RECOGNIZING OUTSTANDING UNDERGRADUATE RESEARCH

2017 Research Day at the Capitol

STUDENT INFORMATION/TRAINING SESSION

2017 Research Day at the Capitol

MARCH 27-28, 2017 * WATERFORD HOTEL & STATE CAPITOL

Congratulations for being selected to represent your institution at the 22nd 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 Experimental 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 about undergraduate student research

2017 Research Day at the Capitol

LET'S HEAR ABOUT YOU! GIVE US THE ELEVATOR PITCH

Tell the Group (in 45 seconds or less)

- WHO YOU ARE
- WHAT INSTITUTION YOU'RE REPRESENTING
- WHAT YOU'RE RESEARCHING
- WHAT THE SOCIETAL IMPACT IS

Remember…not everyone is familiar with your area of expertise, so don't use area-specific lingo or jargon.

OKLAHOMA EPSCoR

Research Day at the Capitol

Research Day at the Capitol

WE’RE EVOLVING AND ADAPTING!

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YOUR OBLIGATIONS FOR THIS EVENT

Your obligations consist of:

March 27 (Waterford Hotel)

- 3-minute oral presentation (judged)
- Poster session & judging (w/3 minutes of Q&A)

March 28 (State Capitol Building)

- Visiting your Legislators in their offices
- Awards ceremony attendance

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TIMELINE OF IMPORTANT DATES

Nov. 13, 2016 – March 26, 2017	Students prepare scientific posters & oral presentations
Monday, February 6, 2017	Students' revised abstracts and lodging requests are due; online registration closes
Monday, March 27, 2017	Poster session and oral presentation/poster judging 4:00 – 8:30 p.m. Waterford Hotel, Oklahoma City
Tuesday, March 28, 2017	Students visit Legislators' offices 8:00– 10:45 a.m.; noon – 1 p.m. State Capitol, Oklahoma City
Tuesday, March 28, 2017	Awards Ceremony 11 a.m. – noon Blue Room, State Capitol, OKC Student Participation Mandatory

All March 27-28 activities are mandatory for student researchers; registered guests are invited to participate.

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MONDAY, MARCH 27 * WATERFORD HOTEL, OKC

4:00 – 6:00 p.m.	Check-in for oral presentation judging—individually scheduled times Take your poster with you! (*Waterford Hotel, Current Room*)
4:00 – 6:00 p.m.	Set up your poster immediately following your oral presentation; return by 6:10 p.m. (*Waterford Hotel, Grand Ballroom*)
6:10 p.m.	Return to Grand Ballroom and prepare for poster session (*Waterford Hotel, Grand Ballroom*)
6:30 – 8:30 p.m.	Poster session & poster judging *Registered guests & students* (*Waterford Hotel, Grand Ballroom*)
8:30 p.m.	Adjourn for the night

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MARCH 27 POSTER SESSION HOUSEKEEPING ITEMS

❖ The following will be provided for you:
- Easel to display your poster
- Firm board to attach your poster to (maximum poster dimensions: 48”x36”)
- Attachment clips

❖ Due to space constraints only your poster may be displayed. We unfortunately cannot accommodate:
- Display tables or materials
- Laptops
- Any additional floor space items

❖ Label your poster tube, as it will be placed in an area with all of the other containers in order to keep the presentation area neat.

❖ Please don’t bring a lot of extra personal items into the room, as it will “junk up” your area.

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HOTEL ACCOMMODATIONS (REQUEST DEADLINE FEB. 6)

EPSCoR will provide lodging on the evening of Monday, March 27 for student participants who live outside the OKC metro area and who have requested lodging prior to the February 6th deadline.

- *Conference hotel: Waterford Hotel, OKC*
  When your request for lodging is received, the EPSCoR office will book the room for you; EPSCoR will make direct payment to the hotel for your room; self-parking is free, valet is not covered.
- Hotel will require a credit/debit card from students at check-in to cover any incurred incidental charges
- If you wish to have a guest stay in the room with you, they may do so at no additional charge
- Notify me no later than Feb. 6 if you wish for me to secure a room on your behalf—a sign up sheet is available today (choose a single king or two queen beds)
- Confirmation numbers will be issued to you in February

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TUESDAY, MARCH 28 * STATE CAPITOL OF OKLAHOMA

8:00 -10:45 a.m.	Student researchers meet their Legislators (Legislator offices) (Pre-scheduled meetings will have been made for you by the OSRHE office when possible.)
10:50 a.m.	Arrive in Blue Room, 2nd Floor
11:00 - noon	Awards ceremony (Blue Room, 2nd Floor)
Noon – 1:00 p .m.	Final meetings of student researchers with Legislators (Legislator offices)

Visiting Your Legislator's Office

MARCH 28, STATE CAPITOL [WWW.OKLEGISLATURE.GOV](http://www.oklegislature.gov)

- Identify your home and school Representatives and Senators (may be different)
- More details and instructions about your Legislator visits will be sent to you as the event nears
- If appointments are set up on your behalf, you MUST be there
- Take your poster with you to the meeting(s) and share information about your research
- Remember: Use layman's terms & outline how your research affects and/or benefits his/her constituents!

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A BRIEF SUMMARY: HOW YOU ARE JUDGED

- ❖ Abstract
  - You may submit revised abstracts to me until 2/6/17
- ❖ Oral Presentation
  - Timed, 3-minutes, in front of a panel of judges
- ❖ Poster
  - Judges will visit you at your poster during the poster session and briefly review your poster
  - 3-minutes of timed Q&A will follow
  - We'll go over "best practices" for poster development and special guidelines for this event later in the presentation

Refer to the sample judging sheet in your packet for more judging details.

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JUDGING CRITERIA

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

- Abstract
  Format, clarity, societal impact, objective of study, results, conclusions, etc.
- Scientific presentation
  Clear 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

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ABOUT THE JUDGES & YOUR PRESENTATIONS

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

Oral Presentation: 3 minutes (timed)

- Walk in- SMILE, introduce yourself, be confident (this is your project, you are your own expert on the matter), and walk them through what you have done - using your poster as a guide or reference.
- No questions may be asked during this presentation.

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ADDITIONAL PRESENTATION 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 non-science friend and listen to their feedback on your presentation.
- Practice presenting in an empty room and practice talking at the volume you plan on speaking at and the hand gestures you will use (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. Make them feel that your scientific findings are important.

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ADDITIONAL PRESENTATION SUGGESTIONS

- Time yourself to make sure you can present in the 3 minutes timeframe.
- If you forget your next point do not panic. Calmly recollect yourself and keep moving.
- Smile and be warm to the judges. They are spending their time listening to you talk. Be gracious.
- Repetition is the key to presentation success.

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ABOUT THE JUDGES & YOUR PRESENTATIONS

Poster Session: up to 1 minute to review poster, 3 minutes Q&A

- One judge will be timing you, all others will be taking notes
- This is when the questions are asked—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…
- If you are asked a question that you do not know the answer to it is acceptable to say you don't know. Do NOT make up an answer.
- Judges may not go in numerical order during the poster session, so be prepared to present when the judges arrive at your booth.
- They may also take a break between the Research-Intensive and Regional Universities.

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ABSTRACT (REVISION DEADLINE FEBRUARY 6, 4 P.M.)

Judges will score your abstract as part of your cumulative judging 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 6th at 4 p.m.

- MS Word format, no PDFs accepted
- Use the provided template for your submission; standard one-paragraph format
- Avoid scientific jargon
- Must be the work of the student
- See the provided sample judging sheet for scoring criteria
- Be sure that you receive a confirmation of receipt from me

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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
- Do mock presentations prior to event with an audience
- 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

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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

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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

2017 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
  - Size limit for this event: 48”x36”
  - Set the page size (in your (program) to match the final print size
  - Ask your mentor for advice regarding

2017 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 NOT necessarily for a scientific crowd, it is for the general public and Legislators
- It is being judged by highly educated researchers from various fields

SO: Make sure your poster can be understood by the non-scientific community, but it must also succinctly express your scientific research and findings

- Include all essential information; keep writing concise
- Avoid jargon

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POSTER PREP: GENERAL POSTER FORMAT

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

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POSTER PREP: GENERAL POSTER FORMAT

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

Poster Title
Authors, Affiliations

Abstract and/or Research Question

Background

Methods

Charts & Figures

Results

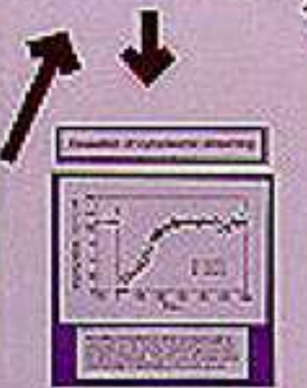
Discussion

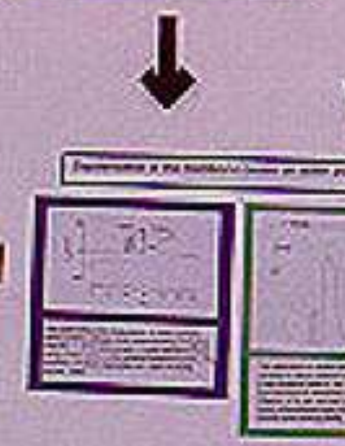
Societ mpact

Acknowledgements

Ca^{2+}-Dependent Cl^- Action Potentials in Chara: Different Points of View

Rebecca Elliott, Bernina Kanado, Sarah Otis, Kyong Kim





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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
- Should be legible from three feet away

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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

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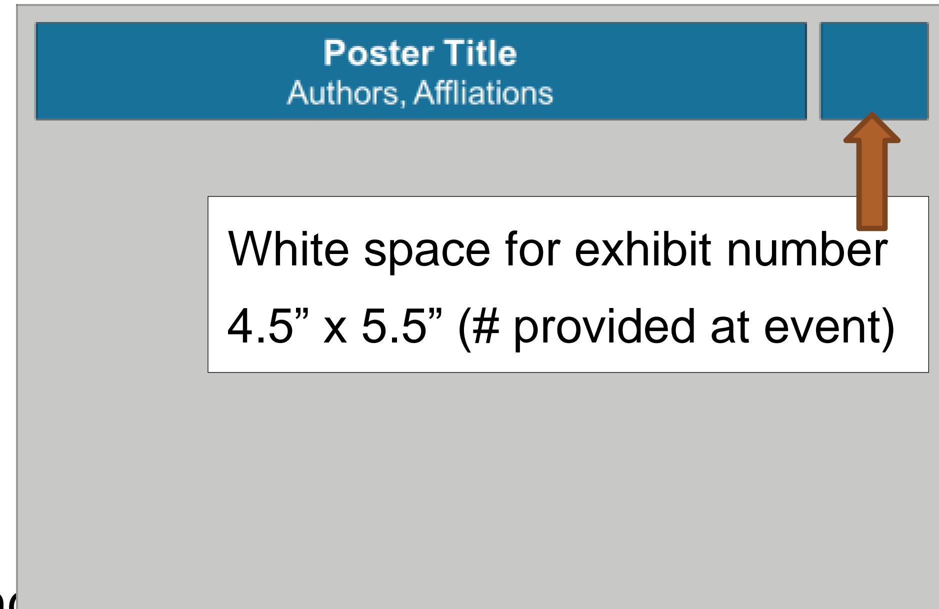
POSTER PREP: A QUICK REFERENCE GUIDE

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



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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

[illegible]

INTRODUCTION

[illegible]

MATERIALS AND METHODS

Colilert test - water

Coliform test - sediment

Antibiotic susceptibility test

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

				Ampicillin resistant		Ofloxacin resistant		Tetracycline resistant	
Date	Site†	Total coliforms	*E. coli*	Total coliforms	*E. coli*	Total coliforms	*E. coli*	Total coliforms	*E. coli*
Nov 07	T	‡	29.9 ± 5.1	2,370.0 ± 20.0	10.2 ± 4.3	4.2 ± 1.1	3.6 ± 0.5	1,878.7 ± 808.7	23.0 ± 2.7
	E§	3,986.7 ± 440.1	273.3 ± 126.7	1,099.8 ± 245.3	84.8 ± 16.0	10.0 ± 2.0	5.8 ± 0.4	341.0 ± 11.1	65.7 ± 12.9

[illegible]

Table 2. Aeromonads Isolated in November 2007

Location	Number	Identification*
Upstream sediment	45	Aeromonas spp. (25) Aeromonas hydrophila (20)
Downstream sediment	28	Aeromonas spp. (5) A. hydrophila (23)
WWTP effluent	1	A. hydrophila (1)

[illegible]

Table 3. Antibiotic Susceptibility of Aeromonads Isolated in November 2007

Location	Antibiotic	Number	Susceptible / Resistant*	Multidrug Resistance
Upstream sediment	Ofloxacin	45	(45 of 45) susceptible — 100% none resistant — 0%	none
	Tetracycline	45	(43 of 45) susceptible — 95.6% (2 of 45) resistant — 4.4%	
	Trimethoprim	45	(44 of 45) susceptible — 97.8% (1 of 45) resistant — 2.2%	
Downstream sediment	Ofloxacin	28	(24 of 28) susceptible — 85.7% (4 of 28) resistant — 14.3%	2 resistant to ofloxacin and trimethoprim 1 resistant to tetracycline and trimethoprim 1 resistant to tetracycline, trimethoprim and ofloxacin
	Tetracycline	28	(24 of 28) susceptible — 85.7% (4 of 28) resistant — 14.3%	
	Trimethoprim	28*	(21 of 27) susceptible — 77.8% (6 of 27) resistant — 22.2%	

[illegible]

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

[illegible]

DISCUSSION

[illegible]

ACKNOWLEDGEMENTS

[illegible]

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POSTER PREP: GENERAL POSTER FORMAT

- Use legends that explain your figures

Charts & Figures

Discussion

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

- Use illustrations to support/explain results

Results

A Novel Assay to Predict Cancer Resistance to Cisplatin

Lacy Brame[1], Vengatesh Ganapathy[1], Ilangovan Ramachandran[1], Lurdes Queimado[1-5]

Departments of [1]Otorhinolaryngology, [2]Cell Biology and [3]Pediatrics; [4]The Oklahoma Tobacco Research Center and [5]The 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 damage[1]. 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.

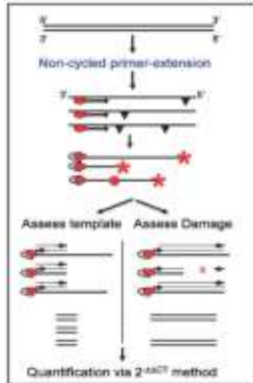

Figure 1. Diagram of PADDA. A single strand-specific non-cycled primer extension performed with a 5'-biotin-tagged primer and Vent exo- DNA polymerase identifies damaged nucleotides (inverted triangles), and generates a pool of highly specific biotin-tagged extended products, each of them derived from 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).

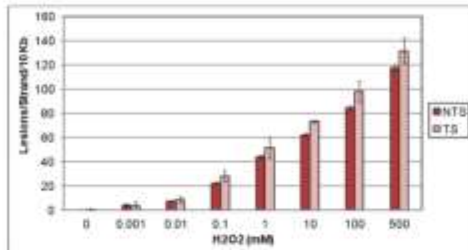

Figure 2. Quantification of induced DNA damage after *in vitro* exposure to a dose escalation of H_2O_2. Strand-specific DNA damage was quantified by q-PADDA. Lesion frequency was estimated via Poisson equation. NTS, non-transcribed strand; TS, transcribed strand; Data represents Mean ± S.E.M.

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

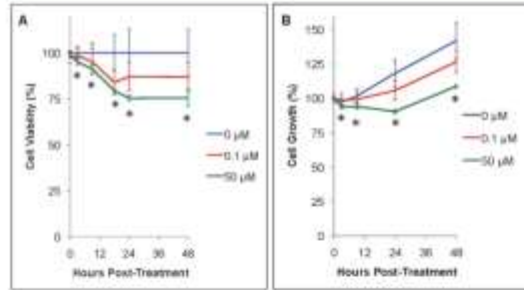

Figure 3. Cell viability assay and cell growth. SCC-1 cells were treated with 0 µM, 0.1 µM, 50 µM concentrations of cisplatin and allowed to repair damage for 0, 3, 9, 18, 24 & 48 hour time intervals. Cell viability (A) and cell growth (B) were determined by MTT assay. Data shown as Mean ± S.D. * $p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

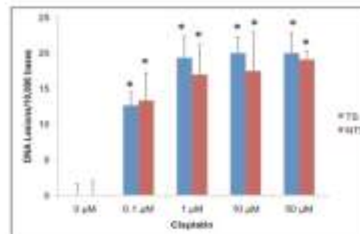

Figure 4. DNA damage measured by q-PADDA in SCC-1 cells exposed to cisplatin for 3 hours. Damage was quantified by q-PADDA in both transcribed (TS) and non-transcribed (NTS) strands. Data shown as Mean ± S.E.M. * $p<0.01$.

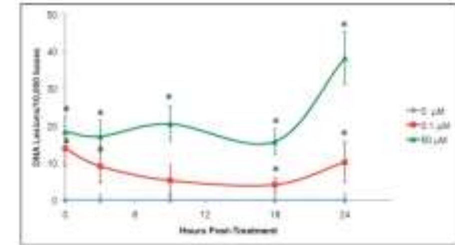

Figure 5. DNA damage measured by q-PADDA in SCC-1 cells after exposure to cisplatin. SCC-1 cells were treated with 0 µM, 0.1 µM, 50 µM concentrations of cisplatin and allowed to repair damage for 0, 3, 9, 18 & 24 hour time intervals. Data shown as Mean ± S.E.M. * $p<0.01$.

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.

- 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

[1]Reis AM, Mills WK, 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.

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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

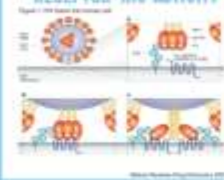
Courtney D. Garcia[1], B. N. Shockey[1], B. Gridley[2], S. J. Archibald[2], Dominique Schols[3], T. J. Hubin[1]

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.

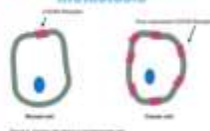
1. Societal Impact:

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.

Receptor-HIV Activity

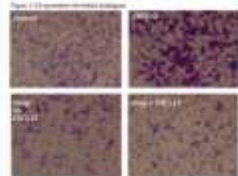


CXCR4 and Cancer Cell Metastasis

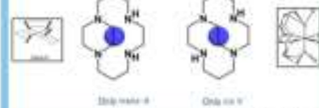


Anti-Cancer Activity: Invasion assays

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



Restrict to one configuration



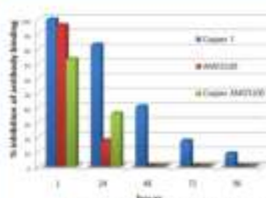


Figure 5. The inhibition of anti-CXCR4 antibody binding over time after exposure to 32nM of the drug. A population of 100,000 cells was isolated for each data point and analyzed by flow cell cytometry using a secondary fluorescein tagged IgG antibody (negative values are not shown).

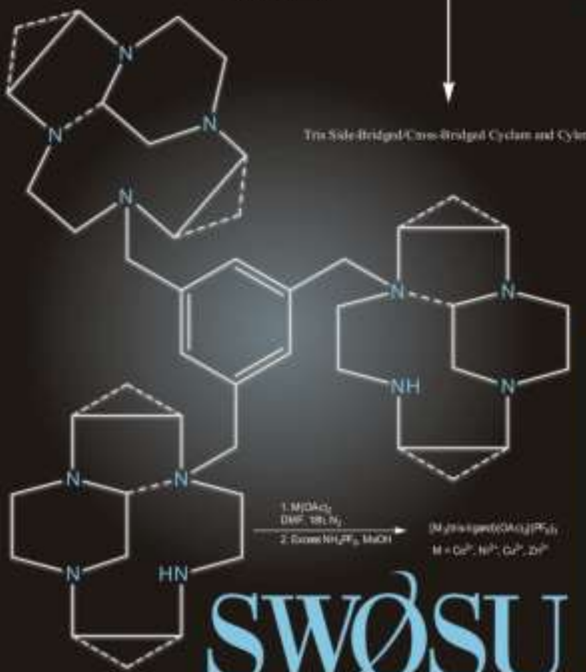
2. Objectives:

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

3. Methods:

Synthetic routes extending our bis-linked ligand syntheses to use the C 3-symmetric linker 1,3,5-tris(bromomethyl)benzene were developed. Copper(II), nickel(II), cobalt(II), and zinc(II) complexes were made using our previous methods. Electrospray 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.

Synthesis of C3-symmetric tris-linked analogues



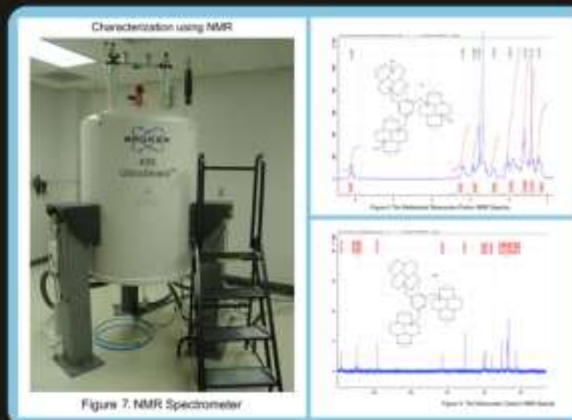


Figure 7. NMR Spectrometer

IC_{50} (μg/ml) values calculated from Ca-signaling experiments	U87.CD4.CXCR4	U87.CD4.CCR5
Zn_2-1	0.05	1.79
Ni_2-2	0.22	10.3
Zn_2-3	0.07	6.7
Cu_2-3	3.93	15.74
Cu_2-4	5.12	15.86
Cu_2-5	0.35	14.84
Zn_2-6	0.44	17.78
AMD3100	0.011	—
Maraviroc	—	0.00208
AMD3451	>1	>1

Figure 10. Binding Experiment CXCR4 & CCR5

4. Results:

The ligand syntheses of the side-bridged and cross-bridged C_3-symmetric ligands proceeded similarly to the previously developed bis-ligand routes. Complexation with the desired metal ions proceeded as expected. Characterization of the metal complexes resulted in publishable 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, just as in the bis-linked compounds. An unexpected benefit of tris 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 adapted 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.

Acknowlegements: Funding was provided by Research Corporation (CC6505); the Oklahoma State Regents for Higher Education; and NIH Grant P20 RR016478 from the INBRE Program of the National Resources.

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

THE UNIVERSITY of TULSA

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

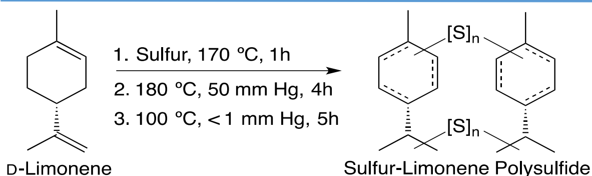


Figure 2. Reaction Scheme with proposed structure

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

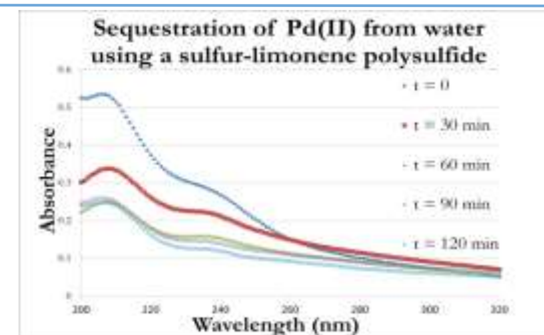


Figure 4. Palladium Catalyst Sequestration from Water
Using UV-Vis Spectroscopy, we monitored a time course of the sequestration of a valuable palladium metal catalyst.

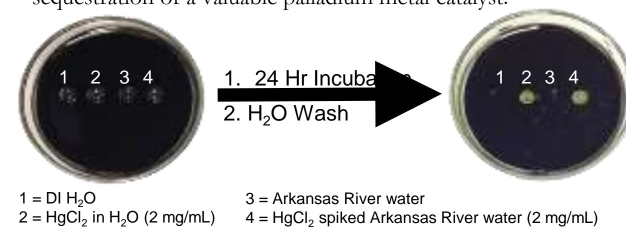


Figure 5. Mercury sensing by a chromogenic response

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

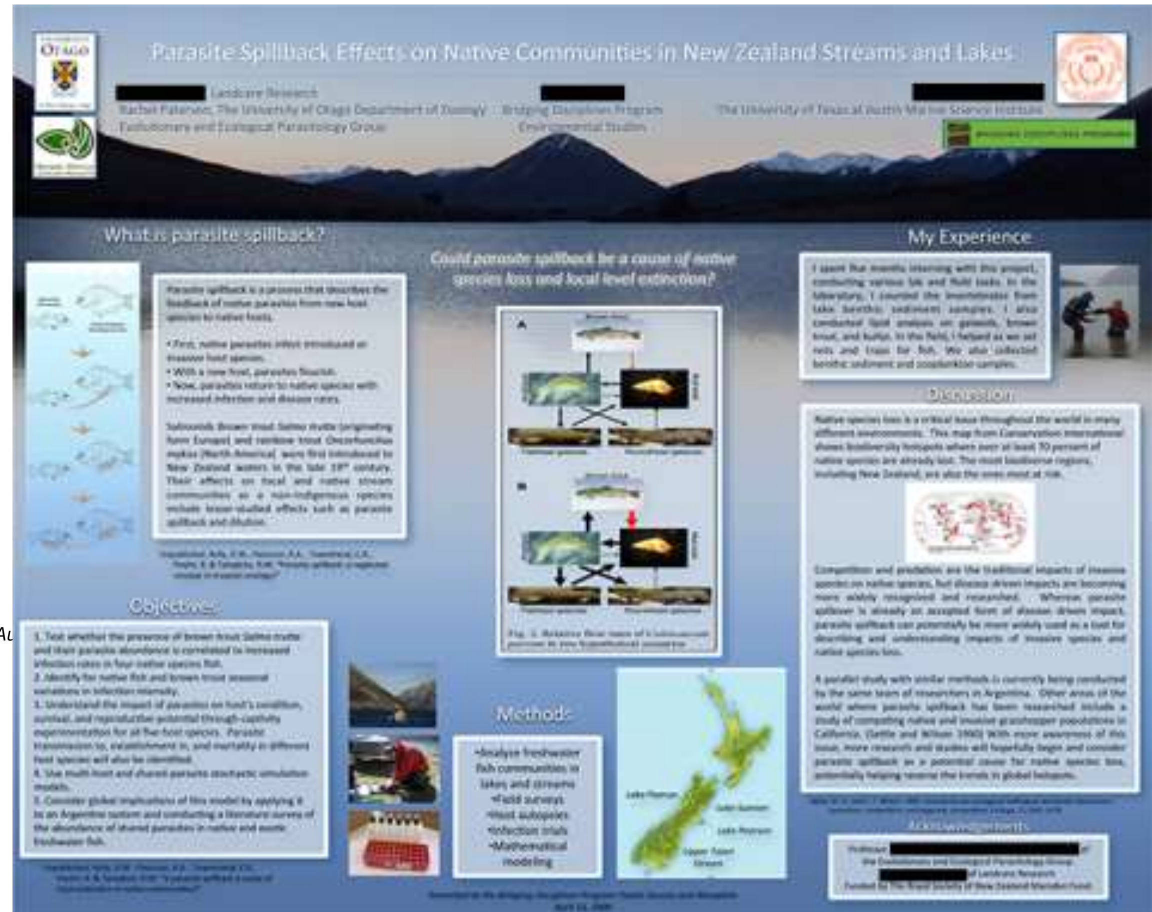
1. Chung, et al. *Nature Chemistry* **2013**, *5*, 518-524.
2. Polymers from Renewable Resources Gandini, A. *Macromolecules* **2008**
3. Crockett, M. P.; Evans, A. M.; Chalker, J. M. Unpublished
4. Sulfur-Limonene Polysulfide. Crockett, M. P.; Evans, A. M.; Chalker, J. M. Provisional patent filed Oct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

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

Credit: University of Texas at Au



Credit: University of Texas at Austin, https://ugs.utexas.edu/our/poster

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

ATLA: Alternatives to Laboratory Animals. 2007; 35(6): 641-659.

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

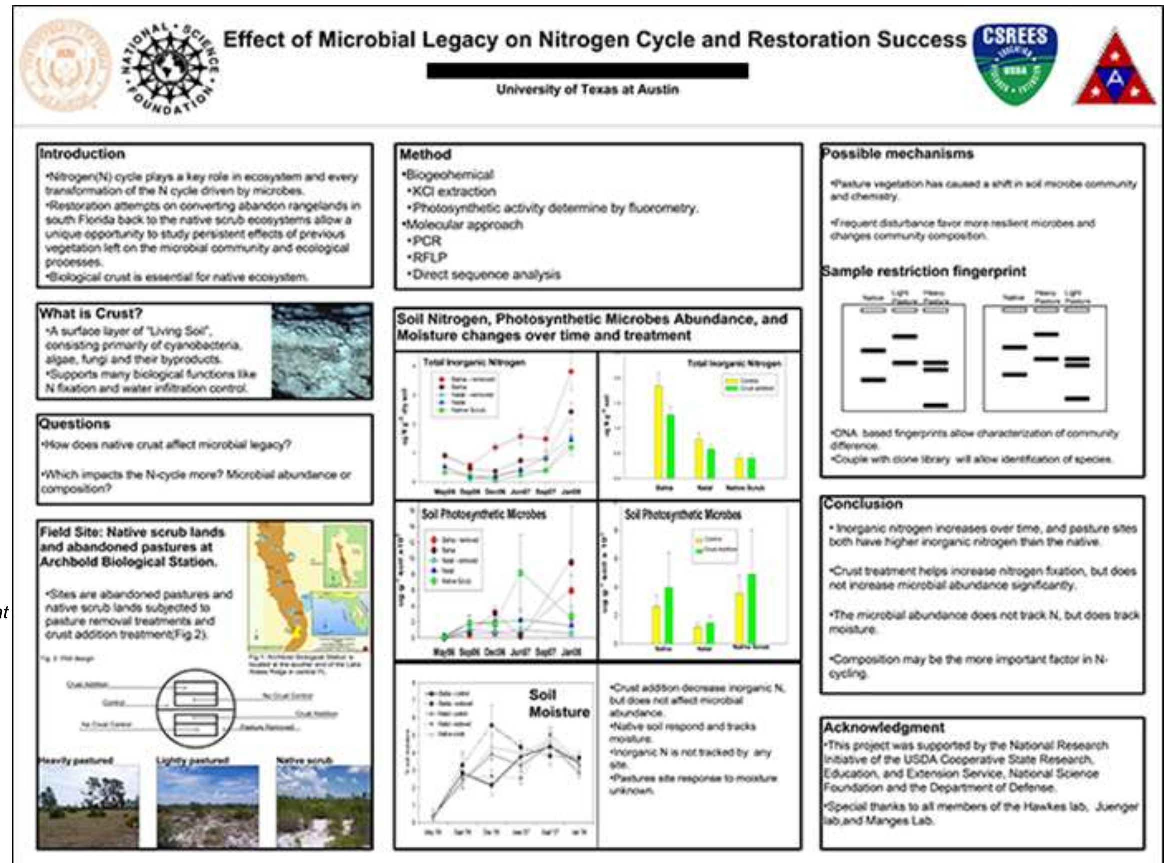
Text is impossible to read and potential observers would be too distracted by the image to sort through the information anyway.

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

Credit: University of Texas at



Credit: University of Texas at Austin, https://ugs.utexas.edu/our/poster

Poster Examples: “Do’s” and “Don’ts”

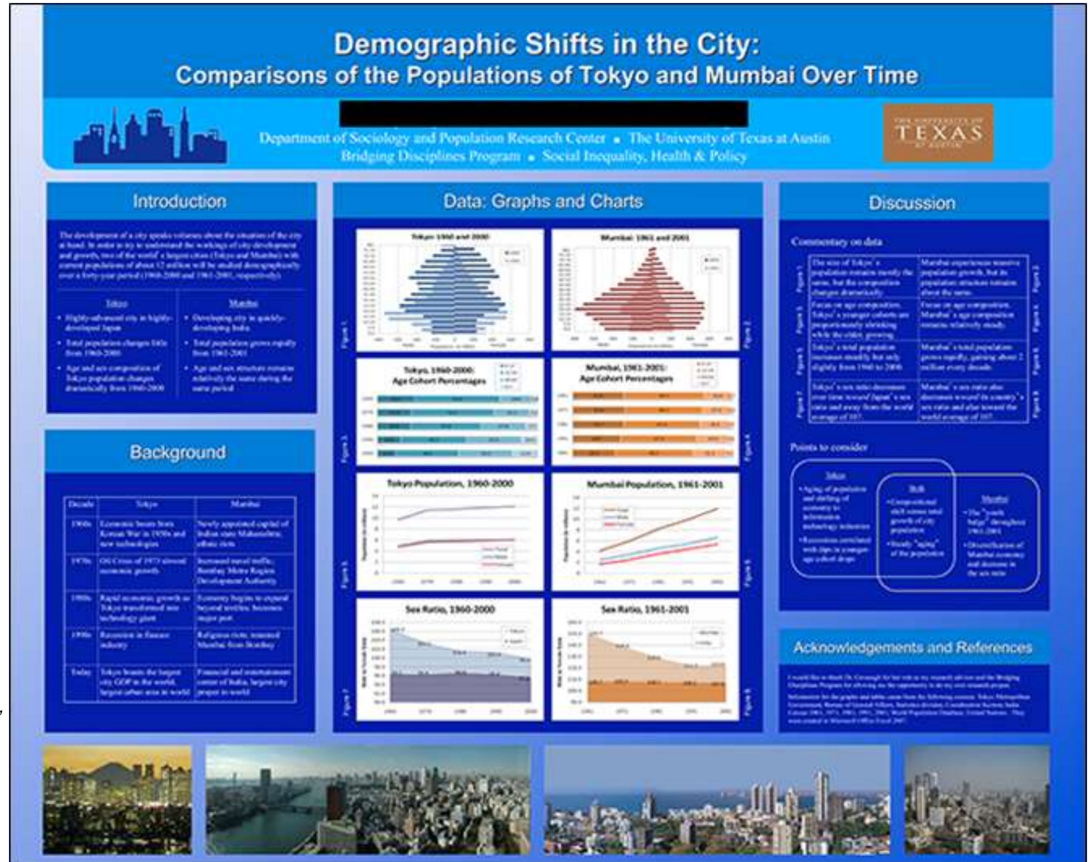
Strengths:

- 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

Credit: University of Texas at Austin,



Credit: University of Texas at Austin, https://ugs.utexas.edu/our/poster

Poster Examples: “Do’s” and “Don’ts”

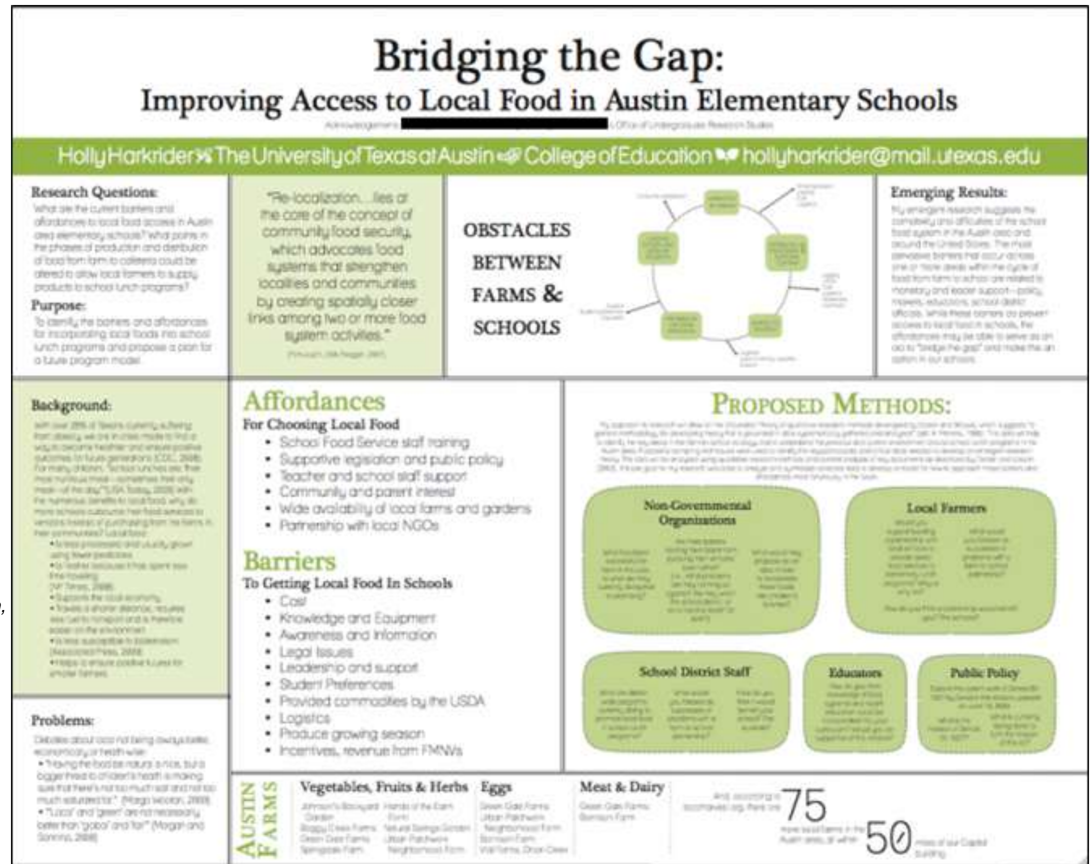
Strengths:

- 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:

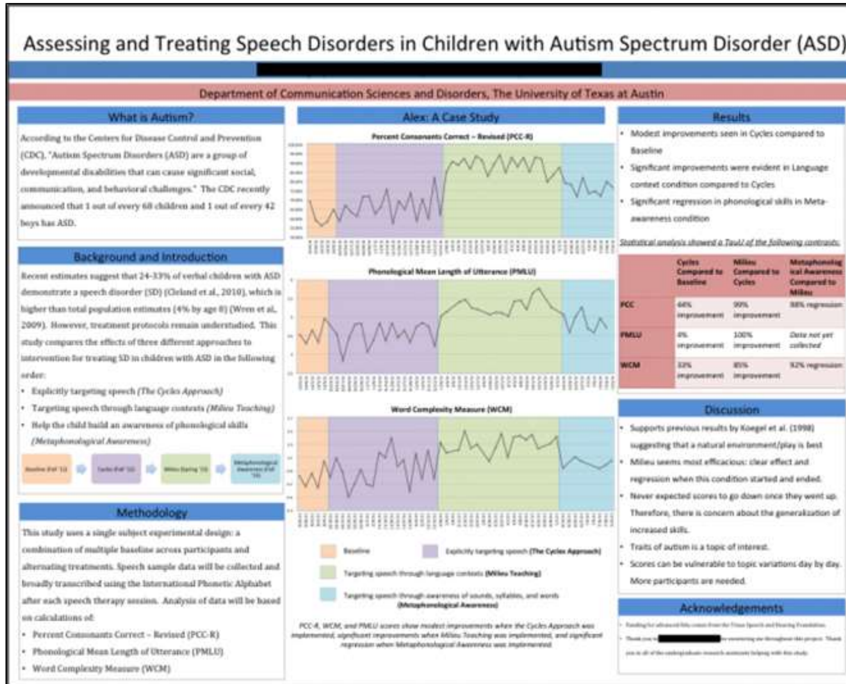
- Many sections without a clear flow between them
- Lacks acknowledgements

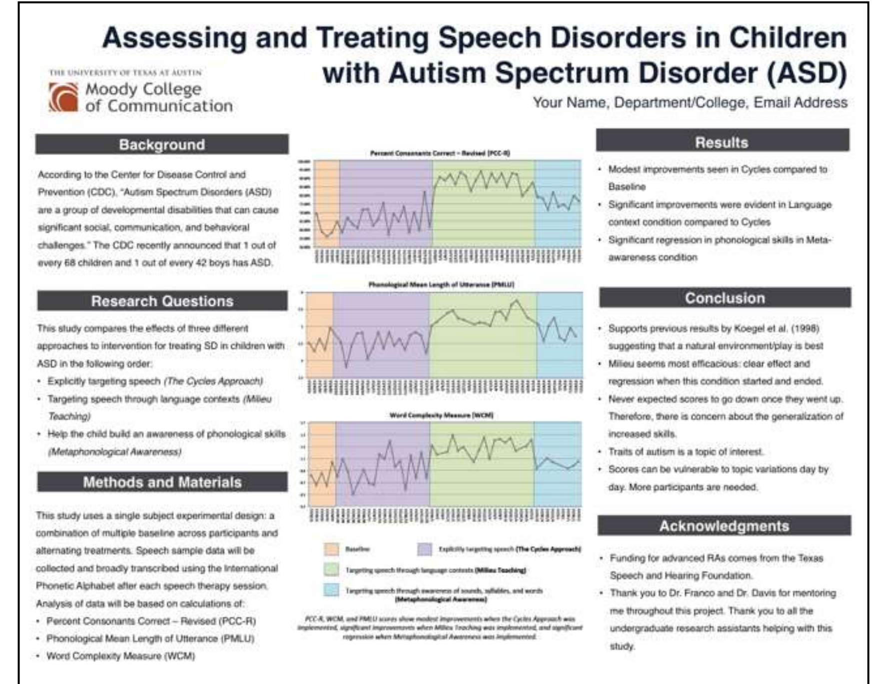
Credit: University of Texas at Austin,



Credit: University of Texas at Austin, https://ugs.utexas.edu/our/poster

Poster Examples: Before/After





Credit: University of Texas at Austin, https://ugs.utexas.edu/our/poster/templates

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

2017 Research Day at the Capitol

MONDAY, MARCH 27 * WATERFORD HOTEL, OKC

4:00 – 6:00 p.m.	Check-in for oral presentation judging—individually scheduled times Take your poster with you! (*Waterford Hotel, Current Room*)
4:00 – 6:00 p.m.	Set up your poster immediately following your oral presentation; return by 6:10 p.m. (*Waterford Hotel, Grand Ballroom*)
6:10 p.m.	Return to Grand Ballroom and prepare for poster session (*Waterford Hotel, Grand Ballroom*)
6:30 – 8:30 p.m.	Poster session & poster judging *Registered guests & students* (*Waterford Hotel, Grand Ballroom*)
8:30 p.m.	Adjourn for the night

2017 Research Day at the Capitol

TUESDAY, MARCH 28 * STATE CAPITOL OF OKLAHOMA

8:00 -10:45 a.m.	Student researchers meet their Legislators (Legislator offices) (Pre-scheduled meetings will have been made for you by the OSRHE office when possible.)
10:50 a.m.	Arrive in Blue Room, 2nd Floor
11:00 - noon	Awards ceremony (Blue Room, 2nd Floor)
Noon – 1:00 p .m.	Final meetings of student researchers with Legislators (Legislator offices)

2017 Research Day at the Capitol

AWARDS CEREMONY AND PRIZES

Winners will be announced at an awards ceremony to be held in the Governor's Blue Room at 11:00 a.m. on March 28.

From the posters presented, EPSCoR will award the following prizes:

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
**Final project report will be required*

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 and research-intensive)
2nd Place: $250 cash prize (1 ea: regional and research-intensive)
3rd Place: $250 cash prize (1 ea: regional and research-intensive)

NOTE: The student identified as the lead on the project must present their poster in person at Research Day to be eligible for prizes.

Research Day at the Capitol

DRESS PROFESSIONALLY; A NEW SUIT ISN’T NECESSARY

Research Day at the Capitol

APPROPRIATE DRESS

OKLAHOMA EPSCoR

2017 Research Day at the Capitol

YOUR STIPEND FUNDING

You can expect delivery of your $250 stipend check within approximately 3-4 weeks. Call our office it is hasn't arrived by Dec. 18 and we'll attempt to track it.

- Funds are to cover your travel to/from OKC and for fees incurred in developing/printing your poster.
- Checks will be mailed to your permanent address, which may/may not be your university address.
- Checks will be issued from "OKLAHOMA STATE UNIVERSITY," NOT "OK EPSCoR."
- OSU students' checks will be processed through the OSU Bursar's Office.

2017 Research Day at the Capitol

ONLINE REGISTRATION REQUIRED (BY FEBRUARY 6)

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/calendar/2017-research-day-capitol

- You should advise parents, friends, family, faculty advisors, etc. to register online (or you may register online for them)
- Registered attendees are invited to attend the poster session on March 27, 6:30-8:30 p.m. (hors d'oeuvres will be served) and the award ceremony on March 28. A University representative and/or family member(s) may also accompany the student as he/she visits their Legislators.
- Registration deadline: February 6

OKLAHOMA EPSCoR

2017 Research Day at the Capitol

FINAL THOUGHTS—THINGS TO REMEMBER

You were chosen for a reason!

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

OKLAHOMA EPSCoR

2017 Research Day at the Capitol

INSIGHT FROM A PAST WINNER

Mary Katherine Randolph, 1st Place

Regional & Community College Category

Oklahoma City Community College

Poster Topic: Cancer Research

PMA induces growth inhibition and morphological changes in HT-1080 cells

Mary Katherine Randolph[1] and Zhizhuang Joe Zhao[2]

[1]Department of Chemistry, Oklahoma City Community College,

[2]Department of Pathology, University of Oklahoma Health Sciences Center

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.[1] 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.[1] 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 Cells

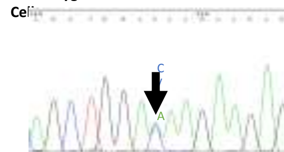


Figure 1. The entire coding region of the N-Ras gene was amplified from HT-1080 cell cDNA by PCR and sequenced. HT-1080 cells show heterozygosity for the N-Ras mutation with a single amino acid substitution at position 61, from a glutamine (Q) to a lysine (K).

Comparative Morphology

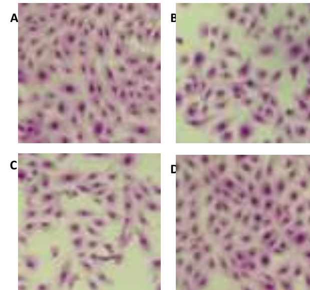


Figure 2. Morphology of investigated HT-1080 cells. (A) Control, (B) 10uM PMA, (C) 100uM PMA, and (D) 1000uM PMA. Cells in B-D are less dense, irregularly shaped and appear elongated. Wright-Giemsa stain, bright field, 100X.

Viable cell count

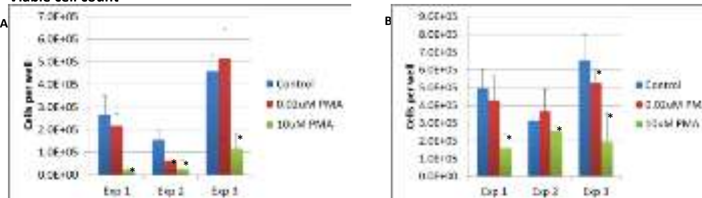


Figure 3. Experiments in set (A) were seeded with half the uL HT-1080 cells seeded in set (B). Four viable cell counts were taken per slide, error bars indicate standard deviation. *P<0.05 indicates PMA-treated cells are significantly different from the corresponding control cells.

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

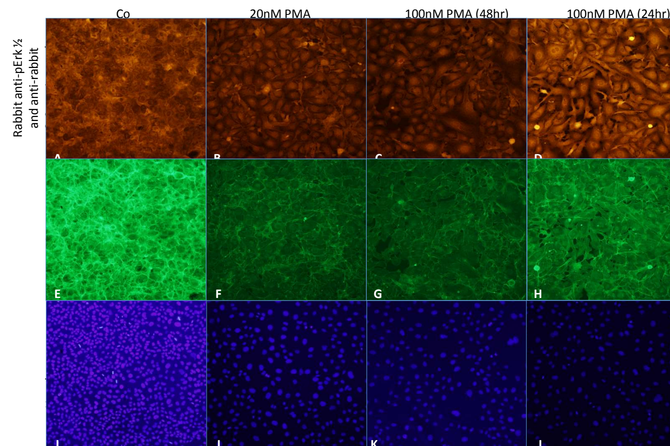


Figure 4. With the addition of PMA, A-D highlight increasing Erk 1/2 activation, E-H actin reorganization, and I-L increasing nucleus size and decreasing cell numbers. Brightly stained mitotic cells are visible in image I which are lacking in J-L after PMA treatment. Immunofluorescence microscopy, 200X.

Materials & Methods

Cell culture. Stock cultures of HT-1080 cells were maintained in DMEM supplemented with 10% FBS at 37°C, 5% CO_2, 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

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

2017 Research Day at the Capitol

INSIGHT FROM A JUDGE

Casey Harness

Oklahoma Center for the Advancement of Science & Technology (OCAST)

Background: BS Biomedical Engineering, Yale University, 2007

OKLAHOMA EPSCoR