2014

CSET Research Symposium

February 27-28, 2014

At Jackson State University

Call for Abstracts

Deadline: Friday, February14th

Abstract Requirements

- The Abstract Submittal Form is shown with a sample on the next page. Abstracts not conforming to the formatting of the sample will not be considered. Please use professional writing protocol (avoid use of all upper or lower case text).
- Abstracts MUST denote if the presentation is to be oral or poster and must be received no later than Friday, February 14, 2014, to be included in the printed program.
- Please proof your abstract carefully. **It will be printed in the conference program as submitted**.

Symposium Presentations

- All presentations must follow the required format and deadline for abstract submission.
- All research presentations should address a valid scientific question.
- Oral Presenters must use Microsoft PowerPoint.
- Presentations will be judged and awards will be presented at the conclusion of the event.

Presentation Requirements

- Poster Presentations must be free-standing and fit on a 30” x 40” board, which will be provided.
- Tape, tacks and other poster board mounting materials must be provided by the presenter.
- If you bring items for demonstration, you are responsible for their security.

The goals of this conference are to showcase faculty-mentored research of undergraduate and graduate students and to foster the leadership and professional development of students, staff and professionals to positively impact the workforce and academic pipeline.

We invite the participation of undergraduate and graduate students from across the nation in this Symposium.

LSAMP

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Abstract Information

Name: _____

Research Code: _____

I would like to make a: _____ Oral Presentation _____ Poster Presentation
(choose one)

Sample Abstract

Di- (2-ethylhexyl) Phthalate (DEHP) Induces Invasion, Migration, and Anchorage Independent Growth through Upregulation of S100P Signaling Pathways in Glioblastoma

First Name Last Name [1,2,3], Kenneth Ndebele[1,2,3], Barbara Graham[1,2,3], and Paul Tchounwou[3].[1]Laboratory of Cancer Immunology Target Identification and Validation, [2]Department of Biology, [3]College of Science, Engineering and Technology, Jackson State University, Jackson, Mississippi, USA.

Glioblastoma multiforme (GBM) is the most aggressive brain cancer with a median survival of approximately 1 to 2 years. The standard treatment of GBM has been surgical resection of the tumor, followed by radiation and chemotherapy. Even with the standard treatment, the mean survival of GBM is only extended from 2 months to about 1 year. This poor prognosis has led to the focus on identifying novel molecular targets against glioblastoma and the signaling mechanism through which they act. S100P is a transmembrane protein that plays a role in many different cancers, and its expression is associated with drug resistance, metastasis, and poor clinical outcome. The functional role of S100P in glioblastoma has not been fully investigated. Therefore, we examine whether the functional role of the environmental contaminant, DEHP, is mediated through S100P expression in glioblastoma. We hypothesize that silencing of S100P by lentiviral abrogates DEHP mediated anchorage independent growth, migration, and invasion in glioblastoma cancer cells. In this study, we have shown that silencing S100P gene expression in glioblastoma cancer cell lines significantly inhibited the proliferation compared to shGFP and control cells under normal *in vitro* conditions. Growth inhibition was further increased by treating cells with DEHP. One of the hallmarks of oncogenic transformation is the loss of anchorage independent growth as demonstrated by the ability to form colonies on soft agar. To evaluate the role of the S100P mechanisms of action in the maintenance of the transforming phenotype of glioblastoma cells, an agar assay was utilized. Glioblastoma cells infected with lenti-shS100P showed significant reduction in the colony formation. No apparent difference was found between the control cells treated with DEHP and the lenti-shS100P cells treated with DEHP. Our results show a possibility for S100P to serve a rate-limiting role in other cancer causing malignancies as well as provide target-dependent transcriptional and proteomic signatures that may be useful as drug response biomakers.

Research Code by Discipline

Discipline	Code
Developmental Biology	A-I
Environmental Biology	A-II
Genetics	A-III
Microbiology	A-IV
Botany	A-V
Cell Biology	B-I
Organic Chemistry	B-II
Molecular Biology	B-III
Biochemistry	B-IV
Pharmacology	B-V
Inorganic Chemistry	C-I
Photo-Chemistry	C-II
Physics & Mathematics	C-III
Physiology	C-IV
Immunology	C-V
Toxicology	D-I
Computer Science	E-I
Computer Engineering	E-II
Civil Engineering	E-III
Electrical Engineering	E-IV
Industrial Technology	E-V
Other (Designate)	Other