RECOGNIZING OUTSTANDING UNDERGRADUATE RESEARCH

2015 Research Day at the Capitol

STUDENT INFORMATION/TRAINING SESSION

OKLAHOMA EPSCOR

Information Session Agenda-Welcome!

SATURDAY, NOVEMBER 15, 2014

12:00 p.m. **Lunch**

12:15 p.m. **Welcome and introductions**
All – Students synthesize their research into 30-second layperson summaries

12:45 p.m. **Research Day housekeeping details (Stipends, deadlines, etc.)**
Gina Miller, EPSCoR Outreach Coordinator

1:00 p.m. **Poster preparation and oral presentation information from a past winner**
Lacy Brame, 2014 Grand Prize Winner

1:30 p.m. **Day-of-the-event information and further insights**
Gina Miller, EPSCoR Outreach Coordinator

1:40 p.m. **Q&A**

2:00 p.m. **Adjourn**

2015 Research Day at the Capitol

HOTEL ACCOMMODATIONS (REQUEST DEADLINE FEB. 9)

EPSCoR will provide lodging on the evening of Monday, March 30 (night before Research Day), for student participants who live outside the OKC metro area and who have requested lodging prior to the February 9th deadline.

- *Conference hotel: Hilton Garden Inn OKC/Bricktown*
  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 fees
- Hotel will require 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 (but extra guests will be responsible for their own parking fees)
- Notify me no later than Feb. 9 if you wish for me to secure a room on your behalf—a sign up sheet is available today

2015 Research Day at the Capitol

YOUR STIPEND FUNDING

If your financial responsibility form has been submitted prior to the training session, you can expect delivery of your $250 stipend check within 2-3 weeks. Call our office if you haven't received your check by Dec. 19 and we'll attempt to track it.

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

2015 Research Day at the Capitol

ABSTRACT (REVISION DEADLINE FEBRUARY 9, 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 you originally submitted, you must submit your final, revised abstract in MS Word format prior to February 9th at 4 p.m.

- MS Word format, no PDFs accepted
- Use the provided template for your submission; standard one-paragraph format
- Avoid scientific jargon; describe the project in lay language
- See the provided sample judging sheet for other detailed scoring criteria

2015 Research Day at the Capitol

ONLINE REGISTRATION (DEADLINE MARCH 1)

All student researchers & anyone who will be attending Research Day at the Capitol in support of the student researcher must register online at: http://www.okepscor.org/calendar/2015-research-day-capitol

- You should advise parents, friends, family, faculty advisors, etc. to register online (or register online for them)
- Registered attendees will be issued conference programs and name tags that will allow them access to meals and snacks throught the day
- Registration deadline: March 1

2015 Research Day at the Capitol

TIMELINE OF IMPORTANT DATES

Saturday, November 15	Student information/training session *(State Capitol, 4th Floor Rotunda)*
Nov. 16-March 29	Students prepare scientific posters and display units
Monday, February 9	Deadline for students to submit: > revised abstracts (MS Word) > hotel requests
Sunday, March 1	Online registration closes: All students & guests should be registered by this date
Monday, March 30	Students with prescheduled overnight arrangements check into hotel after 3 p.m. (*Hilton Garden Inn OKC/Bricktown*) Dinner on your own
Tuesday, March 31	Research Day at the Capitol *(State Capitol, 4th Floor Rotunda)*

OKLAHOMA EPSCoR

2015 Research Day at the Capitol

MARCH 31, 2015 * EVENT AGENDA

7 a.m.	Students arrive
	(*Check in, set up posters and eat breakfast)*
7:45 a.m.	Posters set up and ready to present
7:45 a.m. – 12:45 p.m.	Posters on exhibit
	(*Students meet with legislators & Capitol guests)*
7:45 a.m.	Judging for poster competition begins
7:45 a.m. – 9:15 a.m.	Regional institution posters are judged
	(Times approximate!)
9:45 a.m. – 11 a.m.	Research-intensive institution posters are judged
	(Times approximate!)
11:15 – 1 p.m.	Lunch available, 4th Floor Rotunda
11:40 a.m.	Group photo on Grand Staircase
	(Students, legislators and faculty mentors)
12:45 p.m.	Awards ceremony, Governor's Blue Room
1:45 p.m.	Adjourn and take down posters

March 31* Day of the Event

WHAT'S PROVIDED & WHAT TO BRING WITH YOU

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

You are required to bring:

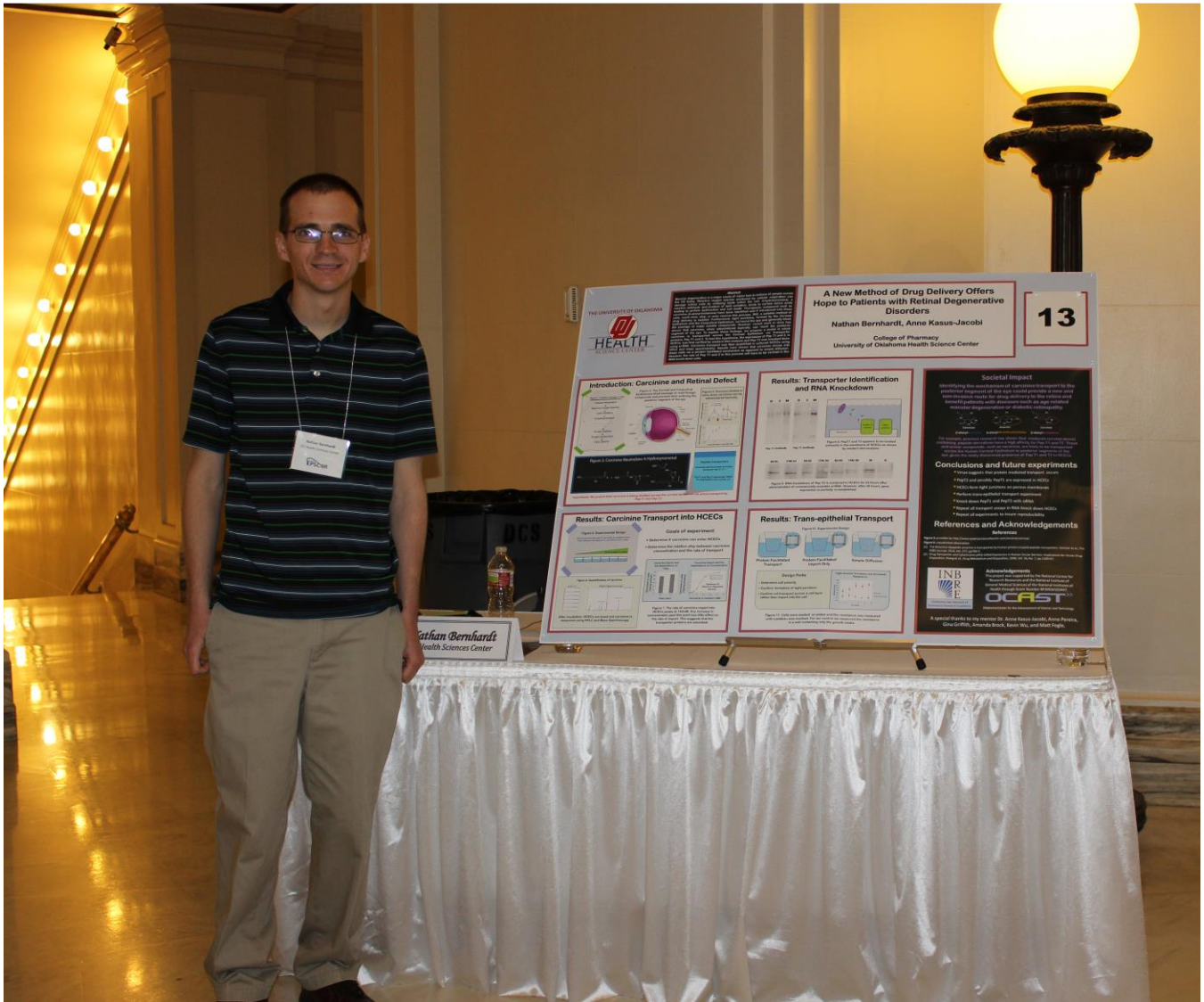
- Photo ID
  (May be requested by security at the Capitol entrance)
- Your poster & firm board backing
- Easel
- Tacks, Velcro or other attachment materials

You are highly encouraged to:

- Bring hands-on demonstration materials
- Identify your home and school legislators prior to coming to the event. Know them by sight and greet them by name, if possible. www.oklegislature.gov

March 31 * Day of the Event

DRESS PROFESSIONALLY; A NEW SUIT ISN'T NECESSARY

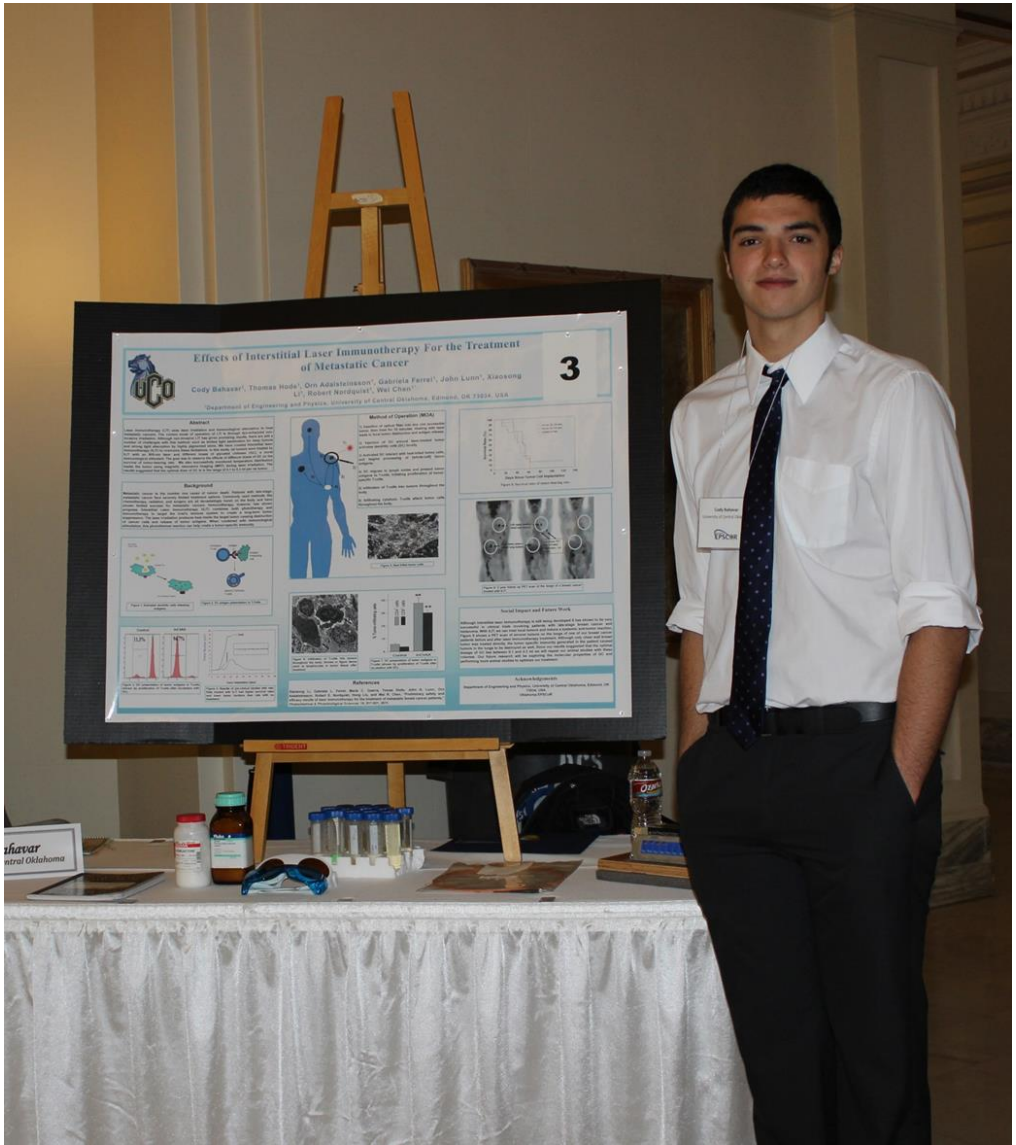


*March 31 * Day of the Event*

TABLETOP OR FREE-STANDING EASEL

March 31 * Day of the Event

BRING DEMONSTRATION MATERIALS



*March 31 * Day of the Event*

ARRIVAL INFORMATION

- Give your self plenty of time—it's hard to navigate around the Capitol streets & parking lots in the dark (and it's dark before 8 a.m.)
- Plan to pull into the parking lot 6:45-7:00 a.m.
- Park in the approved visitor parking area on the primary-entrance side of the building
- Enter via the covered portico entrance that you used today
- Go through security (no knives, etc.) & take elevator to 4th floor
- Check in at the EPSCoR booth & receive name badge & other materials
- Set up poster first, then grab breakfast
  (Name badge serves as your meal ticket throughout the day.)
- **Everyone must be set up and ready to go by 7:45 a.m.**
- Judging begins between 7:45-8:00 a.m.—**be ready to go at 7:45!!**
- Breakfast will be available in the rotunda 7:00-9:00 a.m. Snacks & drinks will be available at 9 a.m. Lunch will be available 11:15-12:30

March 31 * Day of the Event

SHARE YOUR WORK: RDC JUDGES, LEGISLATORS & CAPITOL VISITORS

March 31 * Day of the Event

SHARE YOUR WORK: RDC JUDGES, LEGISLATORS & CAPITOL VISITORS

March 31 * Day of the Event

KNOW YOUR LEGISLATORS! WWW.OKLEGISLATURE.GOV

*March 31 * Day of the Event*

GROUP PHOTO: STUDENTS, MENTORS & LEGISLATORS

March 31 * Day of the Event

JUDGING

March 31 * Day of the Event

JUDGING TIMEFRAME

Judges will visit each student as a group. (There will be ~4 judges.) Each student will be allowed 3 minutes for their oral presentation, followed by 2 minutes of questions from the judges. A stopwatch will be used and judges will take notes.

- Judging begins 7:45-8:00 a.m.—be ready to go at 7:45!
- Regional universities will be judged first: ~7:45 a.m.–9:15 a.m.
- Research-intensive universities will be judged after judges break to confer over regional presentations: ~9:45 a.m.-11 a.m.

Important: Judges may/may not go in numerical order and times noted above are approximate, so be prepared to present when the judges arrive at your booth.

March 31 * Day of the Event

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 of general audiences
- Societal impact statement
- Overall

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.

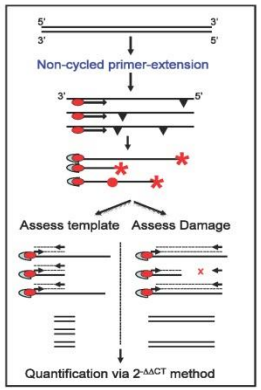


Figure 1. Diagram of PADDA. A single strand-specific non-cycled primer extension performed with a 5'-biotin-tagged primer and Vent exo- DNA polymerase identifies damaged nucleotides (inverted triangles), and generates a pool of highly specific biotin-tagged extended products, each of them derived from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

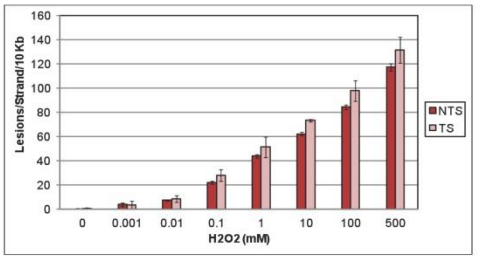


Figure 2. Quantification of induced DNA damage after *in vitro* exposure to a dose escalation of H_2O_2. Strand-specific DNA damage was quantified by q-PADDA. Lesion frequency was estimated via Poisson equation. NTS, non-transcribed strand; TS, transcribed strand; Data represents Mean ± S.E.M

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

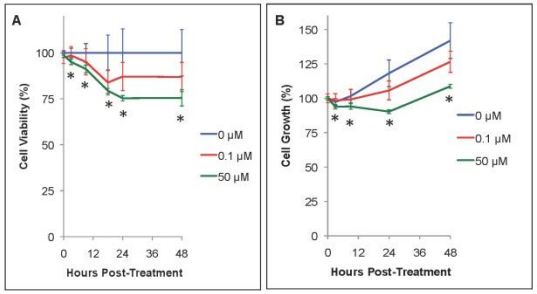


Figure 3. Cell viability assay and cell growth. SCC-1 cells were treated with 0 μM, 0.1 μM, 50 μM concentrations of cisplatin and allowed to repair damage for 0, 3, 9, 18, 24 & 48 hour time intervals. Cell viability (A) and cell growth (B) were determined by MTT assay. Data shown as Mean ± S.D. * $p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

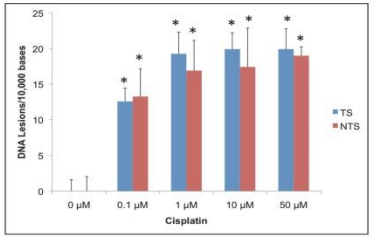


Figure 4. DNA damage measured by q-PADDA in SCC-1 cells exposed to cisplatin for 3 hours. Damage was quantified by q-PADDA in both transcribed (TS) and non-transcribed (NTS) strands. Data shown as Mean ± S.E.M. * $p<0.01$.

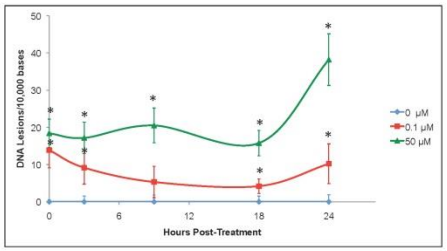


Figure 5. DNA damage measured by q-PADDA in SCC-1 cells after exposure to cisplatin. SCC-1 cells were treated with 0 μM, 0.1 μM, 50 μM concentrations of cisplatin and allowed to repair damage for 0, 3, 9, 18 & 24 hour time intervals. Data shown as Mean ± S.E.M. * $p<0.01$.

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.

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[2]

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:

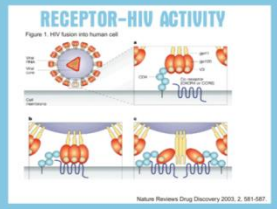
CXCR4 chemokine receptors are found on the surface of immune, and other, cells, and together with the specific natural ligand, CXCL12,have been revealed to play a role in a number of disease states. CXCR4 expression has also been reported in at least 23 different cancers. Target organs for breast metastases 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.

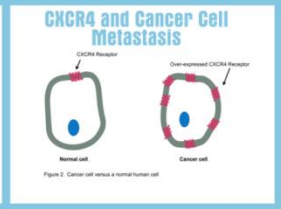
2. Objectives:

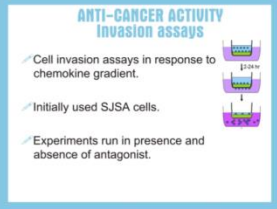
Our objectives were to synthesize C3-symmetric tris-linked analogues of our most effective bis-tetraazamacrocycle metal complexes and to characterize their chemical and physical properties in preparation for determining if the added macrocycle enhances their antagonism of CXCR4.

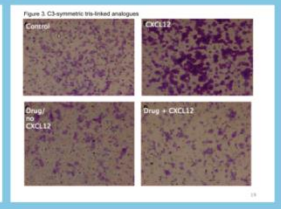
3.Methods:

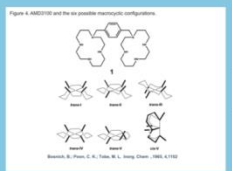
Synthetic routes extending our bis-linked ligand syntheses to use the C 3-symmetric linker 1,3,5-tris(bromomethyl)benzene were developed. Copper(II), nickel(II), cobalt(II), and zinc(II) complexes were made using our previous methods. Electrospray mass spectra, UV-Visible spectra, cyclic voltammograms,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











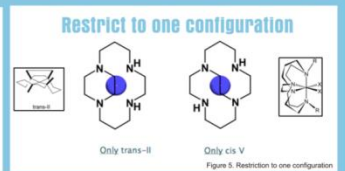


Figure 5. Restriction to one configuration

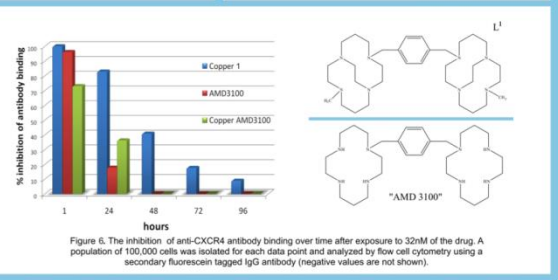


Figure 6. The inhibition of anti-CXCR4 antibody binding over time after exposure to 32nM of the drug. A population of 100,000 cells was isolated for each data point and analyzed by flow cell cytometry using a secondary fluorescein tagged IgG antibody (negative values are not shown).

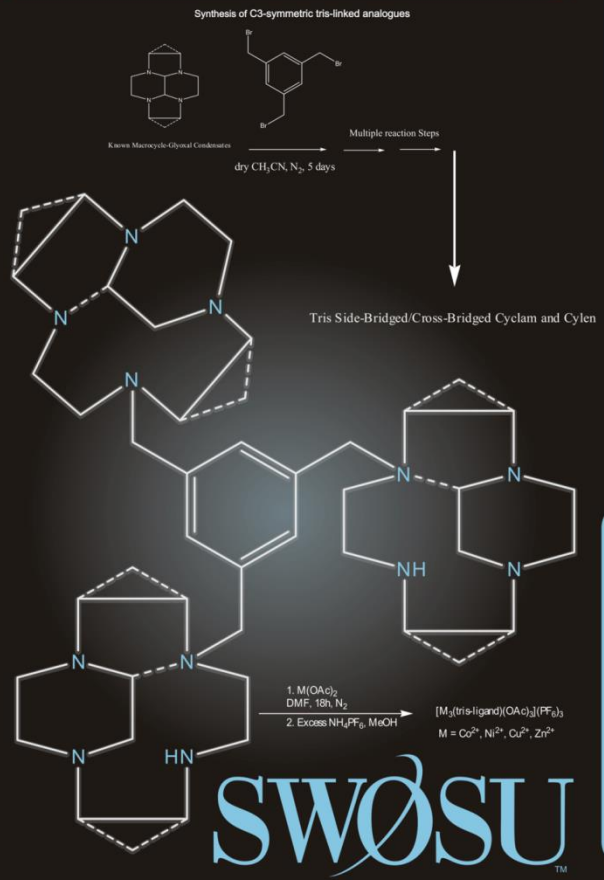


Figure 7. NMR Spectrometer

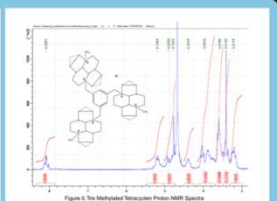


Figure 8. Tris Methylated Tetracyclen Proton NMR Spectra

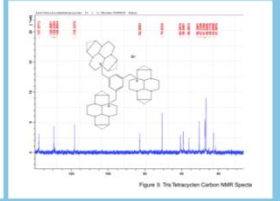


Figure 9. Tris Tetracyclen Carbon NMR Spectra

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
Co_2-**3**	3.93	15.74
Co_2-**4**	5.12	15.89
Cu_2-**5**	0.35	14.84
Zn_2-**5**	0.44	17.78
AMD3100	0.011	---
maraviroc	---	0.00209
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.

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 in freshwater ecosystems. Some species of aeromonads are opportunistic human pathogens while others have been linked to gastroenteritis in humans. Our objective in 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 pressing public health problems. In November 2007, Tahlequah Creek water was analyzed for the presence of antibiotics, and bacteria were isolated from creek sediments. Samples were taken upstream and downstream of the Tahlequah 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: azithromycin, ciprofloxacin, ofloxacin, and trimethoprim. Bacterial 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 1990. 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 the 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 drugs in the United States. However, antibiotics are often overprescribed or taken inappropriately. Bacteria exposed to antibiotics are constantly evolving. Increased levels of antibiotics 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

Colilert test - water

Colilert test - sediment

Antibiotic susceptibility test

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

Date	Site[2]	Total coliforms	*E. coli*	Ampicillin resistant: Total coliforms	Ampicillin resistant: *E. coli*	Ofloxacin resistant: Total coliforms	Ofloxacin resistant: *E. coli*	Tetracycline resistant: Total coliforms	Tetracycline resistant: *E. coli*
Nov 07	T	-[3]	29.9 ± 3.1	2,550.0 ± 20.0	10.2 ± 4.5	4.2 ± 1.1	3.6 ± 0.5	1,676.7 ± 404.7	23.6 ± 2.7
	E[4]	3,980.7 ± 445.1	273.3 ± 126.7	1,689.8 ± 245.3	84.8 ± 16.0	30.6 ± 2.0	5.8 ± 0.4	341.6 ± 31.1	65.7 ± 12.9

[1]MPNs were determined in water samples using the Colisure® quantitray system (IDEXX Laboratories). Values are MPN per 100 ml water ± SEM.
[2]T is water from Tahlequah Creek sampled approximately 0.5 miles upstream of the WWTP. E is the effluent from the Tahlequah WWTP.
[3]No data available.
[4]Tahlequah WWTP was undergoing repairs on the date the effluent was sampled.

Table 2. Aeromonads Isolated in November 2007

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

[1]Identification is based on 16S rDNA sequences. Numbers in parentheses indicate number of isolates.

Table 3. Antibiotic Susceptibility of Aeromonads Isolated in November 2007

Location	Antibiotic	Number	Susceptible / Resistant[1]	Multidrug Resistance
Upstream sediment	Ofloxacin	45	(45 of 45) susceptible — 100% none resistant — 0%	none
	Tetracycline	45	(43 of 45) susceptible — 95.6% (02 of 45) resistant — 4.4%	
	Trimethoprim	45	(44 of 45) susceptible — 97.8% (01 of 45) resistant — 2.2%	
Downstream sediment	Ofloxacin	28	(24 of 28) susceptible — 85.7% (04 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% (04 of 28) resistant — 14.3%	
	Trimethoprim	28[2]	(21 of 27) susceptible — 77.8% (06 of 27) resistant — 22.2%	

[1]Test strips were used to determine susceptibility to antibiotics based on Clinical Laboratory Standards Institute guidelines.
[2]One isolate was not tested.

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 Tahlequah Creek water samples collected downstream of the WWTP: azithromycin (0.062 µg/L), ciprofloxacin (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 Tahlequah Creek water and in WWTP effluent (Table 1). Many bacteria collected from Tahlequah 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, 6 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, OSRS award HR07-124, and by NIH NCRR grant P20RR016478-08.

Effects of Interstitial Laser Immunotherapy For the Treatment of Metastatic Cancer

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[1]Department of Engineering and Physics, University of Central Oklahoma, Edmond, OK 73034, USA

Abstract!

Laser immunotherapy (LIT) uses laser irradiation and immunological stimulation to treat metastatic cancers. The current mode of operation of LIT is through dye-enhanced non-invasive irradiation. Although non-invasive LIT has given promising results, there are still a number of challenges with this method, such as limited light penetration for deep tumors and strong light absorption by highly pigmented skins. We have created Interstitial laser immunotherapy (ILIT) to overcome these limitations. In this study, rat tumors were treated by ILIT with an 805-nm laser and different doses of glycated chitosan (GC), a novel immunological stimulant. The goal was to observe the effects of different doses of GC on the survival of tumor-bearing rats. We also successfully monitored temperature distribution inside the tumor using magnetic resonance imaging (MRT) during laser irradiation. The results suggested that the optimal dose of GC is in the range of 0.1 to 0.3 ml per rat tumor.!

Background!

Metastatic cancer is the number one cause of cancer death. Patients with late-stage, metastatic cancer face severely limited treatment options. Commonly used methods like chemotherapy, radiation, and surgery are all devastatingly harsh on the body and have shown limited success for metastatic cancers. Immunotherapy, however, has shown progress. Interstitial Laser immunotherapy (ILIT) combines both photothermal and immunotherapy to target the host's immune system to create a long-term tumor suppression. The laser irradiation produces heat inside the target tumor causing destruction of cancer cells and release of tumor antigens. When combined with immunological stimulation, this photothermal reaction can help create a tumor-specific immunity. !

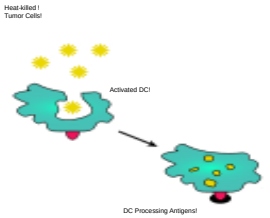


Figure 1. Activated dendritic cells releasing antigens.!

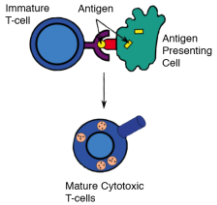


Figure 2. DC antigen presentation to T-Cells.!

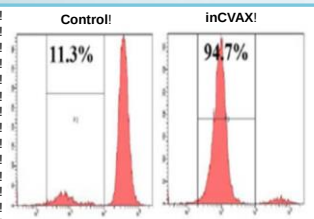


Figure 3. DC presentation of tumor antigens to T-cells (shown by proliferation of T-cells after incubation with DC).!

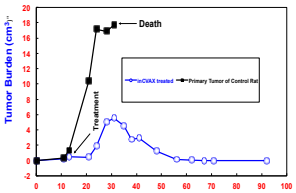
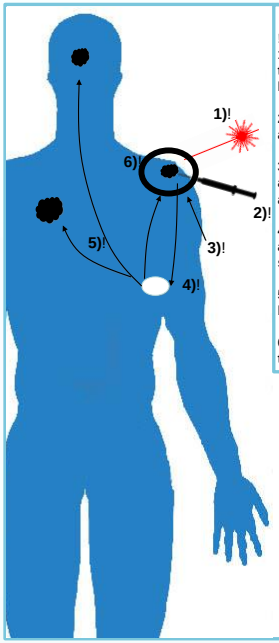


Figure 4. Results of pre-clinical studies with rats. Rats treated with ILIT had higher survival rates and lower tumor burdens than rats with no treatment.!



Method of Operation (MOA)!

1) Insertion of optical fiber into any one accessible tumor, then treat for 10 minutes. Heating with laser leads to local tumor destruction and antigen release. !

2) Injection of GC around laser-treated tumor activates dendritic cells (DC) locally.!

3) Activated DC interact with heat-killed tumor cells, and begins processing of [whole-cell] tumor antigens.!

4) DC migrate to lymph nodes and present tumor antigens to T-cells, initiating proliferation of tumor-specific T-cells. !

5) Infiltration of T-cells into tumors throughout the body. !

6) Infiltrating cytotoxic T-cells attack tumor cells throughout the body.

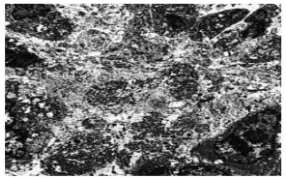


Figure 5. Heat killed tumor cells.!

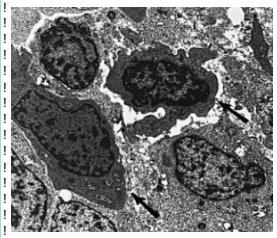


Figure 6. Infiltration of T-cells into tumors throughout the body. Arrows in figure below point at lymphocytes in tumor tissue after treatment.!

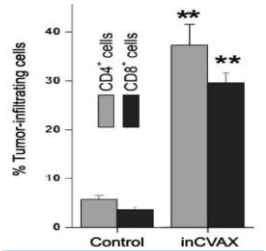


Figure 7. DC presentation of tumor antigens to T-cells (shown by proliferation of T-cells after incubation with DC).!

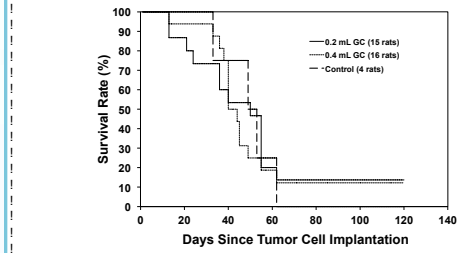


Figure 8. Survival rates of tumor-bearing rats.!

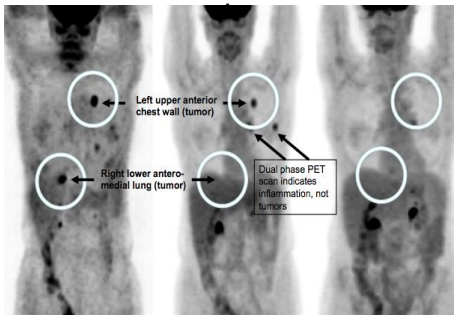


Figure 9. 2 year follow up PET scan of the lungs of a breast cancer treated with ILIT. !

Social Impact and Future Work

Although interstitial laser immunotherapy is still being developed it has shown to be very successful in clinical trials involving patients with late-stage breast cancer and melanoma. With ILIT, we can treat local tumors and induce a systemic anti-tumor reaction. Figure 9 shows a PET scan of several tumors on the lungs of one of our breast cancer patients before and after laser immunotherapy treatment. Although only chest wall breast tumor was treated directly, the tumor specific immunity generated in the patient caused tumors in the lungs to be destroyed as well. Since our results suggested that the optimal dosage of GC lies between 0.1 and 0.3 ml we will repeat our animal studies with these volumes. Our future research will be exploring the molecular properties of GC and performing more animal studies to optimize our treatment. !

References!

Xiaosong Li, Gabriela L. Ferrel, Maria C. Guerra, Tomas Hode, John A. Lunn, Orn Adalsteinsson, Robert E. Nordquist, Hong Liu, and Wei R. Chen, "Preliminary safety and efficacy results of laser immunotherapy for the treatment of metastatic breast cancer patients," *Photochemical & Photobiological Sciences*. 10, 817-821, 2011. !

Acknowledgements

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Oklahoma EPSCoR!

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.

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

Rebecca Elliot, Benina Abdallah, Sarah Ortiz, Kyong Kim

March 31 * Day of the Event

SUGGESTIONS FROM THE JUDGES

- Review sample judging sheet criteria
- Review your abstract and make sure it's accurate
- Do mock presentations prior to event with an audience
- Talk loud and project your voice (room is noisy)
- Pay close attention to societal impact and research objective
- Answer, "What have you accomplished with your research?"
- Statistics are good—provide proof of outcomes
- 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……"

2015 Research Day at the Capitol

AWARD CEREMONY AND PRIZES

Winners will be announced at an awards ceremony to be held in the Governor's Blue Room at 12:45 p.m. on March 31.

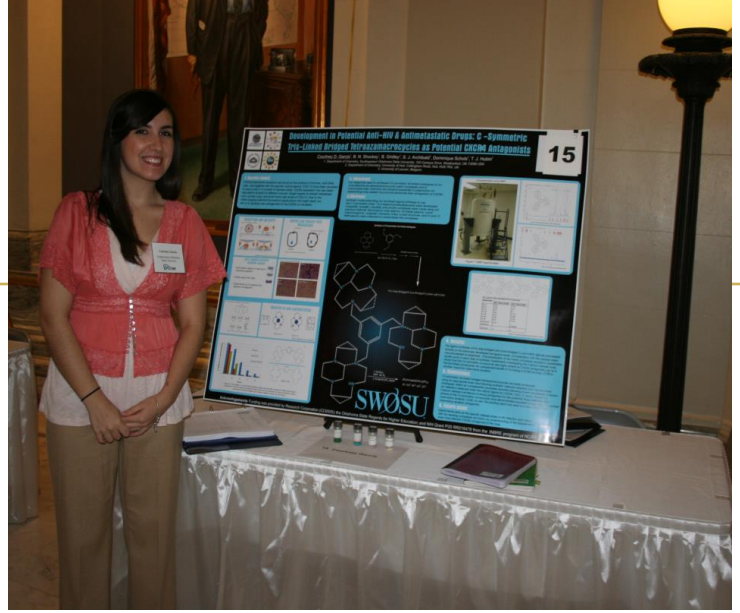
From the posters presented, EPSCoR will award the following prizes:

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)

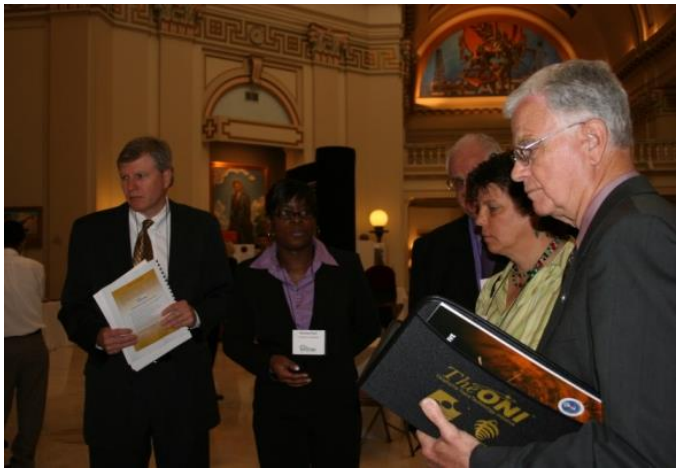
1st Place: $500 cash prize (1 ea: regional and research-intensive)
2nd Place: $250 cash prize (1 ea: regional and research-intensive)
3rd Place: $250 cash prize (1 ea: regional and research-intensive)

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


15
SWOSU


3


Bloodmeal Identification of Mosquitoes in Oklahoma
21
Megan Prouty, Valerie O'Brien, Tyler Ward, and Michael Reiskind
Department of Entomology and Plant Pathology, Oklahoma State University
21 Megan Prouty
Oklahoma State University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