
# LBRN Work-in-Progress

## INBRE Seminar series

## Available via Access Grid

## Wednesday September 25, 2013

## 9:00 - 10:30AM



## Potential Molecular Targets of Fusarochromanone

**DR. TARA WILLIAMS-HART**

Department of Biological Sciences

LOUISIANA STATE UNIVERSITY SHREVEPORT

9:00 AM

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## A study of differential MiRNAs expression patterns discovered for Alzheimer's disease

**DR. PRERNA SETHI-DUA**

Department of Health Informatics and Information Management

LOUISIANA TECH UNIVERSITY

9:45 AM

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# Potential Molecular Targets of Fusarochromanone

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**Tara Williams-Hart, PhD**
**Department of Biological Sciences**
**LOUISIANA STATE UNIVERSITY SHREVEPORT**

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Mentors:

**Robert Rhoads , PhD**
Department of Biochemistry and Molecular Biology
LSU Health Sciences Center in Shreveport

**Urska Cvek, PhD**
Department of Computer Science
LSU in Shreveport

Fusarochromanone (FC101) is a mycotoxin produced by the fungus, *Fusarium equiseti*, a symbiotic fungus found on decaying cereal plants and natural grains. FC101 inhibits human cancer cell growth in vitro, reduces mouse tumor growth and increases apoptosis in vivo. Furthermore, FC101 inhibits growth of a number of human cancer cell lines and represents a putative chemotherapeutic agent. The objective of this study is to use human bladder cancer cell lines and Saccharomyces cerevisiae (budding yeast) as tools to identify the molecular target(s) of FC101. To achieve this goal we have identified genes that are differentially expressed in human bladder cancer cells and budding yeast exposed to sub-lethal concentrations of FC101 and represent potential FC101 molecular targets. Many of these genes are involved in palmitoylation and/or ubiquitylation, histone deacetylation and may be directly connected to the regulation of p53 in the DNA repair pathway. We will discuss our current efforts to study these potential FC101 molecular targets and to develop an anti-FC101 antibody to isolate FC101-interacting proteins. Molecular targets for FC101 identified in human cancer cell lines and yeast will be candidates for validation in future animal model studies.
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# A study of differential MiRNAs expression patterns discovered for Alzheimer's disease

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**Prerna Sethi-Dua , PhD**
**Department of Health Informatics and Information Management**
**LOUISIANA TECH UNIVERSITY**

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Mentors:

**Walter J. Lukiw, PhD**
Neuroscience Center of Excellence
LSU Health Sciences Center in New Orleans

**Mark DeCoster, PhD**
Department of Biomedical Enigineering
Louisiana Tech University

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The superior temporal lobe neocortex and hippocampus of Alzheimer's disease (AD) patients show signs of significant changes in physiological function that accompany amyloid plaque and neurofibrillary tangle formation, synaptic loss and neuroinflammation which are hallmarks of AD. MiRNA networks control a substantial portion of the post-transcriptional gene regulation and hence an alteration in the expression of micro RNAs (miRNAs) is emerging as a significant contributing factor to AD, when compared with age-matched controls. It is imperative to discover the biologically significant correlations among co-regulated miRNAs that play a substantial role in the progression of AD. Recent molecular, genetic and epigenetic evidence indicate that at least 5 miRNAs - including the NF-κB-regulated miRNA-9, miRNA-125b, miRNA-146a, miRNA-34a and miRNA-155 are progressively up-regulated in AD). Our prior investigation has asserted that this quartet of up-regulated miRNAs in turn down-regulate a small brain- and retinal-cell-relevant family of target mRNAs, including that encoding complement factor H (CFH), a major negative regulator of the innate-immune and inflammatory response, and synapsin-II (SYN-2) a critical neurotransmitter release protein. In this study, we are investigating miRNA expression in AD (57 cases) and age-matched controls (29 cases) by specifically concentrating to find discriminatory miRNA-146a, miRNA-9, miRNA-125b, miRNA-34a and miRNA-155 patterns. We have adapted feature selection methods to rank their abundances, which highlight the differentially expressed miRNAs in the diseased as compared to the control. Further, we employ diverse statistical measures to identify the differentially expressed miRNAs with the quartet remaining our miRNAs of interest.