Research Opportunities

There are many research opportunities at LSUHSC-NO during both the school year and summer. The table below provides information on faculty who are interested in mentoring students, including a brief synopsis of their research interests. Many of the programs listed below require students to find a research mentor. Feel free to contact faculty members whose research is of interest to you. If they cannot take on a student at that time, then they may be able to direct you to someone else who can. Click on the department titles for additional faculty information.

An "X" denotes that the student type is accepted by the investigator to perform research in his/her research laboratory. Department titles link to additional faculty information.	MS, PhD, MD/PhD Student	Medical Student	Undergraduate Student	High School Student
BASIC SCIENCE DEPARTMENTS				
BIOCHEMISTRY AND MOLECULAR BIOLOGY				
Suresh K. Alahari, PhD	X	X	—	—
Shyamal Desai, PhD	X	X	X	—
Arthur Haas, PhD	X	X	X	X
Sunyoung Kim, PhD	X	X	X	—
David Worthylake, PhD	X	X	—	—
CELL BIOLOGY AND ANATOMY				
Carmen Canavier, PhD	X	X	—	—
John Cork, PhD	X	X	X	X
Thomas Lallier, PhD	X	X	X	—
Siqiong June Liu, PhD	X	X	X	—
Jason Middleton, PhD	X	X	X	X
Ted Weyand, PhD	X	X	X	X
Matthew Whim, PhD	X	X	X	—
Tiffany Wills, PhD	X	X	X	X
GENETICS				
Judy Crabtree, PhD	X	X	X	—
Ed Grabczyk, PhD	X	—	X	—
Paula Gregory, PhD	—	X	X	—
Chindo Hicks, PhD	X	—	—	—

Andrew Hollenbach, PhD	X	X	X	X
Michael S. Lan, PhD	X	X	—	—
Wanguo Liu, PhD	X	X	X	—
Diptasri Mandal, PhD	X	X	X	—
Fern Tsien, PhD	—	—	X	X
Yaguang Xi, MD, PhD	X	—	—	—
MICROBIOLOGY, IMMUNOLOGY AND PARASITOLOGY				
Ashok Aiyar, PhD	X	X	X	—
Angela Amedee, PhD	X	X	X	X
Jennifer Cameron, PhD	X	X	X	—
Paul Fidel, PhD	X	X	—	—
Timothy Foster, PhD	X	X	X	X
Michael Hagensee, MD, PhD	X	X	X	—
Jeff Hobden, PhD	X	X	X	X
Doug Johnston, PhD	X	X	X	X
Ben Kelly, PhD	X	X	X	X
Pam Kozlowski, PhD	X	—	—	—
Chris L. McGowin, PhD	X	X	X	—
Mairi Noverr, PhD	X	X	X	—
Francesca Peruzzi, PhD	X	X	X	X
Alison Quayle, PhD	X	X	X	—
Alistair Ramsay, PhD	X	X	—	—
Krzysztof Reiss, PhD	X	—	—	—
Li Shen, PhD	X	X	X	—
Joy Sturtevant, PhD	X	X	X	X
Christopher Taylor, PhD	X	X	X	—
Guoshun Wang, DVM, PhD	X	X	X	X
Ping Wang, PhD	X	X	X	X

Z. Tom Wen, PhD	X	X & Dental Students	X	—
Hong Xin, PhD	X	X	X	—
Arnold H. Zea, PhD	X	X	X	—
NEUROSCIENCE				
Haydee E. P. Bazan, PhD	X	X	X	—
Nicolas G. Bazan, MD, PhD	X	X	—	—
Chu Chen, PhD	X	X	—	—
Jeffrey Erickson, PhD	X	X	X	—
Sonia Gasparini, PhD	X	X	—	—
Song Hong, PhD	X	X	X	X
Minghao Jin, VMD, PhD	X	X	X	—
Walter Lukiw, MS, PhD	X	X	—	—
PATHOLOGY				
Richard S. Vander Heide, MD, PhD	X	X	—	—
PHARMACOLOGY & EXPERIMENTAL THERAPEUTICS				
Wayne L. Backes, PhD	X	X	X	—
Hamid Boulares, PhD	X	X	X	—
Andrew Catling, PhD	X	—	X	—
Alfred Geller, PhD	X	X	X	X
Daniel R. Kapusta, PhD	X	X	—	—
Eric Lazartigues, PhD	X	X	X	—
David Lefer, PhD	X	X	X	__
Imran Mungrue, PhD	X	X	X	X
Donna Neumann, PhD	X	X	X	X
Charles Nichols, PhD	X	X	X	—
Dennis Paul, PhD	X	X	X	—
Martin Ronis, PhD	X	X	X	__
Kurt Varner, PhD	X	X	X	—

PHYSIOLOGY				
Scott Edwards, PhD	X	X	X	__
Jason Gardner, PhD	X	X	X	X
Jeff Gidday, PhD	X	X	X	X
Nicholas Gilpin, PhD	X	X	X	—
Lisa M. Harrison-Bernard, PhD	—	—	X	X
Patricia E. Molina, MD, PhD	X	X	X	—
Barry J. Potter, PhD	—	X	X	X
Stefany Primeaux, PhD	X	X	X	X
Robert Siggins, PhD	X	X	X	X
Liz Simon, PhD	X	X	X	X
Flavia Souza-Smith, PhD	X	X	X	X
Xinping Yue, PhD	X	X	X	—
OTHER DEPARTMENTS				
DENTISTRY AND BIOMATERIALS				
Xiaoming Xu, PhD	X	X	X	—
NEUROLOGY				
Harry Gould, MD, PhD	X	X	X	X

BIOCHEMISTRY & MOLECULAR BIOLOGY

Suresh K. Alahari, PhD salaha@lsuhsc.edu

We have identified a novel protein that we termed Nischarin, which is derived from Sanskrit that connotes slowness of motion. This molecule is ubiquitously expressed, is a cytosolic protein, and it interacts with the alpha5beta1 integrin in vivo. Tumor cell migration and invasion are important factors in development of solid tumors and are essential for metastasis to various organs. Recently, it has been shown that PAK regulates motile and invasive phenotypes of breast cancer cells through reorganization of actin cytoskeleton. In addition PAK1 plays a role in breast morphogenesis and differentiation. It has been suggested there is a functional correlation between high-grade breast tumors and enhanced PAK kinase activity, and thus PAK may have an important role in *in vivo* tumorigenesis. Since both PAK1 and PAK4 have been shown to strongly promote growth in soft agar, and since Nischarin binds to both of these kinases, it seems likely that Nischarin may affect anchorage independent growth as well as tumor growth in nude mice and thus Nischarin may be an important regulator of cancer progression.

Thus, we are investigating the role of Nischarin in breast tumor progression, and also we are in the process of identifying proteins that interact with Nischarin in breast cancer cells using proteomics as well as yeast two-hybrid approaches. A detailed understanding of the mechanistic basis of these events can significantly advance the development of new therapeutic approaches for cancer.

Shyamal Desai, PhD Sdesai@lsuhsc.edu

The overall goals of my research program are (1) to understand the role of ubiquitin and ubiquitin-like protein ISG15 in tumorigenesis, (2) to understand the mechanism of tumor cell death and drug resistance, (3) to understand the role of ubiquitin and ubiquitin-like protein ISG15 in neurodegenerative diseases.

Arthur Haas, PhD ahaas@lsuhsc.edu

Ubiquitin is a highly conserved 8600 Dalton polypeptide distributed throughout eukaryotes. The biological effects of ubiquitin are exerted through a unique post-translational modification in which the polypeptide is covalently ligated to free amino groups on various intracellular target proteins in an ATP-coupled reaction. Ubiquitination targets proteins for degradation by the 26S proteasome complex, a large (2 MDa) multi-subunit complex which recognizes multi-ubiquitinated proteins and degrades them to small peptides. The ubiquitin chain is released and is disassembled into single ubiquitin molecules which are then recycled. The major role of The ubiquitin/26S proteasome pathway for targeted degradation is a fundamental regulatory step involved in signal transduction, gene regulation, DNA repair, the stress response, cell cycle progression, apoptosis, and various disease states including muscle atrophy, Alzheimers dementia, Liddle's syndrome (familial hypertension), tumorigenesis, and Fanconi's anemia among others. We have also identified a second constitutive system within cells that is parallel but distinct from ubiquitin in which the 15 kDa interferon-like protein ISG15/UCRP is conjugated to a smaller subset of intracellular targets. ISG15 is the archetype of a small group of function-specific ubiquitin-like proteins that includes SUMO-1 and Nedd8. The conjugation of ISG15 to intracellular targets acts in trans to regulate protein-protein interactions. Work in the lab uses multiple approaches including enzymology, molecular and cell biology, and bioinformatics to study the mechanism and specificity of selected ubiquitin conjugation pathways as well as the function of ISG15 in cell regulation.

Sunyoung Kim, PhD skim3@lsuhsc.edu

The functional heart of enzymes is communication between a protein and its ligand partners, as well as the subsequent dialogue between residues during catalysis. We study members of protein families to pinpoint dynamic structural and chemical changes in the background of polypeptides that adopt similar folds. Biochemical, molecular, and biophysical tools are used to probe a kinesin motor family in dissecting the impact of potential anti-tumor agents on proteins with accepted roles in cell division, and the photolyase/cryptochrome family in answering how the protein matrix controls communication between redox-active species and developing tools to prevent skin cancer.

David Worthylake, PhD dworth@lsuhsc.edu

Both the T-lymphoma invasion and metastasis factor (Tiam1), a guanine nucleotide exchange factor for Rac, and IQGAP1, a novel Rac and Cdc42 effector, have been shown to play a role in metastasis and invasion. We are using X-ray crystallography to study fragments encompassing individual and multiple domains derived from these two large proteins; in isolation and in complex with their interactors. 3-dimensional information will be used to aid in understanding the function and activities of Tiam1 and IQGAP1 in promoting cell migration and invasion.

CELL BIOLOGY & ANATOMY

Carmen Canavier, PhD ccanav@lsuhsc.edu

I use the techniques of computational neuroscientist to quantify the electrical activity of neurons. I study basic mechanisms of synchronization and the production of the firing firing such as pacemaking, irregular firing and bursting. I am interested in central pattern generation and in the dopamine neurons of the mammalian midbrain.

Linear stability analysis of discrete and continuous systems, nonlinear dynamics, the numerical solution of nonlinear systems, and bifurcation theory are some of the tools that I use. Research Interests: Computational Neuroscience: Nonlinear Dynamics of Single Neurons and Small Networks Nonlinear dynamics of single neurons and small networks.

John Cork, PhD jcork@lsuhsc.edu
Research Interest: Digital 3D reconstruction of human embryos from serial sections.

Ray Gasser, PhD rgasse@lsuhsc.edu
Research Interest: Computer imaging of human embryos.

Thomas Lallier, PhD tlalli@lsuhsc.edu
Our lab investigates the interaction of aging, smoking and alcohol use on tissue remodeling. We are currently using an *in vitro* model system to examine how these factors influence cell adhesion, cell motility and the ability of these cells to exert contractile forces on collagen gels. In this system we are examining extracellular matrix (ECM) synthesis and secretion (of collagens and other matrix glycoproteins and proteoglycans), cell-ECM receptors (integrins) and matrix metalloproteinases (MMPs). Our system focuses on gingival and periodontal ligament fibroblasts as a means to examine the reparative properties of connective tissue cells on tissue regeneration. Our current findings indicate that aging selectively reduces the expression of several collagens and MMPs, reducing collagen gel contraction and cell motility without perturbing cell adhesion. In addition, nicotine drastically reduces gel contraction, without altering integrin expression. Finally, aging enhances the sensitivity of cells for nicotine, inducing significant alteration in ECM receptor (integrin) expression. Taken together, these data indicate that smoking and alcohol use may have an additive (or even synergistic) ability to reduce the reparative capabilities of cells in older subjects.

Siqiong June Liu, PhD sliu@lsuhsc.edu
One of the fundamental features of the central nervous system is the ability to learn from previous experience. Neuronal circuits in the cerebellum contribute to motor learning and fear memory. The underlying mechanisms are thought to be experience-dependent long-term changes in synaptic transmission between cerebellar neurons. One critical component of any of neuronal circuit are inhibitory GABAergic interneurons that control the activity of principal neurons. Our research focuses on experience-induced neuronal plasticity of inhibitory interneurons. In particular, how emotional stress and fear memory alters (1) excitatory transmission via AMPA-type glutamate receptors onto GABAergic neurons and (2) the release of GABA from these neurons. Using a combined approach that includes electrophysiology, imaging, molecular biology and behavioral techniques, we investigate the molecular mechanisms and functional consequences of stress and fear-induced synaptic plasticity.

Jason Middleton, PhD jmidd3@lsuhsc.edu
My lab focuses on the functional and structural basis of sensory processing in the auditory system and how experience and disease can change these organizational principles. Neural circuitry is defined by the architecture and pattern of synaptic connections between neurons and forms the underlying basis for how neural assemblies process and represent information about the sensory environment. We use a combination of electrophysiological, optogenetic and computational techniques to quantitatively measure neural circuit properties for the purpose of understanding the role of circuit function and structure in sensory processing. We focus on how modulatory inputs from non-sensory inputs, paired with acoustic stimulation, can reshape auditory neural circuits and allow for sensory and behavioral plasticity. Finally, we are interested in the role of all these factors in the auditory disease tinnitus. Tinnitus sufferers experience phantom auditory perceptions, the most common phenotype of which is a perception of high frequency tones. The neural basis of tinnitus is thought to arise from maladaptive plasticity in response to insult to the peripheral auditory system. Understanding how auditory circuit function can be shaped in a plastic manner under normal conditions will help us understand how the mechanisms go awry during pathological disease states.

Ted Weyand, PhD tweyan@lsuhsc.edu
Physiological and computational approaches to vision and sensorimotor integration. Projects:

1. Show videos to awake animals while recording from neurons early in their visual pathways to understand how information about the world is encoded under almost normal conditions.
2. Record from neurons in humans with movement disorders to understand how the disorder corrupts information flow through the brain.

Record eye movements in normal people and people with movement disorders as they look at pictures and read to better understand how the brain normally programs eye movements.

Matthew Whim, PhD mwhim@lsuhsc.edu
This laboratory is interested in the role of neuropeptides and stress. We are studying adrenal chromaffin cells which are part of the sympathetic nervous system. During the fight-or-flight response these cells become particularly active and release the hormones epinephrine and norepinephrine with consequent effects on blood pressure, heart rate and metabolism. In addition to the catecholamines, chromaffin cells also synthesize and secrete neuropeptides. Using

electrophysiological, molecular and behavioral techniques we are examining the hypothesis that the role of adrenal neuropeptides is to prevent an excessive response to brief periods of stress.

Tiffany Wills, PhD twills@lsuhsc.edu

My lab focuses on understanding the neuroadaptations that occur in alcohol dependence, particularly those produced by adolescent alcohol use. To do this, we utilize a wide range of techniques that include electrophysiology, proteomics, and behavioral analysis using a rodent model of alcohol dependence. Much of our work evaluates alcohol-induced changes in the bed nucleus of the stria terminalis (BNST), a region critical for relapse. Further, our studies in this region have highlighted a key role of NMDA receptor signaling in alcohol's effects.

GENETICS

Judy Crabtree, PhD jcrabt@lsuhsc.edu

Research Interest include the study of neuroendocrine and hormone-dependent oncogenesis. Neuroendocrine tumors (NETs) encompass a broad spectrum of malignancies all derived from neuroendocrine cell lineage, affecting many different organs including the gastrointestinal (GI) tract, the endocrine pancreas, the thyroid, the skin and the respiratory tract. The Crabtree lab focuses on pancreatic neuroendocrine tumors, gut carcinoids and Merkel Cell carcinoma of the skin and the role of a particular protein called RBP2.

Retinoblastoma binding protein 2 (RBP2) is also involved in neuroendocrine tumorigenesis and interfaces with the Notch signaling pathway by serving as a key component of the Notch CSL repressor complex. Further, RBP2 is overexpressed in more than 80% of clinical neuroendocrine tumors and metastases from these primary tumors. The Crabtree lab is exploring the regulation and function of this protein as a causative mechanism using in vitro cell-based and in vivo models of tumorigenesis and metastasis.

Ed Grabczyk, PhD egrabc@lsuhsc.edu

Genomic instability underlies a growing number of genetic disorders, plays a major role in cancer and contributes to aging. A type of focal instability, DNA repeat expansion, causes several dozen progressive degenerative disorders. Friedreich ataxia (FRDA) is a progressive neurodegenerative disease caused by GAA•TTC repeat expansion. The expansion represses frataxin expression in a length dependent manner.

A primary goal in our lab is to understand why GAA•TTC repeats expand, and how the expansion impairs gene expression in FRDA. We are particularly interested in the role transcription instigated structures may have in attracting enzymes of DNA repair, recombination and chromatin modification. We hope that understanding how transcription elongation is impaired in FRDA will lead to a treatment, and that understanding why DNA repeats expand or contract will lead to a cure.

Paula Gregory, PhD pgrego@lsuhsc.edu

Research interest include:

- Research in genetics education for teachers, students, the public, and health care professionals
  Psychological barriers to understanding genetics information and the impact of predictive genetic testing on family dynamics

Chindo Hicks, PhD chick3@lsuhsc.edu

Dr. Hicks is Professor of Genetics in the Department of Genetics and Director of the Bioinformatics and Genomics Program. Research in his lab focuses on five research streams: (1) Bioinformatics and computational genomics: This research stream focuses on development and application of bioinformatics and computational genomics methods and tools to analysis of multiscale-multiplatform omics and sequence data for the discovery of clinically actionable biomarkers, mutations and biological pathways driving human diseases. (2) Population genomics and epigenomics of human diseases: This research streams focuses on decoding the genomic and epigenomic landscapes of common human diseases and understanding the molecular basis of health disparities among ethnic populations. (3) Translational Genomics: This research stream focuses on harnessing multi-omics and sequence data and integrating these data with clinical information to obtain foundational knowledge about the molecular predictors of disease prognosis and clinical outcomes to guide treatment decisions. (4) Knowledge discovery and big data analytics: The goal of this research stream is to develop and apply data analytics and data science techniques to turn BIG DATA into liquidity and knowledge to improve human health. (5) Drug discovery and repurposing: The goal of this research stream is developing bioinformatics and genomics solutions and technologies for drug discovery and repurposing.

Andrew Hollenbach, PhD aholle@lsuhsc.edu

Research interest include:

- The regulation of transcription factors through phosphorylation
- Biochemical mechanisms of chromosomal translocation gene products in cancer formation

Michael Lan, PhD mlan@lsuhsc.edu

1. Transcription factors in neuroendocrine differentiation
2. Insulin gene regulation via INS-VNTR and AIRE in human thymic epithelial cells
3. Role of islet transcription factor in endocrine pancreas development
4. Mechanisms of neuroendocrine transformation and therapeutic study

Wanguo Liu, PhD wliu3@lsuhsc.edu

Research interest include:

- Genetics and biological roles of Wnt signaling in GI tumor development
- Genetics and functional analysis of DNA damage-response defects in prostate cancer susceptibility

Diptasri Mandal, PhD dmanda@lsuhsc.edu

Research interest include:

- Genetic linkage and segregation analysis of complex disorders, in particular humans cancers
- Investigation of properties of statistical genetic analysis methods through computer simulation

Fern Tsien, PhD fmille@lsuhsc.edu

One of my research interests is the correlation between DNA methylation and constitutive heterochromatin with gene silencing. Currently, we are studying chromosome and telomere instability in osteosarcomas and thymic lymphomas. Also, we are evaluating chromosomal instability in human and rhesus macaque adult stem cells. Clinical research includes molecular cytogenetic evaluation of translocation Down syndrome families. Our lab is involved with Genetics education of high school students, teachers, undergraduates, medical students, and graduate students, in the fields of Cytogenetics and Epigenetics.

Yaguang Xi, MD, PhD yxi@lsuhsc.edu

Dr. Yaguang Xi is the Professor and Vice Chair for Research in the Department of Genetics. He joined the LSU School of Medicine in August, 2016. Dr. Xi used to be a practiced surgeon prior to his Ph.D study at the Peking University, China. His lab is focusing on human cancer research, and his team engages in translating the accomplishments from the bench to the clinical practice. Dr. Xi's research interests include: identification of novel biomarkers for tumor progression and metastasis; study of mechanistic roles of non-coding RNA (microRNAs) in human cancers; and development of novel cancer therapeutics. Many state-of-art technologies, such as CRISPR and NGS, have been well integrated into their programs. Dr. Xi's research is funded by the NIH and American Cancer Society (ACS), and his lab has immediate openings to accept 2-3 graduate students. Aside from Dr. Xi's direct mentorship, each student will be assigned to an individual senior postdoc fellow to earn hands-on experience. If you are motivated for medical research, and can work as a good team player in a friendly and solid research environment, please contact Dr. Xi for an individual meeting/lab tour.

MICROBIOLOGY, IMMUNOLOGY, & PARASITOLOGY

Ashok Aiyar, PhD aaiyar@lsuhsc.edu

My laboratory works on the human pathogen Chlamydia trachomatis (Ct), which is associated with ocular and genital infections. The outcome of Ct infections can be very serious; ocular infections can lead to blindness, and genital infections have severe implications on female reproductive health including pelvic inflammatory disease, ecotopic pregnancy, and infertility. The molecular mechanisms by which Ct causes disease have been difficult to identify because the genetic tools currently available to study Ct infections on a single-cell basis are tedious and difficult to employ. We have discovered a method to identify and characterize single human epithelial cells infected by Ct permitting analyses that distinguish between bacterial effects on infected cells relative to surrounding uninfected bystander cells. We are employing this method to understand the effect of the immune response on Ct, specifically the effect of the protective host cytokine interferon gamma. Host genes induced by interferon gamma cause metabolic changes within infected cells that starve Ct of the essential amino acid tryptophan. Genital isolates of Ct have evolved mechanisms to use metabolites produced by other co-infecting pathogens to evade the effect of interferon gamma. Our work is focused on understanding the relationship between a protective host cytokine response, Ct and co-infecting pathogens at a molecular level. This understanding will permit developing effective vaccines against Ct as well as understanding conditions necessary at the site of infection for natural or vaccine-induced responses to protect against Ct.

Angela Amedee, PhD aamede@lsuhsc.edu

The Amedee laboratory investigates the pathogenesis of Human Immunodeficiency Virus (HIV), with a focus on the viral and host factors responsible for transmission. The laboratory conducts animal model studies utilizing the nonhuman primate model of HIV disease, as well as clinical translational studies in HIV-infected populations and cell culture models of HIV infection. One area of research in the laboratory involves collaborative projects with scientist from the LSUHSC

Alcohol and Drug Abuse Center of Excellence to investigate the effects of alcohol consumption and drug abuse on HIV transmission and pathogenesis. Many of these studies are done with macaques infected with Simian Immunodeficiency Virus (SIV) and are designed to identify the mechanisms responsible for sexual transmission and viral shedding in the genital mucosa. Parallel studies in clinical populations are also ongoing. Other studies in the laboratory investigate the mechanisms involved in mother-to-infant transmission of HIV and the establishment and maintenance of viral reservoirs in women. This work focuses on the selection and evolution of viral genotypes in the infant, as well as viral reservoirs, viral expression, and pathogenesis in females, using both clinical and animal model studies. The Amedee laboratory also has ongoing collaborative projects with other LSUHSC faculty that investigate the interaction of sexually-transmitted diseases with HIV. These studies are designed to decipher the dynamics of HIV replication and genital shedding, as well as susceptibility to HIV infection in the presence of co-infections using both clinical and in vitro cell culture studies.

Jennifer Cameron, PhD jcame2@lsuhsc.edu

Nearly a third of all cancer cases are caused by infections with viruses. People who have compromised immune systems, such as people living with HIV, are particularly susceptible to infections with tumor viruses and tumor virus-associated cancers. Dr. Cameron's research interests involve unraveling the mechanisms by which tumor viruses such as human papillomavirus (HPV) and Epstein-Barr virus (EBV) interact with the host and promote cancer. Her ultimate goal is to translate her findings into improved diagnostic and prognostic cancer screening tests, cancer prevention, and cancer treatment. Current projects include the evaluation of miRNAs as diagnostic and prognostic biomarkers for HPV-mediated pre-cancer lesions, and determining the molecular targets of human cellular miRNA-146, an important mediator of immune responses and cancer progression.

Paul Fidel, PhD pfidel@lsuhsc.edu

My office and laboratory are located at the dental school. I am the Associate Dean for Research and Director of the Center of Excellence in Oral and Craniofacial Research at the dental school. The research interests of my laboratory center on mucosal immunology to fungal infections. Specifically, we study host defense mechanisms against oral candidiasis (thrush) and oral microbiome in HIV-infected persons and denture stomatitis in denture wearers, and host defense against vaginal candidiasis (yeast injections) in otherwise healthy women. Animal models are used for several areas of research in addition to clinical studies.

Timothy P. Foster, PhD tfoste@lsuhsc.edu

The Foster lab investigates cellular and molecular virus-host interactions that function to modulate host antiviral responses. A complex array of cellular sensors which signal through a diversity of cell-intrinsic networks continuously surveys host cells, detecting pathogen invasion and initiating both innate and adaptive responses to infection. Despite the diversity of pathogen sensing and subsequent signaling pathways, the various pathways converge on a limited subset of transcriptional activators that initiate the host's response to infection and function in the transition from innate to adaptive responses. For millennia, herpesviruses have evolved with their hosts, over time developing or pirating homologues of cellular proteins, which target these convergence points and function to counteract and subvert the hosts' natural defenses. Subversion of these innate pathways may permit herpesviruses to establish their characteristic lifelong persistent and chronic infection within its host. Findings from these studies have broad reaching implications from regulation of cell-signaling and rationalized vaccine development to understanding the critical role of viral and host proteins in the variance of viral pathogenesis.

Michael Hagensee, PhD mhagen@lsuhsc.edu

The Hagensee laboratory studies the role of human papillomavirus (HPV) in human malignancies. Studies are focused on the increase in HPV-related cervical cancer in HIV+ women and the increase in oral cancer in HIV+ men and women. A new areas of focus is HPV's role in anal cancer from basic biology to clinical trials and similar increases in anal cancer rates in HIV+ individuals. This increase may be due, in part, to an interaction with another DNA virus, Epstein Barr Virus (EBV) which also causes human cancers. Current projects include detection of HPV and EBV in clinical specimens, determination of the systemic and local immune response against each virus, in-vitro modeling techniques and development of xenograft mouse models.. Results from these studies will aid in improved diagnostics and preventive measures for these cancers. Additional projects include development of self-testing methods to detect HPV DNA to improve cervical cancer screening and studies into improvement of HPV vaccine implementation.

Jeff Hobden, PhD jhobde@lsuhsc.edu

Dr. Hobden's laboratory is currently developing clinically relevant model systems to gain an understanding of bacterial biofilm formation on various orthopedic substrates such as alloys of titanium, polymethyl methacrylate bone cement, and ultra-high density polyethylene. With these models, Dr. Hobden's laboratory is examining the efficacy of various antimicrobials and intervention strategies to eliminate or prevent Gram-positive (methicillin-resistant *Staphylococcus aureus* [MRSA, coagulase-negative staphylococci]) and Gram negative (*Acinetobacter baumannii* and *Pseudomonas aeruginosa*) clinical isolates from growing as a biofilm on orthopedic substrates.

Doug Johnston, PhD djoh13@lsuhsc.edu

Our lab is focused on defining mechanisms of fungal pathogenesis, particularly the human host response to bloodstream

infection by the opportunistic pathogen, Candida albicans. The severity of candidiasis ranges from relatively benign superficial infections to fatal systemic disease. In fact, C. albicans is the most commonly isolated fungal pathogen among severely immunocompromised patients and is the fourth most common cause of all nosocomial infections. Although a common commensal, changes in host immune status allow Candida to penetrate the natural barriers to infection, such as the skin or mucosa, where it gains access to the bloodstream. Once bloodborne, Candida is capable of escaping the vasculature and invading nearly every tissue of the body. Mortality attributed to disseminated candidiasis is often greater than 50%, even with aggressive antifungal treatment. Importantly, for Candida to exit the bloodstream, it requires significant interaction between the fungus and both the endothelial cells lining the vascular wall and the underlying extracellular matrix. We use molecular, genetic, biochemical, immunochemical, and cell-based approaches to define the human endothelial cell response to adherence, invasion, and damage by C. albicans. Our most recent results suggest that invasion by C. albicans results in widespread endothelial apoptosis, loss of vascular barrier function, and the suppression of components of the normal wound healing response, including endothelial migration, proliferation, and differentiation. Vascular dysfunction in this setting likely provides Candida a greater window of opportunity for the establishment of deep tissue infection. We believe that clinical manipulation of the endothelial response to Candida will help to preserve vascular barrier function and lead to enhanced immunity. Our goal therefore is to define the key mechanisms involved in the host endothelial response to Candida and to identify novel potential targets for the development of new antifungal therapies.

Ben Kelly, PhD bkell2@lsuhsc.edu

Dr. Kelly's laboratory studies the biology of the protozoan pathogens, *Leishmania* and *Trypanosoma cruzi*. These parasites are transmitted via the bite of their insect vectors and are the etiologic agents of leishmaniasis and Chaga's disease, respectively. Currently, there are no really effective safe treatments to combat these debilitating and often fatal diseases that have infected approximately 20 million people worldwide. Research in my laboratory focuses on understanding molecular functions of specific parasite proteins required for viability and virulence. We are especially interested in identifying important molecular functions that are unique to these parasites, as these may represent potential drug targets for better, low toxicity therapies against these diseases.

Current major research projects are: a) Determining how a molecular scaffolding protein, termed "LACK", promotes expression of parasite genes important for virulence; b) Characterizing phosphorylation pathways important for parasite differentiation and virulence (in collaboration with Dr Juan Pizarro (Tulane University) and c) Identifying novel parasite-therapeutic compounds by screening chemical libraries for anti-parasitic activity against *T. cruzi*) in collaboration with Dr Donna Neumann (LSUHSC, Department of Pharmacology and Experimental Therapeutics) and Branko S. Jursic (University of New Orleans).

Pam Kozlowski, PhD pkozlo@lsuhsc.edu

The majority of the research in the Kozlowski lab is focused on the identification of vaccines and mucosal antibodies that will prevent HIV transmission in adults and infants. Most HIV infections are acquired at mucosal surfaces lining the gastrointestinal or genital tract. Antibodies produced by cells in these mucosal tissues are transported into local secretions, where they could act as a first line of defense in preventing HIV entry. Therefore, a goal in HIV vaccine development is to generate HIV-specific antibody-secreting cells in both gastrointestinal and genital tract tissues. However, it is not entirely clear which determinants on HIV outer surface proteins should specifically be incorporated into vaccines for induction of mucosal antibodies that are optimal for preventing HIV entry. Vaccines administered by a mucosal route are also more likely to generate mucosal antibodies than those given by the intramuscular route, but the optimal mucosal administration routes (e.g. nasal vs. oral) for preventing breast milk transmission in infants or sexual transmission in adults are unknown. Work in the lab is geared toward resolving these issues. Novel anti-HIV monoclonal antibodies that could be used as therapeutic treatments in HIV-infected individuals are also in development.

Chris L. McGowin, PhD cmcgow@lsuhsc.edu The McGowin Lab is focused on several clinical, epidemiological, and basic mechanistic aspects of sexually transmitted infections (STIs), namely related to *Mycoplasma genitalium* and *Trichomonas vaginalis*. With the goal of improving sexual and reproductive health in women, and also maternal/fetal health during pregnancy, we conduct both clinical and laboratory investigations that assess the mechanistic bases of STI pathogenesis. Additionally, with a focus on the mucosal microenvironment during infection, we utilize novel tissue models to dissect the mechanisms by which untreated STIs enhance acquisition and/or transmission of HIV. Lastly, dovetailed with our academic research efforts, and in collaboration with several industry partners, we are also actively involved in the design, development, and/or clinical trial evaluation of molecular in vitro diagnostic (IVD) tests for STIs and other urogenital pathogens.

Mairi Noverr, PhD mnover@lsuhsc.edu

The Noverr laboratory focuses on investigating mechanisms of immunomodulation by the opportunistic yeast Candida albicans during host-pathogen interactions. The majority of humans are chronically colonized at various mucosal surfaces with C. albicans. This fungal pathogen can cause a variety of infections, ranging from mucosal to systemic and invasive Candidiasis. Previous work by Dr. Noverr revealed that C. albicans produces immunomodulatory oxylipins that are similar

in function to host eicosanoids. These fungal oxylipins not only can influence the host immune response, but also alter the microbiology of the fungus, promoting morphogenesis and biofilm formation. The central hypothesis is that production of oxylipins by both fungi and the host modulates the microbiology of the fungus and the host-pathogen interaction in favor of chronic infection or persistence. Projects in the Noverr laboratory are aimed at testing this central hypothesis and include determining the effects of host eicosanoids and fungal oxylipins during Candida pathogenesis in several models of mucosal and systemic infection, in modulating host immune cell function, and in Candida morphogenesis and biofilm formation, both monomicrobial and polymicrobial. In addition, the laboratory is investigating novel in vivo models of biofilm formation at mucosal surfaces, both during experimental vaginitis and denture stomatitis. These models will allow investigation of host, bacterial, and fungal factors that affect Candida biofilms in a clinically relevant setting.

Francesca Peruzzi, PhD fperuz@lsuhsc.edu

Plasma microRNAs as biomarkers for mental health status in HIV+ patients: Human Immunodeficiency Virus (HIV)-infected individuals have an increased risk of developing neurocognitive disorders, which could unfavorably impact their adherence to HIV treatment. Thus, the identification of early markers of neurocognitive impairment in these patients could lead to early clinical interventions that improve psychosocial functioning and slow cognitive decline through the improved adherence to antiretroviral therapies. To date, there are no biomarkers for neurocognitive impairments, and the diagnosis of HIV-associated neurocognitive disorders (HAND) is based on an analysis of neurocognitive function and neuroimaging after manifestation of symptoms, when the mental status of the patients has deteriorated. MicroRNAs are key regulators of synaptic plasticity and brain development and, as such, could contribute to the etiopathology of neurocognitive disorders. Therefore, we hypothesize that a specific set of extracellular microRNAs, which can be detected in plasma, can serve as diagnostic and/or prognostic markers for the development and progression of impaired mental status of HIV+ patients.

We have developed a reliable, accurate and sensitive protocol to profile microRNAs in body fluids by quantitative PCR. In collaboration with the Louisiana State University (LSU) HIV Outpatient Clinic (HOP), we are currently recruiting HIV patients, performing neurocognitive testing, and collecting plasma samples. Our objective is to identify which plasma microRNAs can delineate cognitive impairments and depression with high specificity and sensitivity. The sensitivity of microRNA biomarkers is further assessed by investigating the mechanisms of microRNA stability in plasma through the analysis of exosome and Argonaute2-associated microRNAs. The ultimate goal of this project is to develop non-invasive clinical tools for the early detection of neurocognitive impairments in HIV+ patients.

Alison Quayle, PhD aquayl@lsuhsc.edu

The central theme of our research is immune defense in the human female genital tract, and the mucosal immune response to HIV and the unique obligate intracellular bacteria *Chlamydia trachomatis*. Specific interests include (1) Elucidation of the *Chlamydia trachomatis*-specific adapative response in the human endocervix, and (2) Determination of the immunoevasive strategies used by *C. trachomatis* to adapt to, and survive in, the human genital milieu (3) How *C. trachomatis* influences HIV transmission (4) How female steroid hormones, particularly long-acting progestins, influence the female genital milieu and early pathogen transmission events.

Alistair Ramsay, PhD aramsa@lsuhsc.edu

The research interests of the Ramsay laboratory are centered on the immune biology of infections by intracellular pathogens, particularly via mucosal tissues, and in pulmonary infection by the opportunistic pathogen *Pneumocystis*. We are also interested in the development of novel, recombinant vectors that can effectively deliver key vaccine immunogens to different sites and tissues in the body. Our ultimate goal is to develop improved vaccines, particularly those that stimulate protective immune responses at mucosal sites of infection, against a variety of currently intractable diseases that have presented major difficulties for conventional approaches to immunization. We are also working to gain a clearer understanding of the mechanisms of action of these vaccines and of underlying host:pathogen interactions. A primary focus of the lab at present is understanding host:pathogen interactions in *Mycobacterium tuberculosis* infection, using immune assays, genomics and bioinformatics. Genome-wide transcriptional studies in the lab have begun to reveal novel host cell signaling pathways that could ultimately be involved in defense against TB. We are also interested in finding improved TB immunization strategies. Our approach is based largely on the development and evaluation of recombinant BCG and viral vectors engineered for enhanced immunogenicity, including the identification of new, immunogenic vaccine targets in *M. tuberculosis* using immune genomics and bioinformatics. Identification of host gene expression 'signatures' that correlate with vaccine efficacy has the potential to accelerate the development of novel protective vaccines against TB and other currently intractable infectious diseases.

Krzysztof Reiss, PhD kreiss@lsuhsc.edu

The Reiss lab main focus of research center on the Insulin-like Growth Factor I Receptor, IGF-IR, and its association with cancer. There are three main projects in the lab. 1) *Molecular strategies against Insulin-like Growth Factor I Receptor (IGF-IR) in Brain Tumors.* Work from other laboratories and our recent findings strongly indicate that the signal from activated IGF-IR supports growth and survival of cancer cells, and the functional IGF-IR is required for supporting malignant transformation mediated by different cellular and viral oncogenic proteins. We are developing and testing different strategies aiming at the IGF-IR in vitro and in experimental animals. These include use of dominant negative

mutants, antisense strategies, neutralizing antibodies, small inhibitory RNAs (siRNAs), and small molecular weight inhibitors of the IGF-IR tyrosine kinase activity. 2) *The contribution of IGF-IR to JC virus T-antigen –mediated cellular transformation.* Polyomaviruses are highly suspected to participate in the development of cancer. By investigating functional association between JCV T-antigen and the IGF-IR system, we have found that: **(i)** cells with targeted disruption of the IGF-IR gene are resistant to JCV T-antigen –induced transformation; **(ii)** 1.5 x104 IGF-IR molecules *per* cell fully supports JCV T-antigen –mediated anchorage-independent growth *in vitro*; **(iii)** the major IGF-IR signaling molecule, IRS-1, directly interacts with JCV T-antigen, and translocates IRS-1 to the nucleus. 3) *Role of IGF-I, and Tumor Necrosis Factor α (TNFα*) *in HIV associated dementia.* In a substantial number of AIDS patients HIV infection results in a serious neurological disorder of the central nervous system, HIV–associated dementia (HAD). Currently, there is no specific treatment for HAD, mainly because of an incomplete understanding of how HIV infection causes neuronal injury and apoptosis. Activation of the insulin-like growth factor I receptor (IGF-IR) represents a strong neuro-protective mechanism against a wide variety of insults. Therefore, our future task is to determine molecular pathways involved in the cross-talk between IGF-I, TNFα receptors and HIV proteins in neural progenitors of the Central Nervous System.

Li Shen, PhD lshen@lsuhsc.edu

Research in Dr. Shen's lab is focused on molecular biology and pathogenesis of human pathogen, *Chlamydia* spp. Molecular, genetic, biochemical, cell biology, RNA-seq, proteomics, and computational approaches are utilized in combination to address: 1) *Control of the type III secretion system (T3SS) in Chlamydia*. *Chlamydia* uses its T3SS to deliver anti-host effector proteins into host cells to subvert host immunity. By defining common and *Chlamydia*-specific T3SS controls at the levels of transcription and post-transcription, we seek to more completely understand the "secretion regulation" mechanism that is utilized by bacteria to modulate host function and success infection. 2) *Diversity and development of C. trachomatis*. We are interested in elucidating how *Chlamydia* adapts to adverse conditions, persists in the host cells, and contributes to disease progression. We have developed advanced techniques allowing us to qualitatively and quantitatively monitor the dynamic behaviors of *C. trachomatis* in response to the developmental signal, antimicrobial exposure, and other environmental cues using cell culture models. Insights obtained will pave the way for the future development of novel therapies targeting the T3SS against *Chlamydia* infections.

Joy Sturtevant, PhD JSturt@lsuhsc.edu

Pathogens have evolved to thwart our immune response and/or 'peacefully' reside within our body. How do they do this? Our laboratory focuses on the initial interactions between pathogen and host, and the intracellular signaling events that dictate a response in either host or pathogen. We are studying two different microorganisms to address these responses. First, we are studying *Candida albicans*, the most common opportunistic fungal pathogen and the 4th most common nosocomial infectious agent but also a member of the normal microbiota. The ability of the pathogenic fungus *Candida albicans* to cause disease requires rapid adaptation to changes in the host environment and to an evolving host immune response. We are particularly interested in *Candida* 'survival' factors and their effect on host-pathogen interaction. The second project is a collaborative venture with Dr. Haas in Biochemistry. This project is researching on how *Shigella* Type III secretion proteins reprogram the host innate immune response. *Shigella* is a major contributor to gastroenteritis and the causative agent of dysentery. We are focusing on how *Shigella* E3 type ubiquitin ligases usurp host signaling pathways. We feel that these studies will advance our knowledge of how pathogens adapt and survive in the host and will assist in the identification of new antimicrobial strategies. Molecular genetic, proteomic, cell culture and cell biology techniques are used to answer these questions.

Christopher Taylor, PhD ctay15@lsuhsc.edu

The human body is host to diverse communities of microbial organisms collectively referred to as the human microbiome. These communities include bacteria, fungi and archaea, some of which perform important metabolic functions. Interventions such as antibiotic treatment and environmental interaction can disrupt these communities and changes in community structure have been shown to play a major role in several diseases. We use 16S ribosomal RNA collected from these communities in tandem with high-throughput sequencing to study and analyze these microbial communities and their relationship with human health and disease. The primary focus of the lab is computational analysis and visualization of microbial community structure performed in collaboration with clinicians and basic scientists. In addition to the human microbiome, we also investigate microbial communities in model organisms such as mouse, rat, and non-human primate studies with a focus on translational research.

Guoshun Wang, DVM, PhD gwang@lsuhsc.edu

The Wang lab has several research directions, including 1) cystic fibrosis (CF) pathogenesis and CF gene and stem cell therapy, 2) chloride anion and phagocytic innate immunity, 3) alcohol antiinflammation and immunosuppression. 1) CF, one of the most fatal genetic diseases in the white population, is caused by mutations in CF transmembrane conductance regulator (CFTR), a cAMP-activated chloride channel. Over 90% of CF patients die of uncontrollable lung bacterial infections. It is not fully defined how the chloride channel defect leads to the lung host defense failure. The Wang lab has provided the first evidence that the CFTR defect results in deficient chloride transport to neutrophil phagosomes. Such a deficiency impairs the production of hypochlorous acid (HOCl), the chlorine bleach, in the organelle for effective bacterial killing. Now the lab is further characterizing the importance of this defect in CF lung pathogenesis and is also pursuing

molecular therapies to correct the defect. 2) Chloride is the most abundant anion in human body. This anion plays an important role in regulating cell volume and pH, and equilibrating resting membrane potential. However, neutrophils use this anion in a special way to generate HOCl, a most potent microbicidal oxidant. The molecular mechanism is not known how the cells acquire this anion from the extracellular environment and further transport it to the phagosomes. The lab is characterizing what chloride channels are involved in the ion transport process in neutrophils. 3) Alcohol has long been recognized to have antiinflammation and immunosuppression effects. However, the molecular mechanism underlying the phenomenon is not well defined. The previous research from the Wang lab has identified that ethanol upregulates Glucocorticoid-induced Leucine Zipper, a steroid-responsive gene, thus modulating cell cytokine expression and secretion. The lab is currently characterizing how alcohol utilizes the glucocorticoid signaling pathway to modulate the cell immune status.

Ping Wang, PhD pwang@lsuhsc.edu

Fungi are the lower eukaryotic microorganisms sharing certain cellular characteristics with mammals and some of them can cause serious infections to humans. As such, fungal studies help to understand eukaryotic biology and discover novel therapeutic agents against fungal infections. Research in our laboratory is focused on how the pathogenic fungus *Cryptococcus neoformans* senses the environment and relays signals allowing for coordinated cellular growth, differentiation, and pathogenicity. Our long-term research goal is to advance our basic knowledge of biology and to identify novel targets for effective antifungal drugs. We are particularly interested in deciphering the genetic make up and virulence roles of heterotrimeric G-protein mediated signaling by employing the approaches of genetics, molecular biology, and in vitro models. Our favorite research subjects are cryptococcal G protein subunits, regulators of G protein signaling, and the signal transducing adaptor protein.

Our second research interest is to explore the mechanism by which *C. neoformans* exhibits neurotropism, as the fungus has the predilection for the animal central nervous system and causes meningoencephalitis. We found that the novel intersectin homolog Cin1 exhibits two isoforms, similar to human and mouse intersection ITSN1, whose long isoform is only expressed in the brain. We are actively following a research lead that suggests a link between Cin1 isoform differentiation and fungal neurotropism.

Tom Wen, PhD zwen@lsuhsc.edu

Dr. Wen's research primarily focuses on understanding the molecular mechanisms governing bacterial biofilm formation and the ecology of oral biofilms and on identification of novel strategies against human dental caries. It utilizes various modern techniques, such as functional genomics, transcriptomics and metabolomics, as well as *in vitro* and *in vivo* models that mimic human oral cavity. With the support of NIH/NIDCR, one of Dr. Wen's major projects focuses on biofilm regulatory protein BrpA in virulence modulation of *Streptococcus mutans,* a key etiological agent of human dental caries. BrpA in *S. mutans* is a surface-associated protein with crucial roles in cell envelope biogenesis, stress tolerance response and biofilm formation, traits critical to pathogenicity of this bacterium. Current effort includes elucidation of the mechanisms how *S. mutans* modulates BrpA expression in response to various environmental perturbations and how BrpA regulates *S. mutans'* pathophysiology. In collaborative effort, Dr. Wen *et al.* have recently shown that *S. mutans* can actively release (secrete) eDNA and produces membrane vesicles as a delivery vehicle of eDNA and other virulence attributes. Another major project in Dr. Wen's lab focuses on the biogenesis of membrane vesicles and their role in active eDNA release in *S. mutans,* as well as on identification of factors contributing to eDNA-mediated biofilm formation. In addition, a systems biology approach, which integrates transcriptomics and metabolomics with multiple-species consortium, is also being used to elucidate the interactions between *S. mutans* and other major species in dental plaque biofilms and their impact on the pathophysiology of cariogenic *S. mutans*.

Hong Xin, PhD hxin@lsuhsc.edu

Hematogenously disseminated candidiasis in humans has become the third leading cause of hospital-acquired blood stream infections and despite antifungal therapy at least 40% of affected individuals will die of this disease. As there is no approved antifungal vaccine for use in humans and significant therapeutic challenges remain, our approach is disease prevention through active vaccination and/or passive immunization with protective antibodies. Research interests of my lab are focused on (i) Development of anti-*Candida* vaccine against disseminated candidiasis caused by all the *Candida* species of medical significance. (ii) To develop combination therapy with monoclonal antibody (mAb) cocktails for disseminated candidiasis. (iii) To determine the protective efficacy of vaccines and therapeutic mAb cocktails in neutropenic mouse model of disseminated candidiasis. (iv) To investigate the protective mechanisms of the protective peptide-specific mAbs.

Arnold H. Zea, PhD azea@lsuhsc.edu

Dr. Zea's laboratory research is focused in the immune-biology of cancer and tuberculosis. In cancer, Dr. Zea is studying the mechanisms by which L-arginine and L-glutamine metabolism (Figure 1) regulates tumor growth-inhibition and immune responses. This research will help to better understand mechanisms of resistance, tumor evasion and to develop new therapeutic strategies to control and possibly eradicate tumors. The knowledge and experience gained in cancer related research has allowed Dr. Zea to explore whether similar mechanisms can occur in infection by *Mycobacterium*

tuberculosis (*Mtb*). He is studying *in vitro* as well as *in vivo* the mechanisms by which *Mtb*-cyclic-AMP (cAMP) regulates arginase induction, nitric oxide and cytokine production used by *Mtb* to survive and persist inside macrophages. The main goal of the projects is to identify pathways within the L-arginine metabolism that can be targeted to inhibit tumor and *Mtb* growth-persistence. These findings can be determinant in the development of new unconventional therapies that could eliminate tumors and tuberculosis based on the dependence of L-arginine or L-glutamine. It will also advance in the treatment of multi-drug and extensively-drug resistance tuberculosis where first line drug therapies are useless.

NEUROSCIENCE

Haydee E. P. Bazan, PhD hbazan1@lsuhsc.edu

Corneal inflammation and wound healing. Our laboratories study the effect of lipid inflammatory mediators and growth factors in the cornea and how they affect tissue repair. A second line of research is the role of neurotrophins and pigment epithelial derived factor (PEDF) in the regulation of corneal nerve re-growth after injury. Corneal nerves are important in maintenance of corneal integrity and prevention of dry eye following corneal injury. These studies are of clinical significance. A variety of molecular and cellular biology techniques as well as cells in culture and in vivo models of corneal injury are employed.

Nicolas G. Bazan, MD, PhD nbazan@lsuhsc.edu

Our research has led to the identification of novel cell signaling responses of the nervous system (brain and retina) to various forms of injury. The injuries that are being studied in my laboratory have in common an upsurge of oxidative stress, protein misfolding and apoptotic cell death. The models used include experimental cerebrovascular disease (e.g. stroke), traumatic head injury, epilepsy, retinitis pigmentosa, age-related macular degeneration and other neurodegenerative diseases (e.g. Alzheimer's and Parkinson's).

In addition, the novel cell signaling targets are being explored in eye inflammatory models, in neuropathic pain, and in unique assays for pathoangiogenesis. Current studies seek to identify mechanisms that regulate the synthesis and availability of the newly discovered messenger neuroprotectin D1 as well as the downstream signaling including Bcl-2 proteins and proinflammatory genes. These cellular and molecular events are explored in terms of synaptic remodeling, neuron regeneration and neuroprotection. Overall, the recently uncovered messengers may contribute to integrate responses for cell survival with active participation of cytokines and growth factors/neurotrophins.

This research is leading to the application of novel endogenous molecules, as well as synthetic low molecular weight compounds that penetrate the blood brain barrier to experimental therapeutics. Our research engages cell biology, biochemistry, pharmacology, and experimental models of diseases, complemented with powerful lipidomic tools. We are currently defining the translational implications of these findings as novel therapeutic strategies.

Chu Chen, PhD cchen@lsuhsc.edu

My laboratory studies membrane ion channel modulation, synaptic transmission and plasticity, and neuronal survival using electrophysiological recording, optical imaging and molecular biology approaches. The specific research interests in my lab currently focus on the roles of cyclooxygenase-2 (COX-2)-mediated lipid signaling in hippocampal synaptic transmission, plasticity and neurotoxicity. COX-2, an inducible key enzyme converting arachidonic acid to prostaglandins, is not only essential to our normal physiological function, but it is also involved in inflammatory responses, traumatic brain injury, stroke, multiple sclerosis and several neurological disorders, such as epilepsy, Parkinson's and Alzheimer's diseases.

Recent evidence indicates that COX-2 is also capable of oxygenating endogenous cannabinoids to form novel prostaglandins. However, physiological and pathological functions of these COX-2 oxidative metabolites of endocannabinoids are still unknown. The goals of ongoing projects in this lab are to elucidate cellular and molecular mechanisms underlying COX-2-mediated prostaglandin signaling in hippocampal synaptic physiology and pathology, and to provide important evidence or clues to design drugs aimed at treating, ameliorating, or preventing neuroinflammation-, traumatic brain injury-, ischemia-induced neuronal damage and neurodegenerative diseases, resulting from abnormally excessive activation of COX-2.

Jeffrey Erickson, PhD jerick@lsuhsc.edu

My laboratory studies the molecular and cellular properties of the transporters that package the major excitatory neurotransmitter glutamate into synaptic vesicles. We have cloned three genes that encode these transporters, characterized their functional activity and mapped their distributions in the brain. Using cultured neurons, we have found that they are differentially and cooridinately regulated with the transporter for the major inhibitory transmitter GABA. Thus, the balance of expression of vesicular transporters for glutamate and GABA may determine the balance of excitatory and inhibitory transmission in the brain. We are currently studying the molecular and cellular mechanisms that govern the plasticity of the storage and synaptic release of these neurotransmitters from neurons.

Sonia Gasparini, PhD sgaspa1@lsuhsc.edu

Information in the brain is encoded through patterns of action potentials, that occur in specific sets of neurons at a specific time. I am interested in studying how information is processed in the brain and in particular how neurons integrate the

inputs they receive on their dendrite to generate an output at the soma/axon region. This very complex process depends on the characteristics of the synaptic inputs, the morphology of the dendritic tree and the voltage-dependent currents there expressed. In particular, we are interested in understanding the integrative properties of pyramidal neurons in the hippocampus and the entorhinal cortex, regions that are fundamental in processes of memory and learning.

For our research, we employ electrophysiological techniques (dendritic and somatic recordings) and two-photon imaging and ungaging of caged neurotransmitters (such as glutamate) on brain slices. Using these techniques, we have recently shown that different spatio-temporal input patterns can differentially engage the intrinsic dendritic voltage-dependent currents to generate specific outputs that can be related to the main behavioral states of the animal.

Song Hong, PhD shong@lsuhsc.edu

My laboratory focuses on lipidomic pathways, in macrophages, endothelial/epithelial cells, and organs, for the biosynthesis of omega-3 essential fatty acids derived lipid-mediators that regulate inflammation, angiogenesis, fibrosis, diabetic wound healing and nephropathy; as well as modulate ocular diseases. We identify and elucidate chemical structures and bio-functions of lipid mediators using a variety of biochemical and biological approaches. We study wound healing, inflammation, fibrosis, and diabetic nephropathy and neuropathy. We are interested in the regulation of innate immunity by lipid mediators, and the links to inflammatory diseases. Lipid modification of bioactive proteins and the bio-functions are also of our interest. http://www.medschool.lsuhsc.edu/neuroscience/faculty_detail.aspx?name=hong_song

Minghao Jin, VMD, PhD mjin@lsuhsc.edu

The current research in my laboratory is focusing on understanding the molecular mechanisms involved in: (1) regulation of the visual cycle and (2) degeneration of photoreceptors in blinding diseases caused by mutations in the visual cycle genes. The visual cycle is a biochemical pathway that regenerates 11-*cis*-retinal chromophore responsible for sensing light in the retinal photoreceptors. Using expression cloning and yeast two hybrid screening, we have isolated several genes that may regulate the visual cycle. We are currently investigating the functions of these genes using *in vitro* and *in vivo* biochemical and molecular biological approaches. Since the visual cycle is crucial for vision and retinal function, mutations in the visual cycle genes (e.g. RPE65 and IRBP) cause severe blinding diseases such as Leber's congenital amaurosis (LCA) and retinitis pigmentosa (RP). We are studying the molecular mechanisms by which the disease-associated mutations cause photoreceptor degeneration in mice models for LCA and RP. Other ongoing projects in my laboratory include rescuing or delaying the photoreceptor degeneration in the mice models by genetic and pharmacological approaches.

Walter Lukiw, MS, PhD wlukiw@lsuhsc.edu

The major research interests in my laboratory are: Alzheimer's disease; Parkinson's disease; prion-based neurodegeneration; neurotoxicology; brain-specific gene transcription; chromatin structure; molecular basis for memory; DNA array analysis and bioinformatics; gene expression and factors that modulate gene expression; transcription factors; neuroprotective omega-3 fatty acids; docosahexanoic acid (DHA) and neuroprotectin D1 (NPD1); neurotoxic environmental metals and especially aluminum, iron, mercury, cadmium, zinc and copper neurotoxicity and neurotoxicology; secretory products of aged and stressed brain cells, hypoxia; spreading mechanisms in neurodegenerative disease; novel neuroprotective agents; age-related macular degeneration.

PATHOLOGY

Richard S. Vander Heide, MD, PhD rvande3@lsuhsc.edu

Dr. Vander Heide's laboratory is interested in two projects. The long-term goal of the first project is to design rational approaches to limiting or preventing the cell death that occurs in a myocardial infarction (heart attack). Although much is known about the physical and biochemical status of dead cells, the exact sequence of events that occur leading to cell death is still open to debate. Among the questions addressed are the role of apoptosis versus necrosis and the signaling pathways, both extracellular and intracellular, that activate endogenous protective pathways.

The second project seeks to design and develop a new and unique delivery vehicle capable of delivering therapeutic drugs to ischemic tissue and thereby delay or prevent myocyte death. Many clinical trials of anti-ischemic drugs have been hindered by the inability to achieve effective concentrations of the drug at the critical site, the myocyte. In this project, we will develop a new delivery vehicle using the endogenous anti-oxidant protein catalase and will test the ability of the delivered catalase to inhibit myocyte cell death using both in vitro and in vivo model systems of ischemia/reperfusion injury.

PHARMACOLOGY

Wayne L. Backes, PhD wbacke@lsuhsc.edu

Cytochrome P450 enzymes are responsible for the metabolism of virtually every foreign compound that enters an organism. The major function of the P450 system is to carry out oxidation reactions, usually by hydroxylation of the substrate. Most of these reactions lead to products/intermediates that are more water-soluble and consequently more rapidly excreted. However, some products are reactive, capable of binding to biological macromolecules, an initial step leading to carcinogenesis. P450 enzymes do not act independently, but require formation of a 1:1 complex with the

flavoprotein NADPH-P450 reductase (reductase), which transfers electrons to P450. Because P450 exists in a large excess over reductase in vivo, the P450 enzymes must effectively compete for the reductase or be metabolically silent. The goal of this project is to examine the organization of multiple P450 enzymes and NADPH-cytochrome P450 reductase in the membrane, and to determine the potential for the formation of P450-P450 complexes that can influence the function of these enzymes. The studies include characterization of these interactions, identification of the contact points among the proteins, and determination of their effects on metabolism of hydrocarbons, carcinogens, and other foreign compounds.

Hamid Boulares, PhD hboulr@lsuhsc.edu

My research interests focus on studying the intricate relation between the enzyme poly(ADP) ribose polymerase–1 (PARP-1) and associated endonucleases DNAS1L3 and DFF (DNA fragmentation factor) and oxidative stress in inflammation (asthma and atherosclerosis) and cancer. We use both cell culture systems and animal models to perform our studies. The induction of cellular oxidative stress is associated with the generation of reactive oxygen species (ROS) and consequent DNA damage. The breakage of DNA strands in turn results in activation of PARP-1, which, with NAD as its substrate, catalyzes the addition of long branched chains of poly(ADP-ribose) to a variety of nuclear proteins including itself. Such poly(ADP-ribosyl)ation contributes to various physiological and pathophysiological events that are associated with DNA strand breakage, including DNA replication, repair of DNA damage, gene expression, malignant transformation, and apoptosis. In several pathological situations that involve massive DNA damage, excessive activation of PARP-1 depletes cellular stores of both NAD and its precursor, ATP, leading to irreversible cytotoxicity and cell death (apoptosis or necrosis). Such depletion of cellular energy reserves results in perturbation of the function of mitochondria, the main source of cellular ATP, and a consequent increased generation of ROS. We therefore investigate the cross talk between PARP-1 activation (and depletion of cellular energy reserves), its associated proteins, and mitochondria in an attempt to understand the mechanisms both of cell injury associated with inflammation and of cancer progression and resistance to chemotherapeutic drugs both at the levels of cell and whole animal.

Andrew Catling, PhD acatli@lsuhsc.edu

Cell fate is regulated by adhesion to the extracellular matrix. In normal cells, cell-matrix interactions are required for survival and proliferation, and also for regulated cell movement, for instance during embryogenesis and inflammation. Importantly, this control of cell fate is lost in two leading causes of human mortality, neoplasia and atherosclerosis, resulting in adhesion-independent growth, survival and metastasis of cancer cells, and inappropriate growth and migration of smooth muscle cells following vascular injury. These cellular functions are in part controlled by signaling through the MAP kinases pathway. My laboratory is interested in how adhesion signals are transduced by the MAP kinase pathway, and how signaling is altered during these disease processes.

Alfred Geller, PhD agelle@lsuhsc.edu

Dr. Alfred Geller lab: Genetic analysis of visual learning and neural gene transfer technology

To enable studies on cognitive encoding, the lab has established a system that targets some of the essential information for visual object discriminations to an identified neocortical circuit. Specifically, genetic activation (via a virus vector) of protein kinase C pathways in several hundred neurons in rat postrhinal cortex improves accuracy on new visual shape discriminations (J. Neurosci. 2005, 25;8468-81). Further, some of essential information for performance is encoded in the genetically-modified circuit: After gene transfer and learning, creation of small lesions that ablate the genetically-modified circuit selectively reduces performance for discriminations learned after gene transfer (PNAS 2010 107;14478-83). During learning, both the transduced neurons and the genetically-modified circuit are preferentially activated (Hippocampus 2012, 22;2276-89). Ongoing studies are examining the cellular and molecular mechanisms that encode this learning.

The lab is also developing neural gene transfer technology, including gene transfer to connected neurons and tools to visualize individual neurons, using molecular biology/biotechnology/synthetic biology strategies.

Daniel R. Kapusta, PhD dkapus@lsuhsc.edu

The research in my laboratory focuses on understanding the physiological responses, sites of action (e.g., brain, periphery, kidneys), and neural and hormonal mechanisms by which different opioid systems affect cardiovascular and renal function. These investigations are important from a clinical standpoint since we have demonstrated that the renal excretory responses produced by certain opioids, specifically those that activate *kappa* and *ORL1* opioid receptor subtypes, have important therapeutic applications in management of pathological states associated with edema and/or hyponatremia. Based on knowledge obtained from these investigations, we are developing novel 'aquaretics' (selective water diuretics) for the clinical management of fluid retaining states. In addition to therapeutic applications, we use pharmacological and genetic opioid receptor knockout approaches to investigate the physiological and potential pathological importance of endogenous opioid pathways in the regulation of cardiovascular and renal function and central autonomic control in normal, stressful and pathological states (e.g., hypertension, cirrhosis with ascites and congestive heart failure).

Eric Lazartigues, PhD elazar@lsuhsc.edu

Angiotensin-II (Ang-II) exerts its profound cardiovascular (CV) and volume homeostatic properties through activation of

specific receptors, primarily Ang-II type 1 (AT1), located both in the periphery and in the brain. Evidence has shown the importance of the brain renin-angiotensin system (RAS) in the maintenance of normal blood pressure (BP) and in the development of hypertension. Although, new genetic and pharmacological tools have improved our understanding of the global functioning of this system, the role of its different components and their interactions remain poorly understood due to the difficulty in experimentally dissecting brain versus peripheral RAS. Recently, a new element of the RAS, named ACE2, has been identified and is believed to degrade Ang-II to the vasodilator peptide angiotensin-1-7 (Ang1-7).

Very recently, we identified the presence of ACE2 in the brain and our interests focus on the role of this enzyme in modulating the activity of the brain RAS during the development of neurogenic hypertension. Using non-transgenic (NT) and genetically-engineered mice in combination with molecular, physiological, and pharmacological tools, our laboratory is dedicated in assessing the relative physiological significance of central ACE2 in normal and pathophysiological regulation of BP and other CV diseases.

For additional details, go to http://www.medschool.lsuhsc.edu/pharmacology/faculty/lazartigues_lab.htm.

David Lefer, PhD dlefe1@lsuhsc.edu

Research in my laboratory is focused on the development and translation of novel therapies for the treatment of cardiovascular diseases including: acute myocardial infarction, heart failure, hypertension, and peripheral vascular disease. Our research program is primarily focused on preclinical studies that range from isolated cells and mitochondria to gene targeted mouse, rat, and swine models of cardiovascular diseases. These animal models involve dyslipidemia, hypertension, acute myocardial infarction, and heart failure. We utilize these robust animal models to evaluate the protective effects of cardiovascular drugs and devices to reduce cardiovascular morbidity and mortality in end-stage cardiovascular diseases. One of our major research focus areas is endogenously produced gastotransmitters, nitric oxide (NO) and hydrogen sulfide (H2S) that are involved in cardiovascular homeostasis. Recent experimental and clinical evidence suggests that both NO and H2S levels are significantly reduced in the heart and circulation in cardiovascular disease states. At present, we are involved in both preclinical and clinical studies examining the cardioprotective actions of novel therapeutic agents that increase either nitric oxide or hydrogen sulfide bioavailability.

Imran Mungrue, PhD imungra@lsuhsc.edu

The major goal of my lab is to identify mechanisms to explain how biological systems function, define how dysfunction leads to the genesis of human disease, and develop novel approaches for treatment. The lab attempts to achieve these goals taking advantage of multi-disciplinary and collaborative approaches, using cutting edge cores and equipment available at LSU-HSC, along with hard work. The spectrum of Cardiovascular and Metabolic disease represents the largest human health burden worldwide. The lab aims to define Genetic and environmental factors that affect the development of Cardiovascular and Metabolic disease including hyperglycemia, dyslipidemia, atheroslcerosis, and clinical outcomes of heart attack and stroke. We utilize a wide array of techniques in pharmacology, molecular biology, biochemistry, genetics in the cell culture and mouse model systems.

Donna Neumann, PhD dneum1@lsuhsc.edu

The goal of my research is to understand the molecular mechanisms regulating Herpes Simplex Virus reactivation, and how that reactivation leads to viral pathogenesis in the eye. We use novel approaches to study molecular reactivation in the host ganglia and then combine that with animal studies to understand disease mechanisms and disease prevention in the host, with the hopes of blocking reactivation and lessening ocular HSV disease.

Charles Nichols, PhD cnich1@lsuhsc.edu

For my research, I follow an integrated approach utilizing both mammalian and Drosophila systems to identify and elucidate molecular mechanisms linking serotonin receptor activation to behaviors. The underlying hypothesis of my research program is that molecular events initiated by serotonin receptor activation, such as aberrant gene expression, which contribute to behavioral effects in animal models, represent potential candidates for involvement in the development and etiology of schizophrenia and related disorders in humans, and importantly, targets for new avenues of discovery for therapeutics. I have shown that serotonin receptor activation has a dramatic and dynamic effect on gene expression within the prefrontal cortex of mammalian brain.

The cellular functions regulated by these genes include signal transduction molecules, transcription factors, and structural proteins. A common theme of many of these gene products is the process of synaptic plasticity. Furthermore, I have developed the fruit fly, Drosophila melanogaster, to serve as a powerful genetically tractable model system to study molecular events underlying serotonin receptor-mediated behaviors.

Quite remarkably, pharmacological activation of serotonin 5-HT2 receptors in the fly produces robust quantifiable behaviors not unlike those observed in mammalian systems. Future research projects in my laboratory will continue investigations into molecular mechanisms of serotonergic function that are relevant to human neuropsychiatric diseases utilizing both mammalian and Drosophila systems.

Dennis Paul, PhD dpaul@lsuhsc.edu

The focus of my laboratory is the pharmacology of pain and analgesia. Currently, we have three major projects. First, we

are examining the mechanisms by which drugs and neurotransmitters produce synergistic interactions. Our current focus is on the roles of lipid rafts and receptor dimers on subcellular interactions. Second, we are examining the role of sodium channels and pumps in the developing of diabetic neuropathy. Third, we are developing novel analgesic drug treatments that are as efficacious as morphine, but with reduced abuse liability. We use both in vivo and in vitro techniques and a collaborative approach to study all these questions.

Martin Ronis, PhD mronis@lsuhsc.edu

Dr. Ronis research involves determination of the molecular mechanisms underlying the toxic effects of alcohol, drugs and environmental pollutants on the liver and skeleton. He is funded by NIH to examine the role of oxidative stress and signaling via reactive oxygen species on progression of fatty liver disease from simple steatosis to hepatocellular carcinoma and on alcohol-induced osteopenia. As the previous Associate Director of the Arkansas Children's Nutrition Center, the influence of diet and dietary factors on development of toxic endpoints and potential therapeutic effects of dietary factors and supplements remains a research focus with an emphasis on therapeutic properties and health beneficial effects of vitamins, fruit polyphenols and peptides derived from processing of dietary protein sources.

Kurt Varner, PhD kvarne@lsuhsc.edu

Cardiovascular and cardiac effects elicited by the acute and chronic administration of stimulants and club drugs. Another project is designed to determine the nicotinic receptor subtypes that control cardiovascular function and mediate the cardiovascular responses elicited by nicotine.

Guangyu Wu, PhD gwu@lsuhsc.edu

G protein-coupled receptors (GPCRs) constitute a superfamily of cell surface receptors that regulate a variety of downstream effectors including adenylyl cyclases, phospholipases, protein kinases and ion channels. GPCR function is regulated by the efficient trafficking and positioning of specific receptors. Over the past decades, most studies on the trafficking of GPCRs have focused on the events involved in receptor internalization. However, the molecular mechanism underlying the export of GPCRs from the endoplasmic reticulum (ER) through the Golgi to the cell surface remains poorly understood. The overall objective of my research is to elucidate the molecular mechanism underlying the export of GPCRs form the ER to the cell surface, the regulation of GPCR function by intracellular trafficking and the pathophysiological significance of GPCR trafficking and signaling in the development of cardiovascular disease.

PHYSIOLOGY

Scott Edwards, PhD sedwa5@lsuhsc.edu

Research interests in our laboratory center around the investigation of neurobiological changes associated with altered motivational systems in drug and alcohol dependence. Our research strategy is to first determine significant alterations in neurobiological signaling following excessive drug or alcohol use, and then to investigate which neuroadaptations are most critically involved in driving excessive drug use over time. A closely associated goal is to understand signaling changes induced by re- exposure to drug- or stress-paired contexts and how these processes may contribute to relapse and other motivational disorders. Our most recent focus is on the interaction of addiction and chronic pain, with a focus on central brain systems that regulate both processes. Our hope is to translate these preclinical discoveries into new therapeutics for addiction and associated co-morbidities. These projects involve close collaboration with distinguished LSU Health and national investigators, and provide excellent opportunities for research training in the biomedical sciences.

Jason Gardner, PhD jgardn@lsuhsc.edu

The major research emphasis is focused on understanding the pathogenesis of heart failure. Of particular interest are the mechanisms responsible for the adverse cardiac extracellular matrix (ECM) remodeling associated with the progression of congestive heart failure. Current topics of study include:

- the role of lysyl oxidase, a collagen crosslinking enzyme, and related peptides in myocardial ECM remodeling,
- the cardioprotective effects of estrogenic pathways, including soybean- and plant-derived compounds, and
- the cardiac effects of inhaled particulate matter and cigarette smoke.

Our laboratory utilizes rodent models of cardiac disease, including models of pressure overload and chronic ventricular volume overload. We also use primary adult cell culture to examine specific pathways involved in the remodeling process.

Jeff Gidday, PhD jgidda@lsuhsc.edu

Research in Dr. Gidday's lab focuses on establishing the efficacy of adaptive epigenetics as a therapeutic strategy for treating retinal diseases, including glaucoma and different forms of retinitis pigmentosa, and the mechanisms underlying the protective responses. In addition to exploring ways to induce long-term changes in phenotype, the phenomenon of transgenerational epigenetic inheritance of adaptive traits is also under study. The lab employs mouse models for these studies, an assortment of physiologic, molecular biological, and imaging techniques, and with the help of local collaborators, bioinformatics-based analyses.

Nicholas Gilpin, PhD ngilpi@lsuhsc.edu

My lab examines the neurobiology of alcohol abuse, post-traumatic stress disorder (PTSD), and pain. More specifically, we are interested in understanding the neurobiology underlying the frequent co-morbidity between these conditions. To explore these questions, we use rodent models (e.g., vapor inhalation) of alcohol & drug dependence, oral and intravenous drug self-administration to model substance abuse, and predator odor models of traumatic stress. We combine intra-cranial and intravenous surgical techniques, site-specific pharmacology, and optogenetics with behavioral assays to establish causal relationships between neural signaling and behavior. We also use ex vivo analysis of brain tissue to measure levels and function of specific proteins that can be correlated with behavioral changes produced by alcohol, drugs, and stress.

Lisa M. Harrison-Bernard, PhD lharris@lsuhsc.edu

Diabetes is the 6th leading cause of death in the United States and diabetic nephropathy is the most common cause of end-stage renal disease. Many patients with diabetic renal disease require the use of dialysis to perform the functions of normal, healthy kidneys. Our research program focuses on how the effects of an important hormone system, renin angiotensin system, contributes to the failure of normal kidney blood vessel function and leads to the development and progression of kidney disease in type II diabetic patients. The model system that we use is the obese diabetic db/db mouse which exhibits features similar to human type II diabetic nephropathy. We utilize state-of-the-art techniques which combine in vitro and in vivo approaches. Measurements of molecular expression, enzymatic activity, peptide hormone levels, and quantitative histological examination of renal disease markers are coupled with conscious animal cardiovascular and renal physiological and pharmacological assessments. New therapeutic drug targets are being investigated to protect kidney function during diabetic kidney disease.

Patricia E. Molina, MD, PhD pmolin@lsuhsc.edu

Research in our laboratory focuses on the physiopathological consequences of alcohol and drugs of abuse and their interactions with disease states, including traumatic brain injury (TBI) and HIV/SIV infection. Our scientific team actively collaborates with basic science, clinical, and translational investigators in a wide range of projects that investigate how alcohol and drugs of abuse interact at the molecular, organ system, whole body, and community levels to contribute to disease burden in our society.

Barry J. Potter, PhD bpotte@lsuhsc.edu

My research involves the investigation of free radical generation and nitric oxide production under conditions of oxidative stress in model systems. Current research is focusing on (a) the use of cell lines to mimic the liver and to investigate the effects of alcohol and infection following insults such as alcohol and/or infection, and (b) the estimation of anti-oxidant status using red blood cells from a variety of stressed animal models.

The major technique used in the laboratory for these determinations is electron paramagnetic resonance (epr or esr), which enables the ready detection and quantitation of specific free radicals and reactive oxygen species. Future research will involve purifying relevant sub-cellular components prior to investigating free radical generation and/or nitric oxide synthesis.

Stefany Primeaux, PhD sprime@lsuhsc.edu

The current research in my laboratory focuses on understanding peripheral and central mechanisms leading to obesity and related comorbidities. There are several projects in my laboratory investigating neural, behavioral and physiological factors affecting the susceptibility to developing obesity. These studies include the assessment of fat sensing via the oral cavity in obesity-prone and obesity-resistant rats and the assessment of inflammatory markers on the risk for cardiovascular disease in obesity-prone and resistant rats. We are also interested in the role of the hypothalamic neuropeptide, QRFP, on feeding and other motivated behaviors in male and female rats.

Robert Siggins, PhD rsiggi@lsuhsc.edu

The main focus of our laboratory is to uncover the role of alcohol in disruption of osteoclast-dendritic cell (ODC) progenitor differentiation. Our preliminary studies have shown that alcohol upregulates RANK expression and osteoclast precursor numbers in the bone marrow of chronic alcohol-administered rhesus macaques. Additionally, we have shown that chronic alcohol administration in this model decreases the bone marrow myeloid dendritic cell numbers. We have identified NOTCH signaling at the nexus of ODC lineage specification; alcohol-induced NOTCH inhibition leads to enhanced osteoclastogenesis and decreased dendropoiesis. Additional studies in the lab are focused on the alcohol-induced impairment of endothelial progenitor cells to contribute to wound healing.

Liz Simon, PhD lsimo2@lsuhsc.edu

Research interests in our laboratory focus on the mechanisms of chronic alcohol-induced impairment of skeletal muscle regenerative capacity. Using whole tissue and cell cultures together with molecular techniques, we are studying chronic alcohol-induced epigenetic modifications of myogenic gene transcription. Our current focus is to elucidate microRNA alterations and histone modifications that potentially contribute to impaired regeneration. Our ultimate goal is for integration of basic science and translational research, setting the stage for potential therapeutic or lifestyle interventions for reversing adverse consequences on skeletal muscle function seen with heavy alcohol consumption.

Flavia Souza-Smith, PhD fsouz1@lsuhsc.edu

My research focuses on the effects of alcohol on lymphatic function and the crosstalk between lymphatic vessels and perilymphatic adipose tissue (PLAT). In the US, approximately 25% of individuals over the age of 12 binge drink alcohol at least once a month. Alcohol binge drinking increases the risk of metabolic syndrome. Specifically, chronic and binge alcohol drinking patterns result in gut bacterial toxin translocation, induction of insulin resistance, and impairment of insulin-dependent responses in adipose tissue. Our approach is to investigate the effects of repeated binge-like alcohol intoxication-induced lymphatic bacteria and LPS dissemination, lymphatic vessel hyperpermeability, and lymphatic leakage of bacteria and LPS into PLAT, which promotes a PLAT inflammatory milieu and impairment of adipokine profiles. We predict that these events play a role in the development of alcohol-induced insulin resistance. The goal of these studies is to understand the mechanisms involved in alcohol-mediated disruption of lymphatic function and its impact on local and systemic metabolic regulation.

Xinping Yue, PhD xyue@lsuhsc.edu

Research in my laboratory is focused on understanding the pathogenesis and molecular mechanisms of pulmonary fibrosis. Of particular interest are regulations of transforming growth factor signaling by heparan sulfate. Our laboratory utilizes genetically modified mouse models as well as primary cell cultures to examine specific signaling pathways involved in the fibrotic process. Our goal is to develop novel therapies targeting heparan sulfate in the treatment of pulmonary fibrosis and possibly fibrotic diseases in other organ systems.

DENTISTRY & BIOMATERIALS

Xiaoming Xu, PhD xxu@lsuhsc.edu

The general area of my research is the development and evaluation of new dental materials and biomaterials. Current I have the following on-going projects: (1) Development of novel fluoride-releasing dental composites. This is a six-year project funded by the Brown Foundation. (2) Development of novel fluoride-releasing adhesive monomers and dental adhesives. This is a five-year project funded by NIH, starting from September 2004. (3) Ceramic nanofibers and nanofiber-reinforced dental composites. (4) Fabrication of hydrogel nanofibers and their applications as controlled-releasing and wound-healing materials.

My other research interests include: biocompatibility and tissue reaction of dental materials/biomaterials, biosensors based on nanoparticles and nanofibers, and mass spectrometric study of metal-peptides/protein interactions.

NEUROLOGY

Harry Gould, PhD hgould@lsuhsc.edu

My research involves undestanding the role of sodium channel modulation in models of pain.