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.

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<td>Suresh K. Alahari, PhD</td>
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<td>Shyamal Desai</td>
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<td>Ranney Mize, PhD</td>
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<td>Ya-Ping Tang, MD, PhD</td>
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<td>Matthew Whim, PhD</td>
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**GENETICS**

| Judy Crabtree, PhD | X              | X                     | X                   | —       |
| Ed Grabczyk, PhD  | X              | —                     | X                   | —       |
| Paula Gregory, PhD | —              | X                     | X                   | —       |

| Andrew Hollenbach, PhD | X              | X                     | X                   | —       |
| Wanguo Liu, PhD        | X              | X                     | —                   | —       |
| Diptasri Mandal, PhD   | X              | X                     | —                   | —       |
| Udai Pandey, PhD       | X              | X                     | X                   | X       |
| Fern Tsien, PhD        | —              | —                     | X                   | X       |

**MICROBIOLOGY, IMMUNOLOGY AND PARASITOLOGY**

| Angela Martin Amedee, PhD | X              | X                     | X                   | X       |
| Ashok Aiyar, PhD          | X              | X                     | X                   | —       |
| Yan Cui, PhD              | X              | —                     | —                   | —       |
| Paul Fidel, PhD           | X              | X                     | —                   | —       |
| Timothy Foster, PhD       | X              | X                     | X                   | X       |
| Michael Hagensee, MD, PhD | X              | X                     | —                   | —       |
| Doug Johnston, PhD        | X              | X                     | X                   | X       |
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**NEUROSCIENCE**

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**OTHER DEPARTMENTS**

**DENTISTRY AND BIOMATERIALS**

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**OBGYN**

**OPHTHALMOLOGY**

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**PEDIATRICS**

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<td>Mike Ferris, PhD</td>
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**Surgery**
BIOCHEMISTRY AND MOLECULAR BIOLOGY

Suresh K. Alahari, PhD
salaha@lsuhsc.edu
01/07

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 tumor progression. 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.

William C. Claycomb, PhD
wclayc@lsuhsc.edu

The major interest of this laboratory is to develop mechanisms to repair or regenerate heart muscle tissue in the diseased heart. We are taking two major approaches. One is to understand the molecular machinery in which Nature restricts and irreversibly inhibits heart cell division during early development so that we can design procedures to reinitiate cell division to essentially regenerate muscle tissue.

The other approach is to actually transplant heart muscle cells back into the diseased heart to repair or replace the injured muscle cells. Presently we are using mouse embryonic stem cells (ES Cells) to derive immortalized ventricular, atrial and conducting heart cell lines that retain a cardiac lineage-specific phenotype. This involves using a combinatorial selection process with multiple promoters and selectable markers. The idea is to create three different types of heart muscle cells that could be grown continuously in culture and be used as model systems to study the function and dysfunction of the ventricle, atria and conduction system of the heart. We have recently been successful in utilizing specific molecular markers to identify a primitive cardiac conduction system utilizing differentiating ES cells in embryoid bodies.

We are also utilizing these genetically engineered ES cells in a tissue-engineering project. These cells would be extremely useful as research reagents for basic cardiac muscle cell research such as growth regulation and signal transduction as well as for therapeutic applications such as transplantation of cells into a diseased heart to repair damaged heart muscle and as a cell replacement therapy to treat a wide variety of cardiomyopathies.

Shyamal Desai, PhD
Sdesai@lsuhsc.edu
12/07

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, Alzheimer's disease, Liddle's syndrome (familial hypertension), tumorogenesis, 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  03/08

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  01/07

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.

Carmen Canavier, PhD  ccanav@lsuhsc.edu  1/07

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 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  10/09
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  11/09
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.

Ranney Mize, PhD    rrmize@lsuhsc.edu
I study the development of synapses and axonal pathways in the brain. I am interested in the various molecules which control axon growth and the formation of synapses between neurons. I use the visual system to study this process, specifically the pathway from the retina (eye) to the superior colliculus, a subcortical structure involved in eye movements. I use several techniques, including light and electron microscope immunocytochemistry in which we localize various molecules (receptors, transmitters, growth factors) using antibodies directed against them. We also examine individual synapses with the electron microscope. We do quantitative analysis using digital microscopy and image analysis. Studies are performed in normal, transgenic, and gene knockout mice in order to determine if down-regulating genes which produce various substances alters the development of the pathway.

Ya-Ping Tang, MD, PhD    ytang1@lsuhsc.edu  1/09
The research program in this lab consists of two main topics: neurobiology of learning and memory and neurobiology of neurodegeneration. The long-term goal of this program is to understand the roles of genes in learning and memory as well as the role of gene mutations in the pathogenesis of certain brain diseases with memory deficits such as Alzheimer's disease. A significant advantage in this lab is the combination of multiple disciplinary approaches including molecular biology, histology/morphology, mouse genetics, electrophysiological (collaborating with our colleagues), and mouse behavioral approaches in our studies. Moreover, the use of conditional genetic approaches in this lab allows a time-dependent and/or neuronal cell types/brain region-specific analysis of gene function at the molecular, neuronal, morphological, and behavioral levels. Two representative projects are (1) how a synaptic stability contributes to memory consolidation and (2) how epigenetic events control aging-dependent neurodegeneration. More than 10 genetically engendered mouse models are currently available in this lab.
### 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.
3. 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.

### GENETICS

### Judy Crabtree, PhD  
*jcrabt@lsuhsc.edu*

Research Interest include:

- Understanding biological processes & epigenetics of endocrine tumor disorders.
- Candidate gene transcriptional regulation via methylation in tumors from both human and the Eker rat, and the functional consequences of this epigenetic regulation in uterine fibroid pathogenesis.
- Role of risk factors such as race and obesity in uterine fibroid etiology.
- The role of progesterone and other hormones in the epigenetic regulation of menin expression and function in Multiple Endocrine Neoplasia Type1 (MEN1).

### Ed Grabczyk, PhD  
*egabc@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
Andrew Hollenbach, PhD  
aholle@lsuhsc.edu  
11/09
Research interest include:
- The regulation of transcription factors through phosphorylation
- Biochemical mechanisms of chromosomal translocation gene products in cancer formation

Wanguo Liu, PhD  
wliu3@lsuhsc.edu  
11/09
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  
11/09
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

Udai Pandey, PhD  
Upande@lsuhsc.edu  
11/09
Research interest include:
- Utilizing Drosophila melanogaster as a model system (fruit fly) to apply the power of genetics to understand the pathogenesis of neurodegeneration
- Investigating the role of protein degradation pathways in neurodegeneration

Fem Tsien, PhD  
fmille@lsuhsc.edu  
10/09
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.

Guoshun Wang, DVM, PhD  
gwang@lsuhsc.edu  
11/09
- Phagocytic Innate Immunity
- Cystic Fibrosis
- Gene Therapy and Stem Cells

MICROBIOLOGY, IMMUNOLOGY AND PARASITOLOGY

Ashok Aiyar, PhD  
aaiyar@lsuhsc.edu  
12/07
My research program focuses on two aspects:
1) Understanding the key mechanisms involved in melanoma initiation. In particular, using in vitro and in vivo skin models, we seek to learn how dysfunction of the components of the Retinoblastoma (Rb) pathway deregulates cell death mechanisms and activates cell survival signaling leading to the onset of skin tumours
2) Elucidating the downstream signaling mechanisms of the cell adhesion molecules CD44 and CD146 in cancer metastasis using “gene switch” systems both in vitro and in vivo
Angela Martin Amedee, PhD  
aamede@lsuhsc.edu

Pathogenesis of HIV and mother-to-infant transmission of human and simian immunodeficiency viruses (HIV and SIV). The major focus of research in the Amedee laboratory is to evaluate the parameters involved in transmission of virus from mother-to-infant. Other areas of interest include the identification and quantitation of viral genotypes expressed at mucosal surfaces (vaginal and oral), and characterization of HIV/SIV envelope sequences that emerge during disease progression.

Yan Cui, PhD  
ycui@lsuhsc.edu  
11/09

Research interest include:
- Cancer immunotherapy
- T cell development and lymphomagenesis
- Gene Therapy

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) 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  
10/09

A host’s control of viral infections is mediated through cellular sentinels that detect pathogen invasion and initiate both innate and adaptive responses. Herpesviruses establish a lifelong chronic and persistent infection, and as such must continuously evade host antiviral responses. Despite effective HSV antivirals, two disconcerting trends have been observed: 1) the prevalence of genital herpes has continued to rise with approximately 1.6 million new cases occurring annually; 2) there has been an increased incidence of recurrent genital lesions. These trends are especially alarming considering the role HSV-2 plays in facilitating acquisition of other STIs, including HIV. HSV-2 infections contribute to increased STIs by either modulating cell-intrinsic innate recognition and response pathways or increasing the inflammatory milieu within the genital tract mucosa. Our laboratory’s overarching research interest is to understand the mechanisms by which HSV-2 viral proteins subvert host cell-intrinsic antiviral responses and thereby contribute to HSV-2 pathogenesis. Our laboratory has utilized various molecular techniques to identify multiple viral proteins involved in subverting numerous host antiviral pathways. Understanding the virus/host interplay and the basic mechanisms employed to evade host cell-intrinsic pathogen recognition and response pathways provides new insights and targets for novel vaccine and/or therapeutic development.

Michael Hagensee, PhD  
mhagen@lsuhsc.edu  
10/09

My lab works on the human papillomavirus (HPV) and how it causes cervical cancer. Many men and women are infected with this virus but very few go on to have cancer. We are looking at ways to detect this virus since it does not grow readily in the laboratory. We are examining the role of co-factors that may explain those who progress to cancer from infection. In addition, we are examining how our body reacts and tries to control this virus in order to potentially improve therapeutic options.

Doug Johnston, PhD  
djoh13@lsuhsc.edu  
10/12

The Johnston Lab is focused on vascular responses to the damage induced by bloodborne fungal pathogens; particularly damage induced by Candida albicans and related species. We are interested in characterizing the fungal-induced changes in host cell morphology, gene expression, and defensive/repair mechanisms. Mortality rates associated with disseminated fungal infections reach 40-50%, even with aggressive antifungal therapies, a sobering statistic considering their high costs and toxicity, and further complicated by the recent emergence of several inherently resistant species.
Upon gaining access to the bloodstream, Candida spp rapidly adhere to vessel walls and actively penetrate the endothelium and underlying basement membrane, causing significant damage to vessel integrity and loss of barrier function. These events ultimately lead to symptoms of septic shock, including organ dysfunction and failure. Efficient vessel repair responses require the rapid activation of neighboring cells within the blood vessel and surrounding stroma. Our data suggest that upon C. albicans invasion, several key components of the vascular repair process are significantly dysregulated and neighboring (undamaged) endothelial cells become senescent or undergo apoptosis, leaving the extracellular matrix exposed, and perhaps allowing for an extended window of opportunity for disseminated infection. Therefore, the goal of our research is the identification of mechanisms that can be exploited to enhance the vascular resistance to fungal damage, to limit fungal dissemination, and to ultimately alter the outcome of infection.

**Ben Kelly, PhD**

In my laboratory we study the protozoan parasite Leishmania, the causative agent of leishmaniasis. Leishmaniasis is an infectious disease that impacts approximately 12 million people worldwide, predominantly in tropical and subtropical regions. This sandfly-borne disease is caused by infection of mammalian host phagocytes with Leishmania. Clinical symptoms range from self-healing skin lesions to lethally disseminating visceral disease. Current treatment options against leishmaniasis are largely inadequate; therefore there is an urgent need to develop better drugs against Leishmania. Our major research goal is to understand parasite molecular mechanisms that are essential for parasite viability and virulence in leishmaniasis. With this goal in mind, we are currently investigating the function of “LACK”, a molecular scaffold protein, in control of Leishmania protein synthesis and virulence. Advancing our understanding of these mechanisms will allow us to identify new parasite molecular targets for the development of better anti-leishmanial drugs.

**Chris L. McGowin, PhD**

Sexually transmitted infections are an important public health and economic concern worldwide, particularly as related to women’s health. Currently, we are working to define the mechanisms of reproductive tract disease caused by the emerging sexually transmitted pathogen, *Mycoplasma genitalium*. We have approached this goal from several perspectives: 1) Using next-generation sequencing platforms, we are sequencing the complete genomes of phenotypically variable *M. genitalium* strains found worldwide to identify functional determinants of mucosal infection; 2) We utilize novel 3-dimensional models of the human reproductive tract to investigate acute and persistent host-pathogen interactions; 3) In clinical investigations, we are evaluating directly whether *M. genitalium* infection elicits cervical inflammation and whether this inflammation is linked to HIV disease progression. A second arm of the laboratory is dedicated to in vitro diagnostic (IVD) testing collaborations with industry partners. Our current focus is commercial validation for automated, high-throughput tests for Human Papilloma Virus, *Chlamydia trachomatis*/*Neisseria gonorrhoeae* and *M. genitalium*.

**Mairi Noverr, PhD**

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.

**Glen Palmer, PhD**

Dr. Palmer’s research addresses the mechanisms by which pathogenic fungi adapt to the mammalian host and differentiate during colonization and infection. In particular, the research focuses on the opportunistic pathogen *Candida*...
Current studies are investigating the role played by the fungal vacuole in mediating yeast-hypha differentiation, survival within host cells such as macrophages, and invasion of host tissues.

**Francesca Peruzzi, PhD**  
frperuz@lsuhsc.edu  
10/12

My laboratory is interested in identifying molecular mechanisms of HIV-mediated neuronal damage, with a particular focus on the role of non-coding RNAs. In another project we study signaling pathways and the role of microRNAs in Glioblastoma cells proliferation and migration.

**Joy Sturtevant, PhD**  
JSturt@lsuhsc.edu  
10/12

My scientific interest has always been initial interactions between pathogen and host. Current research interests focus on *Candida albicans* ability to survive and adapt to new environmental challenges. Currently, I have initiated a new project focusing on ‘hypoxic recovery’. The rationale is that a significant portion of disseminated fungal infections appear to arise from endogenous sources in which the fungus is symptomatically dormant (latent state). Fungal pathogens appear to be in environments of low oxygen while the organism is dormant which includes biofilms, granulomas or colonization of tissue. Thus it may be the fungus’s exit from dormancy which initiates fulminating infection. In most instances, this correlates with a readaptation to increased oxygen levels. I am hypothesizing that exit from ‘latency’ could be exploited to identify biomarkers which could be translated into diagnosis tools and/or drug and vaccine targets.

Areas of study in this project are 1) identification of a signature hypoxic recovery response by using global transcriptomics (deep sequencing) and proteomics; 2) identification of hypoxic recovery inhibitors by screening compound libraries; 3) assessing virulence traits (adherence, phagocytosis, induction of cytokines); and 4) validation of hypoxic recovery in vivo using animal models. Studies will be done using the opportunistic fungal pathogens *Candida albicans* and *Aspergillus fumigatus*.

**Guoshun Wang, DVM, PhD**  
gwang@lsuhsc.edu  
11/09

- Phagocytic Innate Immunity
- Cystic Fibrosis
- Gene Therapy and Stem Cells

**Z. Tom Wen, PhD**  
zwen@lsuhsc.edu  
10/09

Research Interest:

Research in my laboratory primarily focuses on molecular characterization of bacterial biofilms. In nature, bacteria exist in highly complex multiple-species communities known as biofilms. Due to the increased resistance to host defense and antimicrobial therapy, biofilms are notoriously difficult to eliminate and are a source of many recalcitrant infections. A better understanding of the mechanism underlying biofilm formation and persistence should ultimately lead to the development of novel and effective therapeutic and preventive strategies against diseases and conditions in which biofilm formation plays a prominent role.

Currently, *Streptococcus mutans*, an opportunistic bacterium primarily living in tenacious biofilms on the tooth surface, serves as the model organism. Major effort is directed, but not limited to (i) intra- and inter-species communication and its impact on establishment, persistence and competitiveness of *S. mutans* on the tooth surface using continuous flowing, mixed-species models and confocal laser scanning microscopy; (ii) identification of genes required for biofilm formation by *S. mutans* using functional genomics approach. A major project is on further characterization of BrpA, a surface-associated protein with a major role in stress response and biofilm formation by *S. mutans*, focusing on structure-function relationship, mechanism underlying BrpA-mediated regulations, and potential for targeting BrpA in anti-caries strategy. In collaborative effort, we are also working on development of novel antibacterial dental composites and identification of small molecules that target intercellular signaling as novel approach in strategy combating dental caries.
Arnold H. Zea, PhD  azea@lsuhsc.edu  01/07

The research focus of this laboratory is to study T cell dysfunction in patients with renal cell carcinoma and tuberculosis. Until now, immune dysfunction in cancer and mycobacterial diseases has primarily been attributed to the loss of DTH responses to multiple tumor antigens or mycobacterial preparations. However, we believe that alterations in T cell signal transduction proteins may play an important role in T cell immune dysfunction. The characterization of these alterations may be used as markers to determine immune dysfunction.

Furthermore, we are interested in studying immunological and molecular mechanisms involved in T cell dysfunction, aiming to prevent or restore an adequate immune response that will benefit the clinical outcome of patients with renal cell carcinoma or tuberculosis. Understanding the mechanism(s) that lead to T cell dysfunction in those patients will allow us to develop new ways of improving the efficacy of the different clinical immunotherapy approaches currently in use. Our research is focus in three aspects: 1) Determine in both patients and cell lines the role of enzyme arginase II in the depletion of L-arginine associated with T cell dysfunction, 2) we are testing the role of arginase in tumor cell growth and mycobacterial growth-induced immunosuppression, 3) study of the immunological and molecular mechanisms of arginase induction in disease.

NEUROSCIENCE

Haydee E. P. Bazan, PhD  hbazan1@lsuhsc.edu  10/09

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  01/07

Our research has lead 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  8/06

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  
1/07

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 coordinately 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  
01/07

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  
03/13

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](http://www.medschool.lsuhsc.edu/neuroscience/faculty_detail.aspx?name=hong_song)

Minghao Jin, VMD, PhD  
mjin@lsuhsc.edu  
10/09

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  
01/07
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.

Alberto Musto, MD, PhD  amusto@lsuhsc.edu  10/09

Dr. Alberto E. Musto’s laboratory is focusing in the basic mechanism/s that mediates the genesis of seizures (ictiogenesis) and the occurrence of spontaneous seizures (epileptogenesis). The central hypothesis is that failure of inhibitory neurotransmission mediated by GABA_A receptors lead to the initiation and propagation of seizures. Several repetitive seizures enhance the accumulation of platelet activating factor (PAF) that activates molecular pathways, which triggers inflammatory and degenerative process in the brain and impair the central inhibition. Dr. Musto tests the mentioned hypothesis integrating his background of clinical neurology and neuroradiology into in vivo animal models of experimental epilepsy, using behavioral procedures, state of the art of electrophysiology in vivo, immunohistology techniques, biochemical protocols and novel chemical compounds (LAU-0901). Dr. Musto’s laboratory hypothesizes that modulation of PAF activity through the PAF-antagonist receptor; LAU-0901, the neuroinflammation, neuronal damage and recurrent epileptic seizures will be attenuated. Dr. Musto collaborates with Dr. Nicolas G. Bazan in studying neuroprotective signaling in experimental epilepsy.

Christian T. Sheline, PhD  chris@lsuhsc.edu  11/09

My lab studies the death induced by excess zinc which occurs after injuries and pathophysiologic conditions in neurons and β-cells. In these cells zinc accumulates resulting in ATP and NAD+ level depletion causing glycolytic inhibition. Measures which restore the NAD+ levels, such as exogenous addition of NAD+, nicotinamide, or pyruvate prevent zinc-induced death. We and others have demonstrated the therapeutic value of nicotinamide, pyruvate, or Zn2+ chelation against neuronal death caused by light-induced retinal damage, retinal, focal, and global ischamias, hypoglycemia, seizures, head trauma, and visual cortex ablation. This research was funded by an R01 grant from NINDS, and similar grant applications to investigate the role these pathways play in visual cortex ablation, and light-induced damage are pending at NINDS and NEI.

Zn2+ toxicity also plays a role in the secondary death of β-cells in pancreatic islets of diabetic mice. Nicotinamide, Zn2+ chelation, and pyruvate attenuated β-cell death and diabetic incidence in mouse models of diabetes. These studies are funded by an R01 from NIDDK. Zn2+ dyshomeostasis through the β-cell specific Zn2+ transporter, ZnT8, has been implicated in type-1 and type-2 diabetes in humans. We have made founder transgenic mice which overexpress human ZnT8 WT and mutant forms in β-cells, and are currently characterizing these founders. An R01 grant application to fund these studies is pending with NIDDK.

Hugh Xia, PhD  hxia@lsuhsc.edu  8/06

Synapse has been shown to undergo persistent modifications in response to different patterns of activity and this change has been hypothesized to underlie the experience-dependent modifications in our brain, including learning and memory. We are interested in how NMDA receptors function in inducing these many forms of synaptic plasticity. We are not only interested in short term modifications in the synaptic protein composition through calcium mediated signaling pathway, but also CREB mediated gene transcription which provides new proteins for long term modification of the synapse. We are in interested in both kinase and phosphatase mechanism of the signaling pathways leading to these changes in the synaptic strength. We use primary hippocampal cultures and hippocampal slices as our model systems. Techniques used include electrophysiological recordings of synaptic transmission, molecular biology for manipulating genes involved in the signaling pathway from NMDA receptor activation to synaptic strength medication and confocal/two-photon microscopy for localization studies of key proteins in these pathways.

PATHOLOGY

Richard S. Vander Heide, MD, PhD  rvande3@lsuhsc.edu  10/09

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  
wbmacke@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.

Allison Berrier, PhD  
aberri@lsuhsc.edu

The aim of research in the Berrier lab is to understand the cellular machinery that promotes oral tumor metastasis. We study the role of cell-matrix interactions in oral cancer because proteins in matrix adhesions are known to perform important functions in tumor progression, proliferation and invasion. Integrins are the principle cell surface receptors that mediate cell-matrix adhesion and disruption of the function of certain integrin receptors can reduce oral cancer tumor metastasis. One project area in the lab focuses on determining whether certain integrin receptors on invasive oral cancer cells recruit particular cytoplasmic proteins in order to regulate the invasive phenotype. A second project area involves determining whether a cell-derived matrix can be bio-engineered to change the behavior of invasive oral cancer cells. Both projects will potentially provide future approaches to reduce the invasive nature of oral tumors.

Hamid Boulares, PhD  
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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-ribose)lation 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
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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.

Stephania A. Cormier, PhD
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12/07

The main goal of my laboratory is to determine if exposure during early neonatal life to environmental factors (i.e. allergens, pollutants, and respiratory viruses) leads to predisposition, development of, or exacerbation of respiratory disease in the adult. Our central hypothesis is that allergic respiratory diseases result, in part, from environmental impact(s) that occur during a critical phase of immuno-maturation. In the short term, we are exploring the validity of this hypothesis by defining the cellular and molecular and pathophysiologic changes in the pulmonary microenvironment following gestational and/or neonatal exposure to allergens; urban pollutants (e.g. particulates such as diesel exhaust particles); and respiratory viral infection. Our present data clearly demonstrate that early exposure to respiratory viral infections, such as respiratory syncytial virus, predisposes to adult respiratory disease and alters subsequent immune responses in the lung. Current work in the lab seeks to determine the mechanisms involved in these processes.

Daniel R. Kapusta, PhD
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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
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01/07

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.
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.

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.

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.

G protein-coupled receptors (GPCRs) constitute a superfamily of cell surface receptors that regulate a variety of downstream effectors including adenyl 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

Gregory J. Bagby, PhD  gbagby@lsuhsc.edu  1/07

My research is a collaborative effort with Steve Nelson, MD, and Ping Zhang, MD, PhD in the Section of Pulmonary/Critical Care Medicine of the Department of Medicine. We explore host defense to systemic and pulmonary bacterial and viral infections in immunocompromised states such as alcohol intoxication, which is well known to increase the incidence and severity of infection, especially of the lung. Studies are performed in rodents and nonhuman primates. We use sophisticated analytical procedures to include molecular biology, multiplex protein analysis, and flow cytometry to identify cells and their individual functional state as well as isolated perfused tissues, and cell culture. In our studies we study immune cell migration and how mediators called cytokines orchestrate host defense against pathogens. As we discover impairments in the host defense system, we attempt to discover novel strategies to return host defense to normal. These include immune augmentation through the use of in vivo recombinant protein and gene delivery.

Jason Gardner, PhD  jgardn@lsuhsc.edu  10/09

*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.

Lisa M. Harrison-Bernard, PhD  lharris@lsuhsc.edu  10/09

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. In addition, direct measurements of renal resistance vessel diameter are accomplished using the mouse in vitro blood perfused juxtamedullary nephrin technique in kidneys obtained from control and diabetic mice. Most important, blocking the actions of a new, kidney specific enzyme, chymase, may provide a new therapeutic drug target to reduce the influence of this hormone system on diabetic kidney disease.

Patricia E. Molina, MD, PhD  pmolin@lsuhsc.edu  12/07

Research in my laboratory is focused on neural control of traumatic injury-induced hemodynamic and host defense responses and how these are affected by acute alcohol intoxication. In addition, studies are also conducted on the mechanisms involved in the immune, behavioral and metabolic alterations associated with progression to AIDS and how drugs of abuse (alcohol and cannabionoids) interact with these systems affecting morbidity and progression of disease.

Barry J. Potter, PhD  bpotte@lsuhsc.edu  8/06

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.

Xinping Yue, PhD  
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10/12

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 AND BIOMATERIALS

Xiaoming Xu, PhD  
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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  
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My research involves understanding the role of sodium channel modulation in models of pain.

OPHTHALMOLOGY

James Hill, PhD  
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12/07

Our laboratory, using animal models that mimic human pathogenesis of viral keratitis, has identified unique agents that can induce or block viral reactivation. We have perfected animal models of HSV-1 reactivation and recurrent disease so that we can assess a whole spectrum of HSV-1 ocular pathogenesis. Current experiments are focused on understanding the molecular basis of HSV latency and reactivation to develop therapeutic strategies that will not only block viral replication but prevent neuronal reactivation, the primary source of the reactivated virus.

Studies on the molecular biology of HSV include recombinant technology to investigate various genomic factors within the virus that are responsible for reactivation and recurrent ocular disease. We also have studies assessing the host changes during latency and reactivation using microarray analysis to identify changes in gene expression. These findings will allow us to identify pathways and specific proteins involved in neuronal viral reactivation and to develop therapeutic strategies to block recurrent ocular disease. We are also using chromatin immuno-precipitation assays to analyze the viral genome and the histone interactions that control permissive or non-permissive transcription. These are critical for understanding the basic mechanisms of the structure and function of chromatin during HSV latency and reactivation. We have constructed recombinant viruses that have significantly different phenotypic reactivation profiles (i.e. very high or very
We have also constructed recombinant herpes viruses that contain an insert designated to enhance green fluorescent protein, which allows visualization of corneal lesions in infected eyes and tracking of viral progression in the neural system. All of these studies have the ultimate goal of preventing recurrent herpetic infection of the eye. We have recently discovered that a human gene, apolipoprotein allele E4 (apoE e4), involved in susceptibility to ocular herpes infections. These studies involve assessment of neural tissues with the risk association of apoE alleles (E3 vs. E4), the HSV-1 genome copy, the neuroinvasive score, and Alzheimer’s disease (AD). ApoE e4 is a known risk factor for AD. Using knock-in mice that are transgenic for either of the human apoE alleles, E3 or E4, we are investigating ocular HSV-1 pathogenesis focusing on recurrent stromal keratitis, the control of the development of viral latency, and reactivation. Our research engages tools of molecular biology, pharmacology, biochemistry, neurovirology, and experimental models of infectious disease.

We combine all of these tools into a practical paradigm that allows the development of new and novel chemotherapeutic agents. Graduate students have the opportunity to utilize the past and ongoing investigations to build on their graduate degree. We are currently defining the translational implication of all of these findings through the development of novel strategies for therapy of HSV stromal keratitis, HSV encephalitis, and AD.

For more details see http://www.lsu-eye.lsuhsc.edu.

Jean T. Jacob, PhD  jjacob@lsuhsc.edu

My research focuses on the development and biocompatibility analysis of ocular implants and drug delivery systems. Current projects include the efficacy of artificial tear formulations, contact lens permeabilities, and manipulation of the epithelial cell response to nanopatterned surfaces. Students would be involved with in vivo and in vitro test methods.

Hilary W. Thompson, PhD  hthomp2@lsuhsc.edu

Statistical research on medical decision analysis from electronic medical record databases. This research includes gathering data from patient charts, database analysis and programming, and image analysis methods for automated feature detection of pathological features in retinal images.

Useful skill sets for students would be computer programming, mathematics, and statistics.

PEDIATRICS

Mary Breslin, PhD  mbreslin@chnola-research.org

My work focuses on a transcriptional factor, IA-1 that is expressed during early embryonic development and is silenced by birth. IA-1 is not found in normal adult tissue, however, it is re-expressed in tumors of neuroendocrine origin such as retinoblastoma, medullablastoma, insulinoma, and small cell lung carcinoma. Lung cancer is the leading cause of cancer death in the US. My work focuses on the role of IA-1 at the transcriptional level in SCLC. Due to the limited expression pattern of IA-1, we speculate that it may play an important role in the maintenance of certain tumor phenotypes. SCLC cells express markers of neural differentiation.

To date, we have determined the sequence specific DNA binding site for IA-1 and have identified at least two potential target genes for its regulation. One of these genes, NeuroD is critical for pancreas and neuronal cell development. We have initiated a yeast two hybrid protein-protein interaction screen to determine proteins that interact with IA-1. A second aspect of the project is to do proteomics analysis to identify new IA-1 target genes in SCLC.

Mike Ferris, PhD  mferris@chnola-research.org

10/09

The human body contains ten times more bacterial cells than human cells, and it is well accepted that these microbial populations play a significant role in both the normal immunity and inflammation. However, while traditional microbiology has focused on the study of cultivated pathogens, researchers using cultivation-independent techniques have shown that only a small percentage of bacterial species in the human body have been studied. Research in our lab makes use of next generation sequencing technologies, bioinformatics and statistics to describe the composition of bacterial communities in the human body.

Studies focus on exploring the association between alterations in bacterial populations and human diseases, for example...
those associated with the human gut and vaginal epithelium. Other basic research projects focus on environmental health associated with fungal growth in flooded/damp buildings and mycolactone producing Mycobacteria in warm water habitats in South Louisiana.

Michael Lan, PhD  
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12/07

My research interests are focused on the gene regulation of pancreatic beta cells. The current project entitled “Transcription factors in neuroendocrine differentiation” has progressed into two additional area of research. One is developed into a basic research of insulin gene regulation. This project will use animal model to study the molecular mechanisms of transcription factor, INSM1, in regulating insulin gene expression during the development of pancreatic beta cells. The other project was recently initiated using neuroendocrine tumor-specific promoter to direct suicide gene for cancer gene therapy. Initially, we will use neuroendocrine cell lines in nude mice animal model to test the efficacy of our construct. It is expected that the INSM1 promoter could direct specific expression of suicide gene in neuroendocrine tumors. The third project is insulin gene therapy for diabetes. This project is ongoing in my laboratory using chimeric promoter to direct human insulin gene expression in liver cells. The regulated insulin gene expression in liver is the key of this study. Currently, we are constructing various chimeric promoters and test their potency and specificity to direct insulin gene expression in liver cells.

Seth Pincus, MD  
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I study the interaction of antibodies with infectious agents. We are interested in how microbes evade the antibody response, but also in developing therapeutic and preventative approaches based on antibodies. In my lab we study HIV, group B streptococci, as well as toxins.

Surgery

Eugene Woltering, MD  
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The development of new blood vessel growth—what are the genetic and environmental influences that trigger these vessels to start growing? What can we do to stop these vessels from growing in cancer patients?