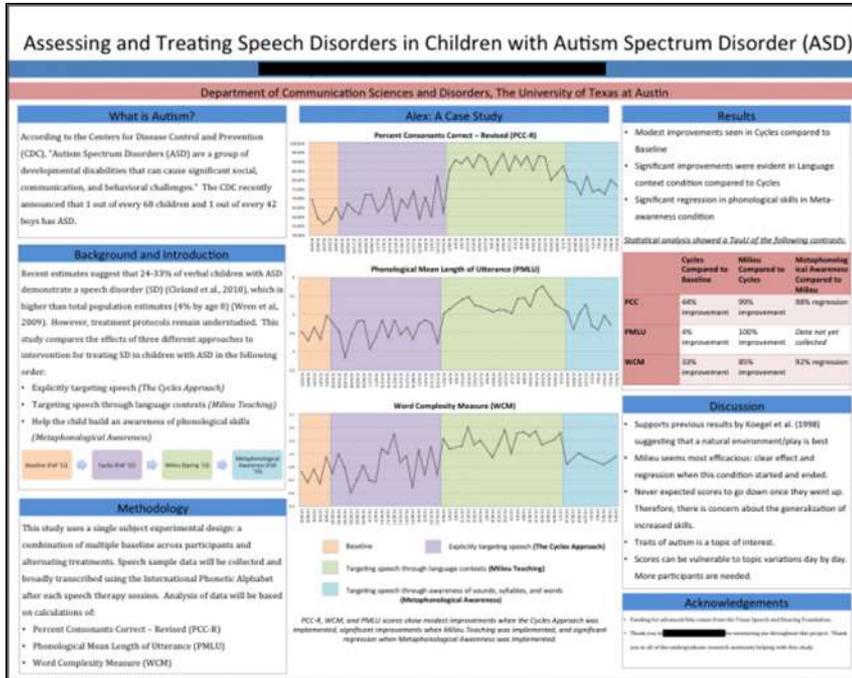
Weaknesses:

- Many sections without a clear flow between them
- Lacks acknowledgements

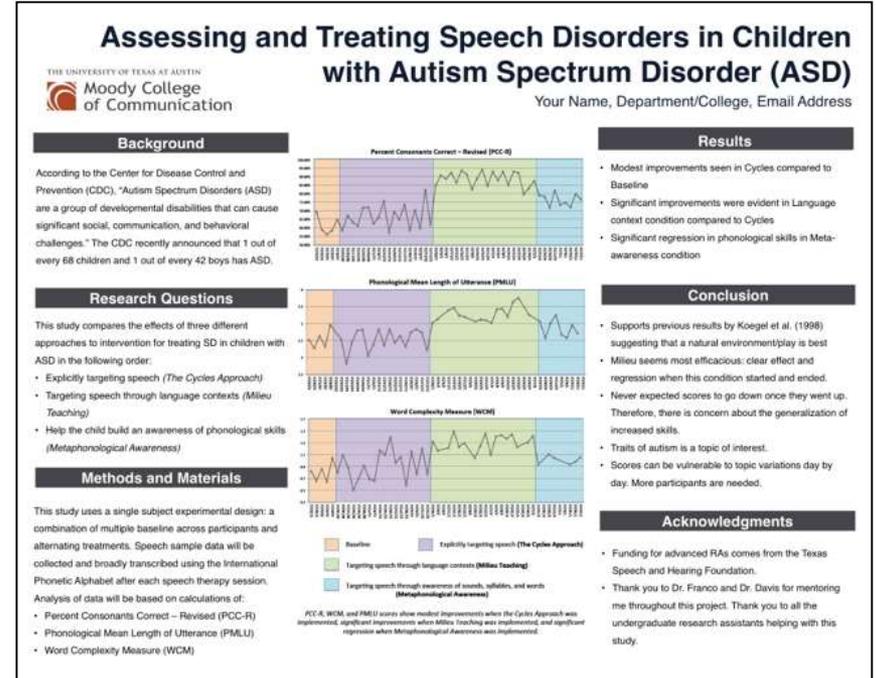


Poster Examples:

Before



After





An Online Poster Prep Resource

<https://ugs.utexas.edu/our/poster>

The University of Texas at Austin's online [Poster Guide](#) is a great resource, providing thorough and easy-to-understand scientific poster design tips and instruction.

- [Guide to Creating Research Posters](#)
- [Poster Samples: What to do and what not to do](#)
- [Poster Content Development](#)
- [Organizing Poster Content](#)
- [Poster Design Elements and Guidelines](#)
- [Review Your Poster](#)
- [Printing Your Poster](#)
- [Presenting Your Poster](#)
- [Talking About Your Poster](#)



2019 Research Day at the Capitol

ONLINE REGISTRATION REQUIRED (BY MARCH 11)

All student researchers & anyone who will be attending Research Day at the Capitol activities in support of the student researcher must register online at:

<http://www.okepscor.org/2019-research-day-capitol-participant-sign-form-guests-faculty-mentors-student-researchers>

- Please advise parents, friends, family, faculty advisors, etc. to register online (or you may register online for them)
- Why? This event is funded through a grant from the National Science Foundation. NSF requires participant information to continue funding for the event.
- Registration deadline: March 11



2019 Research Day at the Capitol

FINAL THOUGHTS—THINGS TO REMEMBER

You were chosen for a reason!

- Be ready and mentally prepared—practice!
- Emphasize your societal impact
- Dress professionally and be punctual
- Know your Legislators and engage them
- Judges are looking for someone who has the whole package!



2019 Research Day at the Capitol

INSIGHT FROM A PAST WINNER

Devin Laurence, Grand Prize Winner 2018

University of Oklahoma

Poster Topic: Cardiovascular Biomechanics





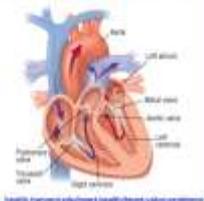
An Integrated Experimental-Computational Approach for Multiscale Investigations of Atrioventricular Heart Valves With Applications to Individual-Optimized Surgery Planning

Devin Laurence and Chung-Hao Lee, Ph.D.

Biomechanics and Biomaterials Design Laboratory, School of Aerospace and Mechanical Engineering, The University of Oklahoma

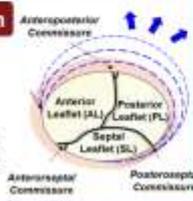
Heart Valves and Their Function

- Four heart valves are located in the heart to regulate unidirectional blood flow.
- Fully leaflet closure is critical to ensure the heart is not overworked, causing further damage.



Valvular Heart Disease – Regurgitation

- Inability of leaflet closure deteriorates the heart function for preventing the corresponding backflow.
- Heart valve regurgitation is a compounding complication, with symptoms oftentimes lowering the quality of life.



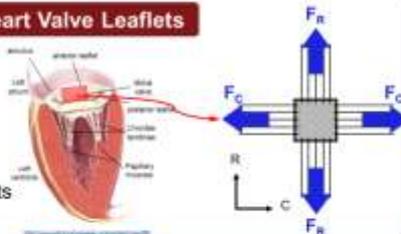
Surgical Repair for Heart Valve Regurgitation

- Surgical installation of an annuloplasty ring around the valve leaflets restores the original shape and proper leaflet closure.
- **Recurrence rate approaches 15%-20% 3-5 years after initial surgical repair**^[1-3]



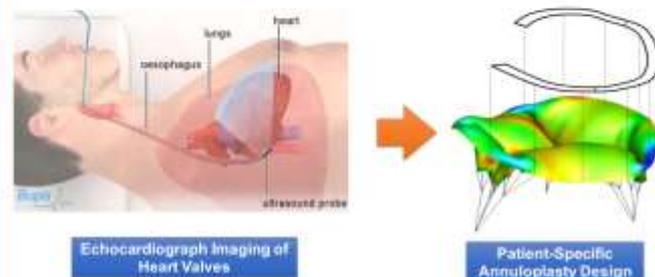
Characterization of Biomechanical Properties of Heart Valve Leaflets

- Accurate computational models demand extensive characterization of the biomechanical properties of the heart valve leaflet tissues.
- The intricate microstructure of the leaflets results in a complex mechanical response, which cannot be simply characterized by unidirectional testing.
- We can comprehensively capture this complex mechanical behavior of the leaflets by performing biaxial mechanical testing with simulated physiological loads.



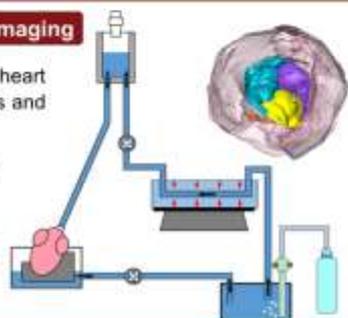
Societal Impact: Patient-Specific Surgery Planning

- The developed computational model will be extended for investigations into patient-specific modelling of the heart valves.
- These investigations enable **patient-specific, simulation-informed**, pre-surgical recommendations to improve the durability of valve surgical repair.
- The increase in repair durability and longevity will significantly benefit the ~50,000 Americans who undergo treatment annually.



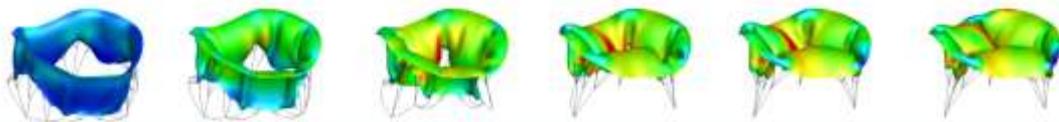
Ex-Vivo Beating Heart Apparatus for Functional Cardiac MR Imaging

- The Langendorff beating heart model facilitates ex-vivo investigations into the heart function; however, it has typically been used in small animal studies, such as for rats and rabbits, but is not comparable to human anatomy.
- Imaging of living animals can obscure leaflet geometry and does not provide key modeling parameters such as ventricle pressure and heart temperature.
- Combining the previously developed Langendorff beating heart apparatus with high-resolution MR imaging for large animal hearts (e.g., pig or sheep), we can obtain high-fidelity valve geometries for more accurate modeling.



Multiscale Computational Model for Patient-Specific Heart Valve Surgery Simulations

- 3D multiscale computational models will be developed using the previously acquired mechanical data and valve geometry.
- These developed computational models will provide insight into the effect of tissue-level modifications (annuloplasty repair or valve regurgitation) on the underlying microstructure and overall organ function.
- Furthermore, the developed multiscale model will elucidate mechanisms behind regurgitation initiation and progression.



Acknowledgments

We greatly appreciate the support from the American Heart Association Scientist Development Grant (16SDG2776014) (CHL), the O.U.R. Mentored Research Fellowship (DL), and OU UROP (DL). We also acknowledge graduate student Samuel Jett for his work with the biaxial testing as well as graduate student Mehri Mobaraki and undergraduate students McKenzie Makovec, Daniel Tsai, and Cortland Johns for their assistance with the development of the Langendorff beating heart model.

References

- [1] Condado, et al., *Journal of Interventional Cardiology*, 16(6):523-534, 2003.
- [2] Flameng, et al., *Circulation*, 107(12):1609-1613, 2003.
- [3] Taramasso, et al., *European Heart Journal*, 38(9):639-647, 2016.



2019 Research Day at the Capitol

INSIGHT FROM A JUDGE

Sherry Marshall

President & CEO, Science Museum Oklahoma

Educational background: Physics, with additional emphasis in Chemistry, Applied Behavioral Science in Education, and Curriculum and Instruction

