Dalton Cardiovascular Research Center

2015

Front picture is an angled shot of Dalton Cardiovascular Research Center.

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The Dalton Cardiovascular Research Center (DCRC) supports the objectives of the University of Missouri in its mission of teaching, research and service. Yet it is unique in its commitment to interdisciplinary collaborative research and teaching among various colleges, schools and departments across the Columbia campus. Under the auspices of DCRC, scientists from the fields of biochemistry, biological engineering, biological sciences, biomedical sciences, electrical engineering, medicine, pharmacology, physiology, physics, and veterinary medicine and surgery all come together and apply their particular expertise to research problems.

Our commitment to collaboration is grounded in the belief that interactions among scientists of diverse backgrounds will lead to multidisciplinary research producing meaningful, far-reaching results. Research programs at DCRC include investigations into cardiac functions, cystic fibrosis, exercise, kidney failure, membrane transport, muscular dystrophy, neurohumoral control of the circulation, shock, vascular wall biology, diabetes, hypertension, biomedical engineering, protein-protein interactions, and tumor angiogenesis. Because the mission of DCRC is to promote interaction and collaboration, no single group completely defines the research activity of its members.

The center is committed to excellence in cardiovascular research and in the education of students and fellows. Our investigators provide service to the University, the State of Missouri, and the nation through memberships on committees, peer review panels and editorial boards of scientific journals. During the period of this report, our Resident and Non-Resident Investigators have over $43 million in active research funding, have published 145 manuscripts in nationally recognized journals and books and gave 32 Invited presentations.
The Dalton Cardiovascular Research Center is accredited by both the American Association for the Advancement of Laboratory Animal Care and the American Association of Laboratory Animal Sciences.

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Dr. Mark McIntosh

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Mr. Mark Baepler

Director's Office and Administrative Contacts

Interim Director, Dr. Michael Hill
Phone: 573-882-9662 E-mail: hillmi@missouri.edu

Development, Gerald Meininger
Phone: 573-884-4954 E-mail: meiningerg@missouri.edu

Business Manager, Brenda Dudley
Phone: 573-882-7587 E-mail: dudleyb@missouri.edu

Business Support Specialist Sr, Bin Ke
Phone: 573-882-9502 E-mail: keb@missouri.edu

Business Ops Associate Sr, Liang Xu Pre/Post Awards
Phone: 573-882-7433 E-mail: liangxu@missouri.edu

Executive Assistant/Building Coordinator, Laura McClaskey
Phone: 573-882-9482 E-mail: mclaskeyl@missouri.edu

Office Support Assistant IV, Marcia Brewer
Phone: (573) 884-9123 E-mail: brewermj@missouri.edu

System Administrator-Expert, Jason Lee
Phone: 573-882-6348 E-mail: leejb@missouri.edu

User Support Analyst-Expert, John Donahue
Phone: 573-882-6348 E-mail: donahuejt@missouri.edu

Animal Facility Manager, Mark Baepler
Phone: 573-884-2318 E-mail: baeplerm@missouri.edu

Animal Technician, Assistant Lab, Stacey Mathes
Phone: 573-884-2318 E-mail: mathess@missouri.edu

Interdisciplinary Research Interests Groups

Biomedical Engineering
Investigators: M.J. Davis, Ding, Gillis, Gu, Hill, Huxley, Hwang, Jones, Meininger, Milanick, Polo-Parada, Segal, Zou, Sun

Cystic Fibrosis
Investigators: Clarke, Hwang, Milanick, Soma, Zou,

Exercise/Inactivity Including Atherosclerosis, Muscle Biology, Obesity, Type II Diabetes, and Vascular Biology
Investigators: Bender, Booth, Bowles, Hasser, Hill, Huxley, Jones, Korthuis, Laughlin, Martinez-Lemus, Meininger, Polo-Parada, Rubin, Segal, Soma, Sun, Fay, Sowers

Membrane Transport
Investigators: Clarke, Gillis, Gu, Huxley, Hwang, Milanick, Polo-Parada, Rovetto, Zou, Bompadre

Microcirculation
Investigators: Bender, M.J. Davis, Ding, Hill, Huxley, Korthuis, Laughlin, Martinez-Lemus, Meininger, Segal, Sun

Neurohumoral Control of the Circulation Including Ageing, Hypertension, Heart Failure, Respiration and Salt and Water Homeostasis
Investigators: Ding, Hasser, Heesch, Kline, Meininger, Milanick, Segal, Nichols

Tumor Angiogenesis
Investigators: G.E. Davis, Hyder, Liang

Cardiac Muscle, Development & Disease
Investigators: Baines, Krenz, Meininger, Sun, Polo-Parada, Bysani

Interim Director

Michael A. Hill

Office: 133A DCRC
Phone: 573-882-9662
E-mail: hillmi@missouri.edu

Education:
PhD & MS University of Melbourne

Appointment: Professor, Department of Medical Pharmacology and Physiology; Associate Director, Dalton Cardiovascular Research Center

Research

Our laboratory has a principal interest in understanding the signaling mechanisms that underlie the vasoconstrictor response of an arteriole following an acute rise in intraluminal pressure (myogenic response). Our studies have examined the roles of a number of vascular smooth muscle signaling molecules including various kinases and intra-cellular Ca2+. More recently these studies have been extended to include approaches aimed at determining the relationships between pressure induced changes in smooth muscle membrane potential and the resulting signaling events that ultimately lead to the contractile response.

In addition to basic studies on myogenic signaling, we are also studying how myogenic tone interacts with other vasoregulatory mechanisms (principally, endothelial-dependent dilation) and how myogenic response is altered in diabetes mellitus.

Resident Investigators

Christopher P. Baines

Office : 323 DCRC
Office : 573-884-8767
E-mail: Bainesc@missouri.edu

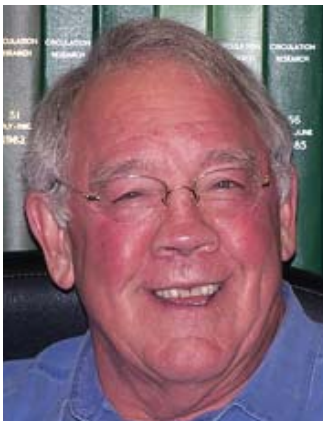
Education:
B.Sc. University of Bath, Great Britain
PhD University of South Alabama

Appointment: Assistant Professor, Department of Biomedical Sciences

Research

Mitochondrial dysfunction is often an underlying cause of myocardial disease. In particular, cardiac pathologies such as ischemia/reperfusion injury, heart failure, diabetic cardiomyopathy, anti-cancer agent-induced cardiotoxicity, etc., are associated with rapid and dramatic increases in mitochondrial permeability.

These changes in permeability lead to ATP depletion, excessive production of reactive oxygen species, and ultimately swelling and rupture of the organelle, thereby instigating a molecular chain of events that leads to cardiomyocyte death. The long-range goal of the lab is to understand how specific mechanisms of mitochondrial-driven death can be targeted for the prevention of myocardial disease.



Edward H. Blaine, Emeritus Dalton Investigator

Office: 355 DCRC
Phone: 573-882-9014
E-mail: blainee@missouri.edu

Education:
PhD, MA & AB University of Missouri-Columbia

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

Currently retired, but the primary focus of my laboratory was the underlying cause of hypertension that is associated with an adverse uterine environment. It is well documented that offspring of mothers who have undergone a variety of stressful circumstances during pregnancy may give birth to offspring who suffer from conditions such as hypertension, type II diabetes, and metabolic syndrome as adults. This phenomenon is known as fetal programming or the fetal origin of adult disease. One characteristic of these susceptible offspring is a reduced nephron number and several investigators have suggested that the lack of a full nephron complement is the underlying cause of the adult onset hypertension. How the reduction in nephron number causes or contributes to hypertension is not known. We worked on the hypothesis that there is a differential loss of nephrons. Based on inherent heterogeneity, loss of more "salt-losing" nephrons and retention of more "salt-saving nephrons" results in an overall tendency for the kidney of these offspring to retain sodium and, therefore, be more susceptible to hypertension as adults.

Our work involved whole animal studies which detail changes in the kidney's ability to excrete sodium and whether changes in ability to excrete a sodium load is a function of changes in glomerular filtration, sodium reabsorption, or both. These studies were conducted in conscious, freely moving animals to obtain data that is not compromised by anesthesia or restraint. We were also studying changes in renal morphology, especially distribution of the loops of Henle, using immunohistochemistry techniques.

Our interest was in neuroendocrine regulation of fluid balance and cardiovascular function, with emphasis on hypertension and heart failure. We were particularly interested in the actions of angiotensin II and vasopressin on brain cardiovascular centers. Our work has demonstrated two distinct actions of angiotensin on blood pressure regulation: acutely, angiotensin has its primary effect directly on vascular smooth muscle, while chronically, the hypertensive effects of angiotensin are mediated through activation of central nervous system pathways. Not only are the circumventricular organs important, but other brain areas are activated during acute and chronic angiotensin infusion. Our next step was to map the distribution of heightened nervous system activity in the peripheral vascular beds that is associated with chronic angiotensin hypertension. We also investigated receptor regulation and post receptor signaling associated with angiotensin infusion.

We were also interested in the role of the renin-angiotensin system in diabetic nephropathy and the mechanism by which drugs that inhibit this system are effective in preventing renal damage.

Lane Clarke

Office: 324D DCRC
Phone: 573-882-7049
E-mail: clarkel@missouri.edu

Education:
PhD North Carolina State University,
DVM, MS & AB University of Missouri,-Columbia

Appointments: Professor, Department of Biomedical Sciences

Research

Our laboratory investigates abnormalities of acid-base transporters in cystic fibrosis and other genetic diseases that contribute to epithelial hyperproliferation and dysfunction of secretory cells in the intestinal crypts, i.e., the site of stem cell activity and cell differentiation. Studies of mice with gene-targeted deletion of CFTR (the cystic fibrosis gene) or other acid-base transporters employ in vivo, ex vivo or primary murine or human organoid culture. Mechanistic studies of molecular interactions are performed using cell lines. Functional activity of acid-base or nutrient transporters is measured in real time using fluorescence confocal or conventional microscopy and electrophysiological methods. Gene or protein expression is measured using quantitative real-time PCR, microarrays, immunoblots, immunofluorescence and laser capture microdissection. Currently, three major projects in the laboratory are funded by NIDDK or the Cystic Fibrosis Foundation. The first project investigates the role of CFTR in down-regulating the cell cycle dynamics and Wnt/□-catenin signaling in intestinal stem cells (ISCs). Loss of this regulation in cystic fibrosis (CF) results in intestinal hyperproliferation which likely contributes to the six-fold increase in the incidence of gastrointestinal cancer in the relatively young population of CF patients. The second project investigates the acid-base transporters expressed in ISCs that determine intracellular pH (pHi). Manipulation of pHi is used to control proliferation in a timed manner to offset the "bystander" damage to ISCs resulting from therapeutic doses of chemotherapeutic reagents and radiation during cancer treatment. The third project investigates goblet cell (mucus secreting) dysfunction in the CF intestine, i.e., mucoviscidosis. The goals are to investigate the factors contributing to hyperplasia of goblet cells and the causes of abnormal exocytosis. To facilitate the translational potential of the above projects, our laboratory is developing a human CFTR "rescue" mouse model in which murine CFTR is replaced by the human ortholog of the gene. This humanized CFTR mouse will also enable pharmacological testing of reagents designed to correct defective function CFTR in CF patients and pharmacological/probiotic strategies designed to combat infectious diarrheal diseases in humans.

Shinghua Ding

Office: 324E DCRC
Phone: 573-884-2489
E-mail: dings@missouri.edu

Education:
PhD & MS State University of New York,
BS Zhejiang University of Technology

Appointment: Assistant Professor, Department of Biological Engineering

Research

Cerebral ischemia (ischemic stroke) is a leading neural disorder that causes brain damage and human death, and has a major impact on public health. Though various mechanisms by which ischemia induce brain damage have been proposed, clinically there is limited therapeutic approach that is effective to brain recovery after ischemia. Therefore, my research generally focuses on seeking and identifying new mechanisms that can reduce brain injury and improving long-term outcomes after stroke. My research focuses on two distinct but related areas: 1) Glial function and role in stroke; 2) Neuronal mechanisms in brain protection in stroke. We use mice (in vivo) and primary cultured cells (in vitro) including neurons and astrocytes isolated from mouse brains as experimental preparations. We use both in vivo and in vitro ischemic models for ischemic study. Approaches including molecular biology, fluorescent imaging including 2-in vivo two-photon (2-P) microscopy, confocal and epi-fluorescent microscopy, biochemistry, electrophysiology, cell culture, and immunocytochemistry are integrated in our research.

Kevin D. Gillis

Office: 222B DCRC
Phone: 573-884-8805
E-mail: gillisk@missouri.edu

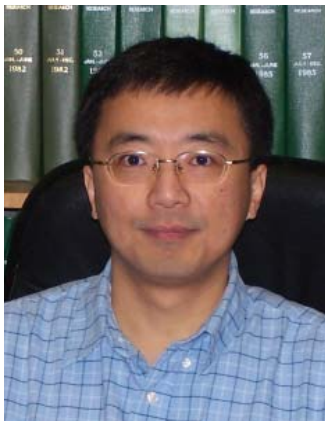
Education:
DSc, MSEE & BSEE Washington University,
BA St. Louis University

Appointment: Professor Biological Engineering, Professor Medical Pharmacology and Physiology

Research

My main area of interest is understanding the final steps of cell secretion and the modulation of these steps by calcium and second messengers. We use multiple biophysical approaches to assay dynamic aspects of secretion from individual adrenal chromaffin cells. Since calcium and second messengers play a central role in regulating both secretion of hormones and release of neurotransmitter at synapses, the results of our research have an impact on understanding such diverse phenomena as the "fight or flight" response and the formation of short-term memory.

Our research also has a strong engineering component with particular emphasis on developing or refining electrical and optical techniques for studying secretion. In particular, we have been developing microchips with arrays of transparent electrochemical electrodes to measure secretion of catecholamines from individual cells simultaneously with optical measurements. Other techniques in use in the lab include patch-clamp electrophysiology with membrane capacitance measurements as an assay of exocytosis/ endocytosis, photometric measurement of the intracellular Ca2+ concentration with indicator dyes, and photo- release of intracellular Ca2+ from caged compounds.



Liqun (Andrew) Gu

Office: 229 DCRC
Phone: 573-882-2057
E-mail: gul@missouri.edu

Education:
PhD Nankai University

Appointment: Associate Professor, Department of Biological Engineering

Research

We are a newly established nano-biotech laboratory focusing on application of lab-in-hand nanotechnology to the exploration of life science problems and practical biomedical detection. Currently, we utilize various nanopores as receptive probes for single molecule detections. Nanopore refers to a pore structure adopting dimension from one to hundreds nanometer, which can either be formed by naturally-occurred self-assembled protein pore or fabricated by fashion nanotechnology on solid substrates. With the nanopore probe, any analyte, whether it is a single molecule, a molecular complex, or a single viral particle, can reversibly bind to a receptor which is pre-engineered in a properly sized nanopore, and be identified by recognizing the characteristic blocks to the nanopore conductance.

Our laboratory is initially supported by the University of Missouri Startup Fund, Research Board and Research Council. Recently, this laboratory successfully got a National Science Foundation (NSF) Career Award grant in support of a new direction focusing on single molecular protein-oligonucleotide interaction and molecular folding in a nanopore. Specifically, we apply the nanopore technology to the single molecule investigation of dynamic aptamer-protein interactions and related folding processes involved in molecular recognition. Aptamers are engineered DNA/RNA that can specifically recognize broad species of proteins with high affinities, such as HIV-1 Integrase. Upon binding, these powerful molecules can form complex three-dimensional structures and possess sophisticated functions to inhibit pathogen protein, catalyze chemical reactions, control gene expression, and regulate cellular functions, therefore potentially be applied as tools for exploring biological systems. A complete understanding of dynamic processes in aptamer-target interactions and molecular folding is not only important to application-driven rational design, but also gives deep insight into the complexity of various nucleic acid-protein interactions in living cells.

Our research will be significant in the quantitative characterization and precise control of molecular scale components or nanomachinery of living cells by employing fashioning tools that allow us to manipulate complex biological processes in unique ways. Research using nanopore technology can be broadly applied to the study of diverse nucleic acid-protein and protein-protein interactions, which is essential for rational design of small organic molecules that block malfunctions of the cellular machinery, or act as new therapeutic reagents and products for biotechnology and bioengineering applications. This research will also greatly expand the capability of nanopore as the new generation of detection technology for analysis, high-throughput screening, bio-defense, and environmental engineering. In a broader impact, nanopore research will shed light on nanobiotechnology, an interdisciplinary and collaborative area related to biomolecular science, biotechnology, chemical engineering and nanotechnology.

Eileen M. Hasser

Office: 351 DCRC
Phone: 573-882-6125
E-mail: hassere@missouri.edu

Education:
PhD University of Oklahoma, BA Gettysburg College

Appointments:Professor, Department of Biomedical Sciences
Adjunct Professor, Medical Pharmacology and Physiology

Research

The primary interest of the laboratory is in central nervous system control of the sympathetic nervous system and blood pressure, and how these reflex systems are modulated under physiological and pathophysiological conditions. Currently, the laboratory is involved in three primary projects.

The primary goal of the first project is to determine the neurotransmitter/receptor mechanisms involved in altered regulation of blood pressure in acute and chronic hypertension. The second project examines potential changes in both cardiovascular regulation and vascular function which may be responsible for cardiovascular deconditioning following prolonged exposure to spaceflight or bedrest. The focus here is on plasticity in specific brain regions that influence cardiovascular control. The third area of investigation focuses on the reflex effects of circulating humoral factors which act in the central nervous system to alter cardiovascular regulation. This project examines the central nervous system neurotransmitters and pathways involved in these effects. These questions are examined utilizing conscious animals which are chronically instrumented for recording of blood pressure, blood flow and sympathetic nerve activity. In addition, anesthetized preparations are utilized for experiments involving electrical and chemical stimulation of specific brain regions, microinjections of neurotransmitter agonists and antagonists, and central neurophysiological recording.

The overall goal of this work is to understand the central nervous system mechanisms underlying cardiovascular regulation in normal and disease states.

Cheryl M. Heesch

Office: 324C DCRC
Phone: 573-882-2359
E-mail: heeschc@missouri.edu

Education:
PhD University of Texas Health Science Center,
BS New Mexico State University

Appointment: Professor, Department of Biomedical Sciences

Research

Our laboratory's major focus is to understand how the central nervous system controls arterial blood pressure and how blood pressure is modulated in physiological and pathophysiological states. We evaluate basic mechanisms involved in central nervous system control of autonomic outputs, particularly in brainstem regions which are critical for providing tonic drive to the sympathetic nervous system. We also study how ovarian hormones modulate these basic control mechanisms. Ongoing projects in the laboratory are focused on elucidating the role of central nervous system effects of ovarian hormones and progesterone metabolites in the alterations in control of sympathetic outflow associated with term pregnancy. Once we understand the mechanisms for attenuated sympathoexcitation in normal pregnancy, it may be possible to determine the mechanisms for elevations of arterial blood pressure in hypertensive disorders, where sympathoexcitatory responses are exaggerated. Also, women are generally protected from heart disease and hypertension until the onset of menopause. Understanding the mechanisms for the protective role of ovarian hormones could have important implications for treatment and prevention of cardiovascular disorders.

The possibility that a metabolite of progesterone, 3 -OH-dihydroprogesterone, may play a major role in suppression of sympathoexcitatory responses is especially intriguing. 3 -OH-dihydroprogesterone is the most potent endogenous positive modulator of central nervous system GABAA receptors and physiologically significant levels have been reported in the central nervous systems of both males and females. Experimental approaches include: 1.) measurement of sympathetic nerve activity; 2.) CNS microinjection of putative transmitters and modulators; 3.) extracellular single unit neuronal recording; and 4.) evaluation of neurotransmitter receptor expression in relevant brain regions.

Tzyh-Chang Hwang

Office: 222C DCRC
Phone: 573-882-2181
E-mail: hwangt@missouri.edu

Education:
PhD Johns Hopkins University, MD National Yang-Ming Medical School,
MS National Tawain University School of Medicine

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

CFTR (Cystic Fibrosis Transmembrane conductance Regulator) is a chloride channel that plays a critical role in secretion and absorption of water and electrolytes across epithelia. Since CFTR channels are also expressed in cardiac myocytes and are found to shorten action potential duration and induce repetitive activity, they are implicated to be arrhythmogenic. One unique feature of the CFTR protein as an ion channel is that the free energy of ATP hydrolysis is harvested to drive the conformational changes that open and close the channel. Studies using mutant CFTR and various ATP or phosphate analogs have suggested a model that ATP hydrolysis at two nucleotide binding sites is tightly coupled to the opening and closing of the channel pore. Our understanding of the molecular basis of the coupling mechanism, however, remains primitive. Unresolved questions include: What is the stoichiometry of ATP binding/hydrolysis to gating transitions? How are the biochemical states in ATP hydrolysis cycles translated to the open and closed states in the gating transitions? Which part of the protein forms the aqueous pore? What is the relationship between the gate and the pore? These are the fundamental questions that interest a broad spectrum of physiologists.

A combinational approach is being adopted to tackle the molecular physiology of CFTR chloride channels. Different configurations of the patch-clamp techniques will be used to record CFTR channel activity so that both the cytoplasmic and extracellular sides of the channel are accessible to channel blockers, modifiers, or channel openers. Structure-guided mutagenesis approaches will be employed to study the functional consequences of single amino acid substitutions on gating and permeation/blocking. State-dependent chemical modifications of engineered cysteines allow us to explore the dynamic protein conformational changes during gating transitions. The aims of our ongoing research are: 1. To understand the role of ATP binding and hydrolysis in controlling the opening and closing transitions of CFTR. 2. To probe the CFTR pore with permeant and impermeant anions. 3. To explore the structure/function relationship between the gate and the pore of CFTR. 4. To characterize how CFTR activators act to increase the activity of CFTR. 5. To apply structure-based drug design to identify chemicals that can correct trafficking defects in CF-associated mutations. A clear understanding of the molecular mechanism of CFTR function will aid in the design of pharmacological agents for therapeutic intervention in cystic fibrosis, secretory diarrhea and cardiac arrhythmia.

Salman M. Hyder

Office: 133B DCRC
Phone: 573-882-1261
E-mail: hyders@missouri.edu

Education:
PhD University of Glasgow, BS University of Kent

Appointment: Professor, Department of Biomedical Sciences

Research

The main aim of our laboratory is to identify steroid hormone dependent molecular targets that can be utilized for anti-angiogenic therapy of endocrine dependent disease such as the breast, uterine, and prostate cancer. Formation of new blood vessels, or angiogenesis, is crucial for normal processes such as embryonic development, wound healing, and endometrial regeneration following menstruation. Angiogenesis is also essential for tumor growth, and metastasis. An emerging field in cancer therapeutics is the targeting of new blood vessels to curtail tumor growth. Our laboratory is currently focusing on the role of steroid hormones, clinically relevant anti-hormones, and endocrine-disruptors to understand the basic mechanisms involved in hormone driven angiogenesis in breast, and uterine tissue. We are currently focusing on the role of estrogens, progestins, and their nuclear receptors in controlling the expression of potent angiogenic growth factors (e.g. VEGF and its receptors) at both molecular and cellular level. Another focus of the laboratory is to investigate the molecular mechanisms of steroid hormone action at the level of gene transcription. We are especially interested in the role of natural and synthetic ligands that have diverse biological effects in different target tissues (e.g. SERMS such as tamoxifen). Many synthetic ligands (agonists/antagonists) are consumed by millions of women all over the world for oral contraception, hormone replacement therapy, or treatment of breast cancer. Consumption of some of these ligands lead to increased risk of breast and/or uterine abnormalities, including cancer. We anticipate that understanding the molecular basis/pharmacology of ligand-nuclear receptor interactions will allow development of better therapeutic modalities for treatment of hormone dependent tumors, as well as endometriosis, osteoporosis and infertility.

David D. Kline

Office: 354 DCRC
Phone: 573-884-0505
E-mail: klinedd@missouri.edu

Education:
PhD Case Western Reserve University, BA Miami University

Appointment: Associate Professor, Department of Biomedical Sciences

Research

Our laboratory focuses on the autonomic nervous system, in particular the cardiovascular and respiratory system. These vital systems operate to keep our bodies within "normal" physiological limits to preserve homeostasis. When challenged acutely or chronically with low environmental oxygen levels (hypoxia) respiration, blood pressure and heart rate compensate to maintain arterial blood gas levels. This can happen during high altitude assent or disease states such as sleep apnea by activation of the chemoreceptor reflex. Additionally, arterial blood pressure is maintained during swings in pressure by the baroreceptor reflex. Both reflex pathways result from activation of neurons in the peripheral and central nervous system. Determining the mechanism of action of these reflex pathways during health and disease is the focus of the laboratory.

Several techniques are used to elucidate these mechanisms. These include 1) radiotelemetry in conscious animals to measure respiration, blood pressure or heart rate; 2) immunohistochemical localization of ion channels and neurotransmitter receptors to specific regions of the nervous system and individual neurons; 3) patch clamp techniques in isolated neurons for recording current flow through ion channels and 4) electrical recording of synaptic transmission in brainstem slices.
Using these techniques, we have recently discovered that chronic intermittent hypoxia, a model for obstructive sleep apnea, elicits a form of neural adaptation or plasticity in the brainstem. This includes changes in neurotransmitter release from presynaptic chemoreceptor afferent neurons as well as postsynaptic action potential firing. We are currently determining the mechanism of this altered neurotransmitter release.

Maike Krenz

Office : 355 Dalton Cardiovascular Research Center
Phone: 884-8761
Email: krenzm@missouri.edu
Education:
MD Germany

Appointment: Assistant Professor, Department of Medical Pharmacology and Physiology

Research

Congenital heart defects remain the most common birth defect, occurring in about 1% of live births and constituting the leading cause of infant deaths in the US. Over the past decade, genetic analyses of families with congenital heart disease have directed us to the molecular causes of certain defects. In particular, gain-of-function mutations in the protein tyrosine phosphatase Shp2 have recently been discovered in families with Noonan syndrome. In the majority of cases, NS follows autosomal dominant inheritance and is characterized by short stature, facial dysmorphia, skeletal anomalies, and congenital heart disease. Among the heart defects, pulmonary valve stenosis and hypertrophic cardiomyopathy are most prominent. Understanding the exact cellular mechanism(s) by which dysfunction of Shp2 causes valve malformation may provide the basis for future development of novel therapeutic approaches in congenital heart disease.

To study the pathomechanisms of heart malformations, we have been creating genetically altered mouse models that recapitulate human congenital heart disease. In particular, our mouse models are designed to express the mutant proteins in a time- and tissue-specific manner. These models can then be used to study in detail which developmental steps in the heart play a role in the disease process. Subsequently, we can dissect the downstream signaling pathways through which mutant Shp2 mediates its effects in vivo. For example, we were recently able to show that the ERK pathway is both necessary and sufficient for the development of valve defects in a Noonan Syndrome mouse model. Furthermore, we use tissue culture approaches to investigate the effects of Shp2 mutations on the cellular and biochemical level to complement the in vivo mouse studies.

Yayun Liang

Office Location: 133D Dalton Cardiovascular Research Center
Office Phone: 573-884-8116
E-mail: liangya@missouri.edu

Education:

PhD Beijing Medical University

Research

The concept of specific molecular targeting has been applied to the development of innovative cancer-treatment strategies. At present, two main approaches are available for use in clinical practice: therapeutic monoclonal antibodies and small-molecule agents. Both antibodies and small-molecule compounds are therefore promising tools for target-protein-based cancer therapy. Mutations in p53 or the p53 pathway are thought to play a key role in promoting tumor cell survival and tumor cell resistance to chemotherapeutic drugs. Therefore restoring p53 function in tumors has been pursued as a promising strategy for cancer therapy. Furthermore, Tumor cell survival, growth, and metastasis require persistent blood vessel growth or angiogenesis. A tumor cannot grow beyond the size of about 1mm in diameter without acquiring new blood vessels to nurture it. Hence, targeting tumor blood vessels and tumor angiogenesis has been as a new strategy for treatment cancer.

The aims of Liang's research are to 1) Develop innovative cancer-treatment strategies targeting mut-p53, tumor angiogenesis, and tumor blood vessels with novel antibodies and small molecules in advanced breast cancer models; 2) Define molecular signaling pathways involved in inhibition of tumor angiogenesis and induction of tumor cell apoptosis; and (c) Define the role of VEGF in tumor angiogenesis, growth, metastasis, and drug resistance.

Luis Martinez-Lemus

Office: 222A DCRC
Phone: 573-882-3244
E-mail: martinezlemusl@missouri.edu

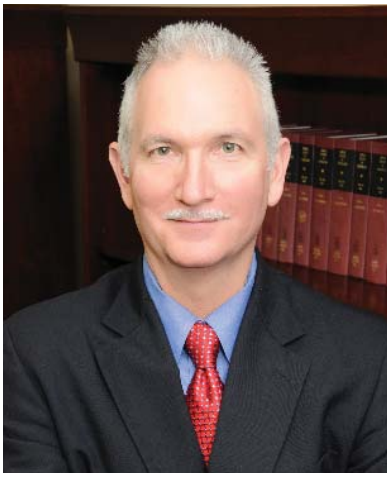
Education:
PhD Texas A&M, MS Auburn University,
DVM from Universidad Nacional Autonoma de México

Appointment: Associate Professor, Department of Medical Pharmacology and Physiology

Research

My research is focused on the mechanisms responsible for the architectural transformation of blood vessels also known as vascular remodeling. Vascular remodeling is a hallmark for numerous cardiovascular diseases, yet numerous questions remain to be answered regarding this process. What stimuli drive the remodeling process? How do blood vessels detect those stimuli? What are the mechanisms initiating the remodeling and under which conditions are they counterproductive participating in disease states?

Currently funded research in my laboratory is focused at determining the changes in the position and function of cells within the intact blood vessel wall that occur in response to common mechanical and vasoactive biochemical stimuli. Our studies indicate that cells within the vascular wall rapidly change their position in response to stimulation in as little as four hours. This adaptive cell behavior appears to allow the vessel to maintain a reduced diameter for extended periods of time with reduced levels of activation and energy expenditure. An additional goal is focused at determining the changes in the structure and compliance of the extracellular matrix that occur during the initial stages of the remodeling process.



Gerald A. Meininger

Office: 133B DCRC
Phone: 573-882-9662
E-mail: meiningerg@missouri.edu

Education:
PhD University of Missouri-Columbia,
MS & BS Central Michigan University

Appointments:
Margaret Proctor Mulligan Professor in Medical Research
Professor, Department of Medical Pharmacology and Physiology
Adjunct Professor, Department of Biomedical Sciences
Adjunct Professor, Department of Physics

Research

Research interests in my laboratory have been focused on vascular physiology and cell biology, with an emphasis on the microvasculature. Currently active areas of research include: 1.) Cell adhesion, extracellular matrix and cell signaling in the vascular wall; 2.) Mechanotransduction in vascular cells; 3.) Regulation of vascular and cardiac responses to tissue injury by extracellular matrix derived signals; 4.) Cellular and molecular mechanisms responsible for mechanotransduction; 5.) Mechanisms of vascular remodeling; 6.) Mechanisms responsible for the myogenic properties of vascular smooth muscle; 7.) Application of fluorescence microscopy and 3D-image analysis for studies of microvascular cell biology and the cyto-architecture and function of the microvessel wall; and 8.) Mechanisms of blood flow autoregulation.

Laboratory models include studies of both the intact microcirculation and of isolated arterioles, freshly dispersed or cultured vascular smooth muscle cells, endothelial cells and cardiac muscle cells. Examples of technical approaches include pharmacology of the intact microvasculature and isolated microvessels; ability to manipulate pressure and flow in isolated microvessels; vessel culture and transfection; immuno-cytochemistry of the microvessel wall and isolated cells; three dimensional fluorescence imaging using confocal, multiphoton or wide field microscopy in combination with deconvolution; atomic force microscopy combined with fluorescence microscopy (TIRF and FRET), and software development for high through-put analysis and display of atomic force microscopy force data.

An emphasis over the last several years has been to understand the role of the extracellular matrix, adhesion molecules and the cytoskeleton in regulation of vascular and cardiac cells, especially in the control of contractile function. A fundamental aim of this work has been to determine to what extent this matrix-adhesion-cytoskeletal axis may be involved in mechanotransduction phenomena that underlie the vascular myogenic response, flow-dependent responses of the endothelium and vascular remodeling. Advances in hybridizing atomic force microscopy with fluorescence microscopy are permitting higher through-put evaluation of cell surface receptors and their interactions with specific ligands.

Our future plans include continuing to study the role of the extracellular matrix and cell adhesion molecules in the regulation of vascular and cardiac cell function. We are extending our studies of mechanotransduction down to the level of single molecular interactions between integrins and extracellular matrix molecules, as well as to the mechanical dynamics of single focal contacts.

Luis Polo-Parada

Office: 302 DCRC
Phone: 573-884-4599
E-mail: poloparadal@missouri.edu

Education:
PhD Case Western Reserve University, MS University of Connecticut,
BS School of Physics and Mathematics, National Polytechnic Institute

Appointment: Assistant Professor, Department of Medical Pharmacology and Physiology

Research

The heart is the first organ to form during embryogenesis, and its function is critical for the proper development and survival of the embryo. Although some information on ion-transport genes and their protein products in normal and diseased myocardial tissue is available, little is known about the role of cardiac extracellular matrix (ECM) proteins during cardiac development or in healthy and diseased adult hearts. My interest is to elucidate the role of the ECM in the ionic-transport proteins and molecular basis of cardiac regional electrical specialization during development and in the adult heart.
My recent studies of neural cell adhesion molecule (NCAM) null mice have indicated that NCAM plays a fundamental role in the transmitter release mechanism in neuroendocrine cells through mediation of granule recruitment. Other studies have shown that NCAM plays a functional role in the proper segregation of cell during development of islets of Langerhans in the pancreas. My interest is to elucidate whether NCAM or other cell adhesion molecules, are directly involved in the proper organization of the islets of Langerhans and in glucose-mediated insulin secretion. I also intend to investigate, whether the expression of any of these cell adhesion molecules is affected in diabetic type II patients and animal models of the disease.

Zhe Sun

Office: 250 Dalton Cardiovascular Research Center
Phone: 573-884-2499
E-mail: sunzh@missouri.edu

Education:
BS in chemical engineering, Chengdu University of Science & Technology, China
MS in chemical engineering, Sichuan Union University, China
PhD in bioengineering, University of Toledo, Ohio

Appointment: Medical Pharmacology & Physiology

Research

Sun's primary interest is in development of novel techniques towards understanding the dynamics of cellular interactions with the extracellular matrix (ECM). The goal is to access the cellular dynamics from both biochemical and biophysical perspectives in real-time, for example to monitor the intracellular signaling, cell adhesion with extracellular matrix proteins and the cellular mechanical activities etc.

The approaches used include live cell fluorescence imaging and FRET to monitor cellular signaling events and specific molecular interactions, and atomic force microscopy (AFM) methods to monitor the cellular mechanical activities and the interaction force between cell and ECM (usually falls in pN~nN range). As an integrated part of these studies, Sun is also interested in developing software for image processing, data analysis and computational modeling of the cellular force transmission. By integrating these techniques together, the understanding of the nature of the cell-ECM interactions will be furthered.

Xiaoqin Zou

Office: 222D DCRC
Phone: 573-882-6045
E-mail: xiaoqinz@missouri.edu

Education:
PhD University of California, San Diego,
BS Wuhan University

Appointment: Associate Professor, Department of Biochemistry

Research

The molecular interactions that drive ligand-protein binding are a key to quantitatively understanding the basis of molecular recognition and to designing therapeutic interventions through rational drug design. Drug molecules usually act by binding to specific target proteins. Drug candidates that have high binding affinities can be identified by their geometric and chemical complementarity to the target in a process analogous to solving a "jigsaw puzzle," if the target structure is known. An energy model that can give rapid and accurate evaluation of the molecular interaction strength is thus essential for selecting plausible candidate compounds from a chemical database consisting of hundreds of thousands of molecules. We are developing novel and efficient algorithms to calculate binding free energies for ligand-receptor complexes. The derived energy models will be applied to protein-substrate interactions, protein-protein interactions, and structure-based drug design. We are also developing new docking algorithms to account for protein flexibility. Methods used in our laboratory include computer modeling, simulation and graphics display. A second line of research in the laboratory is quantitative studies on structure-function relationship of membrane proteins. Structures of membrane proteins will be predicted using homology modeling and structure alignment techniques. Structural information often suggests mechanisms of protein function, which will be experimentally tested in collaboration with other Dalton Investigators.

Non Resident Investigators

Shawn B. Bender, Ph.D.

Office: E102 Veterinary Medicine
Office: 573-882-7011
Email: benders@missouri.ed

Appointment: Assistant Professor, Department of Biomedical Sciences

Research

The long-term goal of our research program is to elucidate mechanisms of obesity-associated coronary microvascular dysfunction and thereby identify novel pathways and therapeutic targets to reduce cardiovascular complications in these patients. The central premise of our work is that coronary microvascular dysfunction and the resultant impairment of coronary blood flow control is an independent predictor of cardiovascular morbidity and mortality in obese, diabetic patients. Impaired coronary microvascular function is estimated to account for more than 60% of cardiac perfusion defects in patients with type 2 diabetes. Thus, coronary microvascular dysfunction is a significant contributor to the increased risk of impaired cardiac function, ischemia, and infarct in these patients.

Current projects in the laboratory focus on the role of the aldosterone-binding mineralocorticoid receptor (MR) as a mediator of coronary dysfunction in obesity. A growing body of evidence has implicated MR signaling in vascular cells as an important mediator of vascular dysfunction in various disease states. Our studies utilize an integrative combination of in vivo and in vitro approaches including cell/tissue culture and knockout mouse models coupled with molecular techniques.

Frank W. Booth

Office: W145 VMED
Phone: 573-882-6652
E-mail: boothf@missouri.edu

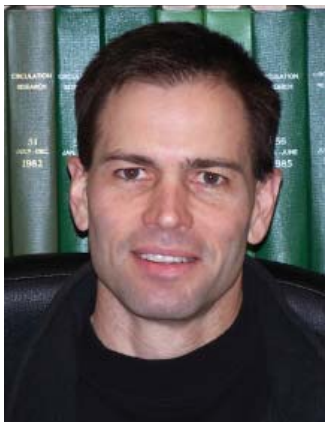
Education:
PhD University of Iowa, BS Denison University

Appointments: Professor, Department of Biomedical Sciences
Adjunct Professor, Department of Medical Pharmacology and Physiology and Department of Nutrition and Exercise Physiology

Research
Research interests in my laboratory currently focus on two areas.

First, we are addressing the question: what is the identity of genes in the nucleus accumbens that motivate rats that were bred to be "born-to-run" to have long distances of voluntary running in wheels, as compared to other rats that were bred to mimic "couch potato" behavior by having low distances of voluntary running?

Second, we are tackling the questions: 1) does voluntary running in wheels produce higher peak lifetime aerobic capacities than in rats without wheels for voluntary running; 2) does voluntary running attenuate primary aging-induced loss of aerobic capacity; and 3) which genes are responsible for the previous two questions?



Doug Bowles

Office: W124 VMED
Phone: 573-882-7193
E-mail: bowlesd@missouri.edu

Education:
PhD University of Texas-Austin

Professor, Department of Biomedical Sciences,Adjunct Professor, Medical Pharmacology and Physiology

Research
The goal of our lab's research is to understand how ion channels affect the role of coronary smooth muscle (CSM) in vascular pathology, i.e. atherosclerosis and post-angioplasty restenosis. Our major research question is how physical activity/exercise and sex hormones alter ion channel function in coronary smooth muscle to affect the onset and progression of coronary vascular disease.

Currently, the major focus is on two distinct channels, the intermediate-conductance, calcium-activated K channel (IKCa1) and channels underlying store-operated calcium entry, most likely TRPC channels. We are interested on these as they are upregulated during CSM phenotype modulation that occurs during vascular injury. Our lab strives for a molecule to animal approach using cultured and native cells, intact vessels and in vivo models. The primary model is the swine, as its coronary physiology, anatomy and gene expression profile is most similar to human. Experimental techniques include whole-cell and patch clamp, fluorescent based calcium imaging, isometric vessel recordings, cannulated microvessels, immunoblot, real-time RT-PCR, laser capture microdissection and molecular biology (promoter/reporter constructs, etc) to examine gene regulation by ion channels.

Lastly, we have a state-of-the-art, fully digital cath lab exclusively for large animal (e.g. pig) research. With this we can do angiography, intravascular ultrasound, intracoronary flow and pressure. We can also induce coronary injury/restenosis with balloons and stents. Our goal is to use this to provide direct, in vivo, "translational" endpoints to our cellular/genetic studies.

The results of these experiments will provide novel mechanisms by which a sedentary lifestyle and hormonal status impacts cardiovascular health and well being as well as define potential therapeutic targets for the treatment and prevention of cardiovascular diseases.

Chandrasekar Bysani, D.V.M., Ph.D.
Office: Harry S. Truman Memorial Veterans Hospital
Office: 573-814-6000, ext. 53734
E-mail: chandrasekarb@health.missouri.edu

Education:
Ph.D. Meerut University, Meerut, India and Central Drug Research Institute, Lucknow, India
D.V.M. Andhra Pradesh Agricultural University, Hyderabad, India

Appointment: Margaret Proctor Mulligan Endowed Professor, Department of Medicine/Cardiology

Research

We focus on understanding the patho-physiology of cardiovascular diseases, specifically on investigating the role of pro-inflammatory cytokines and chemokines in cardiovascular disease progression, including ischemic heart disease, pressure-overload hypertrophy, and heart failure. Recently, we identified TRAF3IP2 as a nodal point through which various signal transduction pathways can converge. TRAF3IP2 activates two critical pathways that lead to the induction of NF-□B, AP-1 and C/EBP□, and the induction of inflammatory cytokines with negative inotropic effects. TRAF3IP2 gene deletion markedly reduces myocardial injury and vastly improves post-ischemic myocardial recovery and function.
We have also found that expression of RECK, an MMP inhibitor, is significantly suppressed in the injured heart,

George E. Davis

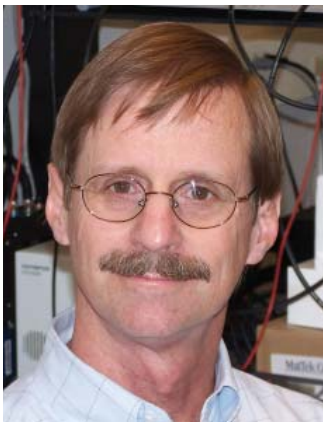
Office: MA415 HSC
Phone: 573-882-5474
E-mail: davisgeo@missouri.edu

Education:
PhD, MD University of California-San Diego,
BS Arizona State University

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

My laboratory focuses on the following questions relevant to angiogenesis, wound repair and cancer research:
How do endothelial cells form cell-lined tube structures with lumens in three-dimensional (3D) extracellular matrices?
How do endothelial cells and other cell types such as tumor cells invade 3D matrices?
To what extent do endothelial cells directly or indirectly play a role in tumor invasion and metastasis?
What molecular events control the process of vascular regression?
How do vascular supporting cells, such as pericytes, stabilize vascular tubes?
How do distinct matrix metalloproteinases and their inhibitors control the processes of vascular morphogenesis versus regression in 3D matrices?
How do extracellular matrix fragments (matricryptins) regulate vascular morphogenesis versus regression in normal versus diseased states (such as diabetes)?



Michael J. Davis

Office: MA415F
Lab: 256 DCRC
Phone: 573-882-5181
Email: davismj@missouri.edu

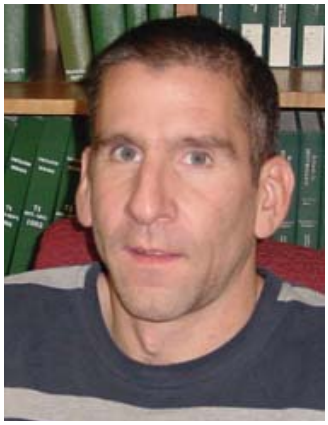
Education:
PhD University of Nebraska
BS University of California, Davis

Appointments:Associate Department Head and Professor, Department of Medical Pharmacology and Physiology

Research

Current research in my laboratory focuses on the ionic mechanisms controlling contraction/pumping of lymphatic vessels. We use mouse models to address questions about the ion channels that normally control pacemaking in lymphatic muscle as well as the underlying causes of lymphatic dysfunction associated with genetic mutations in human patients with primary lymphedema. Methods used in our studies include sharp-electrode and patch-clamp electrophysiology, small vessel pressure myography, confocal microscopy, molecular biology and transgenic mouse models.

My laboratory is supported by the NIH NHLBI to investigate: 1) the ionic basis of pacemaking in lymphatic smooth muscle, 2) the pathophysiology of lymphatic muscle and valves in various models of lymphedema, and 3) the role of smooth muscle and endothelial cell connexins in controlling how electrical signals are conducted and coordinated within and along the lymphatic wall. We also collaborate with other laboratories around the world to investigate the nature of the contractile and valve defects that result in several types of primary lymphedema, including lymphedema distichiasis, Noonan syndrome and Cantu syndrome.



Paul J. Fadel

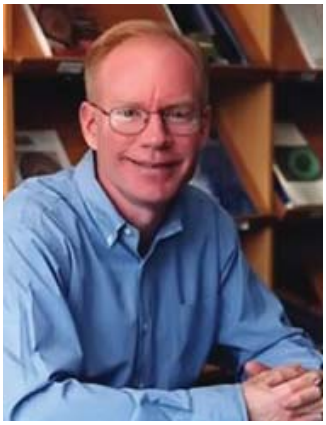
Office: M417 HSC
Phone: 573-884-5220
E-mail: fadelp@health.missouri.edu

Education:
PhD University of North Texas,
MS Northeastern University, Health Science Center, BS Brooklyn College

Appointments:Associate Professor, Department of Medical Pharmacology and Physiology

Research

Our laboratory's research focus entails the investigation of neural cardiovascular control at rest and during exercise in humans with a specific emphasis on the sympathetic branch of the autonomic nervous system. Ongoing studies involve assessing sympathetic responses during various physiological manipulations including isometric and aerobic forms of exercise, lower body negative pressure to simulate the effect of gravity when one stands up, and infusions of pharmacological agents. Studies are performed in normal healthy subjects as well as in patients with various pathophysiological conditions such as heart failure and hypertension. Our laboratory obtains direct measures of sympathetic neural firing using the technique of microneurography. This measurement allows for the assessment of moment-to-moment as well as long-term changes in sympathetic nerve activity. Also, with the application of partial autospectral and time series analyses to muscle sympathetic neurograms we are beginning to investigate the central origin(s) and pattern(s) of sympathetic discharge in humans. Our current research focus is on the neural mechanisms that contribute to exercise-induced sympathoexcitation as well as the peripheral modulators of sympathetically medicated vasoconstriction in contracting skeletal muscle with a particular emphasis on the potential roles of free radicals and changes in nitric oxide signaling in altering these responses. Considering the continually increasing population of elderly individuals, we are beginning to examine age-related alterations in neural cardiovascular control during exercise. Research in this area has been limited and is extremely important considering that an exaggerated blood pressure response to exercise increases the risk for mortality in otherwise healthy adults.



William P. Fay

Office Location: 306 Cs&E
Office Phone: 882-2296
E-mail: fayw@missouri.edu

Education: BS, MD, University of Illinois

Appointments: Professor of Internal Medicine and Medical Pharmacology & Physiology

Research

- Role of leukocyte-derived tissue factor in thrombosis
- Role of plasminogen activator inhibitor-1 (PAI-1) in the proliferative response to vascular injury
- Mechanisms by which C-reactive protein (CRP) modulates thrombosis
- Role of heme oxygenase-1 in thrombosis
- Regulation of fibrinolysis by thrombin activatable fibrinolysis inhibitor (TAFI)

Techniques / Methodology:

- Mouse gene targeting
- Rodent models of human vascular disease
- Structure-function studies of blood coagulation proteins
- Thrombosis models

Research Description

Our research laboratory focuses on the roles of the blood coagulation and fibrinolytic systems in vascular disease. We are interested in the molecular processes that determine acute thrombus formation after vascular injury, as well as those that regulate subsequent thrombolysis. We also are interested in how components of the blood clotting and fibrinolytic systems contribute to the pathogenesis of chronic vascular disorders, such as atherosclerosis and restenosis after percutaneous coronary interventions. We study these issues by a variety of experimental approaches, ranging from in vitro studies with purified proteins to intact animal studies. In particular, we rely heavily on murine models of vascular injury and thrombosis, since they enable us to examine the impact of specific genes on complex biologic processes within the living animal.

Gabor Forgacs

Office Location: 420 Physics Building
Office Phone: 573-882-3036
forgacsg@missouri.edu

Education:
Ph.D. Eotvos Lorand University, Budapest Hungary and Landau Institute for Theoretical Physics, Moscow, Russia

Appointments: George H Vineyard Professor of Theoretical Physics

Research

My research is focused on the physical mechanisms in cell and development biology. In particular we study (both experimentally and by computer modeling) the biomechanical (i.e. viscoelastic) properties of cells and tissues and their relevance to morphogenetic shape transformations. Current activity is concentrated on the application of these physical mechanisms to "organ printing" a fundamentally new approach to tissue engineering, whereby, sperical cell aggregates with composition appropriate for the particular organ (the bioink) are delivered (with a modified ink-jet printer) according to the organ's anatomical blueprint into biocompatible scaffolding gels (the paper).

Shubra Gangopadhyay

Office Location: 243 Engineering Building West
Office Phone: 573-882-4070
Email: GangopadhyayS@missouri.edu

Education:
PhD in physics, Indian Institute of Technology, Kharagpur
MSc in physics, Jabalpur University, Jabalpur
BSc, Jabalpur University, Jabalpur

Appointment: LaPierre Chair and Joint Professor, Departments of Electrical Engineering, Biological Engineering and Physics

Research

Gangopadhyay, an acclaimed researcher in the fields of material science and physics, heads the group. The group has set up a high class research facility — the first of its kind in Missouri — with plans to upgrade and expand the facilities over the next two years.

Research Description
The Gangopadhyay Research Group is an electrical engineering and materials science research facility at the University of Missouri Columbia's College of Engineering and is associated with the International Center for Nano/Micro Systems and Nanotechnology. It is dedicated to expanding the realm of science and technology through optimization of existing techniques and exploration of new dimensions of knowledge. The group's research includes discovering, integrating, and optimizing new materials, processing methods, and characterization techniques. By promoting an interdisciplinary approach, our unique and modern research facility was designed to train, educate and prepare students to join and lead the workforce in innovative solutions to scientific challenges

Kenneth A. Gruber

Office: MU Life Sciences Business Incubator
Phone: 909-210-1441
E-mail: gruberke@missouri.edu

Education:
B.A. & Ph.D., New York University

Appointment: Adjunct Professor, Department of Medical Pharmacology and Physiology. CEO/President: Tensive Controls, Inc.

Research

Our current interests involve two classes of peptides with therapeutic potential: melanocortins and RFamides. Melanocortins are a family of peptides that have a pivotal role in the regulation of vertebrate food intake and metabolism. Drugs with melanocortin receptor activity (agonists or antagonists) show therapeutic potential in obesity and disease-induced cachexia. Cachexia, the focus of our current melanocortin drug development efforts, is a hyper-metabolic state that produces preferential loss of lean body mass and multi-organ failure. Development of melanocortin drugs has been inhibited by the persistent presence of cardiovascular side-effects. We showed that an overlapping RFamide pharmacophore is the cause of melanocortin cardiovascular activity: the melanocortin pharmacophore (HFRW) contains a "synonym" (RW) of the RFamide pharmacophore. We are currently developing anti-cachexia melanocortin-based drugs free of side-effects. As a consequence of this work we have developed new concepts for the detection and regulation of overlapping pharmacophores in drug development.

An unanticipated outcome of our melanocortin research was the observation that RFamides produce electrocardiogram abnormalities resembling the clinical presentation of "sick sinus syndrome." Increasing RFamide peptide doses evoke other arrhythmic predictors of sudden cardiac death, and eventually produce sudden cardiac arrest. Our current goal is to use RFamide ligands to produce experimental models of cardiac arrhythmias, an important medical problem that has eluded model development. Eventually, our goal is to develop RFamide-based anti-arrhythmic drugs.

Virginia Huxley

Office: M404 HSC
Phone: 573-882-8069
E-mail: huxleyv@missouri.edu

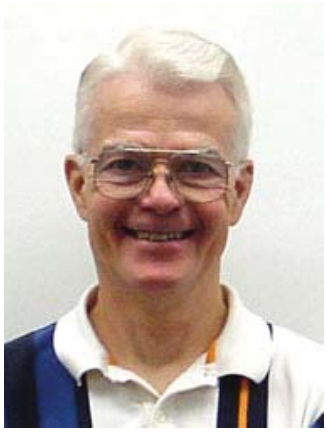
Education:
PhD University of Virginia, BA Hollins University

Appointments: Director of the National Center for Gender Physiology, J.O. Davis Chair of Cardiovascular Research , Professor, Department of Medical Pharmacology and Physiology, Adjunct Professor, Department of Biomedical Sciences

Research

Research Interests: Microvascular transport, in vivo imaging/microscopy, in vivo mass transfer, tissue engineering, mathematical modeling. We focus on the mechanisms controlling solute, water, and gas transfer between circulating blood and metabolizing tissue. Knowledge of the barriers to transport in living tissue is essential for elucidating the processes providing moment-to-moment regulation organ function and the etiology of dysfunctional states. These data facilitate design and implementation of rational strategies for treating diseases with the progressive goals of abating, arresting, and ultimately reversing disease processes.

Solute and water transfer results from "passive" and "cell mediated" mechanisms. Most have studied the passive mechanisms fewer study the cellular processes. From engaging in research in both arenas we find the exchange barrier to be a dynamic structure whose properties vary in time and space over time scales of seconds to days. Our global intent is to use this understanding of transport through pathways of microscopic geometry to investigate the relationship between blood supply and metabolic demand.
We are developing methods to extend these quantitative studies to mammalian microvessels in skeletal muscle, heart, gut, and brain in collaboration with colleagues in DCRC, MPP, Biomedical Sciences at MU and the University of Rochester. Knowing that microvascular exchange is subject to regulation under normal and pathological situations, we collaborate with clinical colleagues (Nephrology, Surgery, Anesthesia, Cardiology, Pulmonary Medicine, Infectious Disease, and Critical Care Medicine) to elucidate the cellular and molecular mechanisms involved in structuring and restructuring the barrier under conditions of peritoneal dialysis, following endurance exercise training, in coronary occlusive vessel disease, neurogenic edema, diabetes, cancer metastasis, and conditions of remote organ (brain) injury following burn. The combined clinical and basic science expertise provides the possibility of realizing our goal of designing and implementing treatments of permeability dysfunction. Consequent to the collaborative interactions at MU and in light of fundamental sex-related differences in physiological function and pathophysiology observed by us and others, we established the National Center for Gender Physiology. This virtual center is acting as a focus for research scientists at MU and collaborating institutions and augments the services provided by DCRC.



Allan Jones

Office Location: MA 415 Medical Sciences
Office Phone: 573-882-8029
E-mail: JonesA@missouri.edu

Education:
PhD University of Pennsylvania

Appointments: Professor Emeritus, Medical Pharmacology & Physiology

Research

Jones' research program currently focuses on mechanisms of membrane regulation and vascular smooth muscle function leading to abnormalities associated with hyper-lipidemia as well as adaptative mechanisms during exercise training. He is studying mechanisms of adenosine transport and adenosine regulation of smooth muscle responses to acute metabolic depression in porcine coronary arteries. These studies have shown a novel mechanism by which smooth muscle generated adenosine has an autocoid function during an ischemic response. Mechanisms being pursued relate to adenosine interaction with receptors and subsequent cellular events causing relaxation, as well as adenosine interaction with a target enzyme, AMP kinase, which in turn regulates both cell metabolism and functional responses. It has been observed that exercise training may alter the sensitivity of vascular smooth muscle in the porcine coronary arteries especially in males. Gender studies have also been initiated.

Ronald J. Korthuis

Office: MA415 HSC
Phone: 573-882-8059
E-mail: korthuisr@missouri.edu

Education:
PhD & BS from Michigan State University

Appointment: Chair Medical Pharmacology and Physiology
Professor, Department of Medical Pharmacology and Physiology
George L. and Melna A. Bolm Distinguished Chair in Cardiovascular Health

Research

Our research focuses on the mechanisms underlying the inflammatory responses to ischemia and reperfusion (I/R) and how blood vessels in the microcirculation (arterioles, capillaries and venules) can be preconditioned to resist the deleterious proinflammatory effects of I/R. When the blood supply is reduced (ischemia) and then subsequently reestablished (reperfusion), the ability of arterioles to regulate the distribution of blood flow is impaired, many capillaries fail to perfuse (capillary no-reflow), and white blood cells and platelets become adherent to and emigrate across the walls of postcapillary venules. Once in the tissues, these inflammatory phagocytes attack parenchymal cells, thereby exacerbating injury induced by ischemia. In addition, the permeability of the cells lining capillaries and postcapillary venules is increased, leading to edema formation. We are studying how white blood cells which adhere to and emigrate across the walls of postcapillary venules, alter vasoregulatory function in arterioles, cause no-reflow in capillaries, and increase permeability in postcapillary venules.

Laboratory models include study of the intact microcirculation using intravital microscopy coupled with computerized image analysis, examination of vasomotor responses in isolated arterioles, assessment of cell adhesion and permeability in single postcapillary venules, and evaluation of cell signaling mechanisms in cultured endothelial cells. A wide variety of mutant mouse models (gene knockout and transgenic overexpression), coupled with pharmacologic approaches, immunocytochemistry of the microvessel wall and cultured cells, western blotting and RT-qPCR to measure protein and mRNA expression, quantitative assessment of adhesion molecule expression using a dual radiolabeled monoclonal antibody technique, and three-dimensional fluorescence imaging using confocal microscopy are used to explore signaling mechanisms cultured cells, isolated microvessels, and intact microcirculatory networks in vivo.

A major area of emphasis over the last several years has been to understand how exposing tissues to preconditioning stimuli such as nitric oxide donors, calcitonin gene-related peptide, or by ingestion of ethanol (at doses equivalent to drinking one to two alcoholic beverages) 24 hours prior to the onset of prolonged ischemia followed by reperfusion prevents postischemic microvascular and parenchymal cell dysfunction. A fundamental aim of this work is to determine the signaling mechanisms that are activated in response to these preconditioning stimuli to upregulate gene expression for proteins that mediate the development of the anti-inflammatory and anti-thrombogenic phenotype in postischemic tissues.

Our future plans include continuing the examination of the mechanisms whereby microvascular function is protected in preconditioned states by focusing on the role of the extracellular matrix and cell adhesion molecules, ion channel function and extravascular constituents such as mast cells in these responses. In addition, we are exploring how leukocytes, which adhere almost exclusively in postcapillary venules in our models, alter the function of upstream arterioles in tissues exposed to ischemia and reperfusion.

M. Harold Laughlin

Office: E102VMED
Phone: 573-882-7011
E-mail: laughlinm@missouri.edu

Education: PhD University of Iowa, BA Simpson College

Appointments: Professor, Department of Biomedical Sciences, Adjunct Professor, Department of Medical Pharmacology & Physiology

Research

I focus my research on cardiorespiratory effects of exercise. The primary goal is understanding of the effects of exercise training on the coronary circulation and skeletal muscle vascular beds. Exercise training produces increases in the capacity of myocardial and skeletal muscle vascular beds to transport oxygen and other nutrients. The training induced changes in vascular transport capacity are associated with growth of new capillaries, enlargement of artieries and veins, and alterations in factos that control blood flow in the heart and skeletal muscle. The laboratory is currently investigating the mechanisms responsible for these changes. Studies are conducted with: isolated hearts, isolated muscle tissue, single blood vessels and in conscious, chronically instrumented animals during exercise. To allow examination of the relationships among vascular adaptations and the response of the myocytes to training induced increases in the functional demands of the muscles, the effects of training on biochemical and histological characteristics of the muscles are also measured. The biochemical systems examined include: the metabolic pathways involved in supplying the myocytes with ATP, the contractile proteins, the systems responsible for controlling intracellular Ca++ levels and endothelin nitric oxide synthase. Most of our current experiments are focused on endothelial cell biology. We are determining the effects of physical activity on endothelial phenotype in normal animals and in models of vascular disease. We are also using genetically modified pigs to examine the role of endothelial nitric oxide synthase in the impact of endothelial cell phenotype on vascular health.

Mark Milanick

Office Location: Ma415 Medical Science Bldg
Office Phone: (573) 882-8055
E-mail: MilanickM@missouri.edu

Education:
Swarthmore College, Pennsylvania B.A. Physics
University of St. Andrews, Scotland Math/Pharmacology
University of Chicago, Illinois Ph.D. Biophysics
Yale University, Connecticut postdoctoral Physiology

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research Interests

Erythrosensors, Approaches for resource poor areas, Educational Innovations, Membrane Transport kinetics

Research Description

Erythrosensors
Our long term goal is to encapsulate glucose sensitive near infrared fluorescent dye inside red blood cells (erythrocytes). Return red cells to patient. Patient can monitor glucose levels non-invasively using a pulse oximeter type of detector for half of the lifetime of the red cells. Since the red cells live about 100 days, this means the erythrosensors only need to be made and injected about every 2 months.
This project is being done in collaboration with Tim Glass, Xiaole Shao, and Nick Cooley (Chemistry, MU) and Ken Meissner and Sarah Ritter (Bioengineering, Texas A&M).
Engineering erythrocytes to be erythrosensors: first steps. Milanick MA, Ritter S, Meissner K.
Blood Cells Mol Dis. 2011 Aug 15;47(2):100-6.
Encapsulation of FITC to monitor extracellular pH: a step towards the development of red blood cells as circulating blood analyte biosensors. Ritter SC, Milanick MA, Meissner KE. Biomed Opt Express. 2011 Jul 1;2(7):2012-21.

Approaches for resource poor areas

1. Soymilk: an effective and inexpensive blocking agent for immunoblotting. Galva C, Gatto C, Milanick M. Anal Biochem. 2012 Jul 1;426(1):22-3.
2. We are developing dipsticks for measuring levels of stress markers, including salivary cortisol and amylase for home use, as well as for detection of pesticides.
3. We have developed an educational laboratory exercise examining enzyme activity using acetylcholinesterase activity from grocery store frozen fish.
Why is that dog paralyzed? A problem-based case & laboratory exercise about neuromuscular transmission. Milanick, M., Graham, K. & Wessel, M. (2013). American Biology Teacher, 75, 36–39.

Educational innovations

1. We have published several education articles that use novel approaches to interest students in various scientific activities.

- Fact or Fiction? General Chemistry Helps Students Determine the Legitimacy of Television Program Situations. Mark A. Milanick* and Ruth L. Prewitt J. Chem. Educ., April 19, 2013
- Units, Jargon, G-forces and Squirting Blood. Milanick, MA. The Physics Teacher 50, 410, 2012.
- Changes of membrane potential demonstrated by changes in solution color. Milanick M. Adv Physiol Educ. 2009 Sep;33(3):230.
- Fluorescence Using Turmeric Mark A. Milanick J. Chem. Educ., 2011, 88 (3), pp 260.

2. We have developed novel interactive course offerings, including

- Ethics Education through Enactment, Engagement and Empowerment (Graduate)
- The Science of Sex, Drugs, and Rock'n'Roll (Undergraduate, non-majors)
- Clinical Biodetection (Graduate)

Membrane Transport Kinetics

We were funded for about 18 years by NIH for studies on membrane transport. Some of the highlights include

- Eosin, a Potent Inhibitor of the Plasma Membrane Ca Pump, Does Not Inhibit the Cardiac Na-Ca Ex changer
- Kinetic characterization of tetrapropylammonium inhibition reveals how ATP and Pi alter access to the Na+-K+-ATPase transport site
- Extracellular protons regulate the extracellular cation selectivity of the sodium pump.
- Probing the extracellular release site of the plasma membrane calcium pump.
- Na-Ca exchange: evidence against a ping-pong mechanism and against a Ca pool in ferret red blood cells.
- Proton fluxes associated with the Ca pump in human red blood cells.
- Na-Ca exchange in ferret red blood cells.
- Proton inhibition of chloride exchange: asynchrony of band 3 proton and anion transport sites?
- Proton-sulfate co-transport: mechanism of H+ and sulfate addition to the chloride transporter of human red blood cells.

Nicole L. Nichols, PhD

Office: Veterinary Medicine Building W145
Lab: Veterinary Medicine Building W146-W151
Phone: 573-882-2534 (office); 573-882-3715 (lab)
Email: nicholsn@missouri.edu

Education:
PhD: Wright State University
Postdoctoral Training: University of Wisconsin-Madison (Prof. Gordon S. Mitchell)

Research

Understanding how plasticity can be enhanced in models of motor neuron death to restore breathing

Our laboratory focuses on the central nervous system, in particular the control of breathing in models of motor neuron death. Breathing is essential to life and cannot cease for more than the briefest periods, or life will not continue. At the same time, breathing must be continuously adjusted throughout life to maintain homeostasis in response to physiological (for example exercise, pregnancy or high altitude) or pathological (for example disease or disorder) situations. One way the neural system controlling breathing maintains homeostasis is to express plasticity which is defined as a persistent change in the neural control system based on a prior experience. A well-known model of respiratory plasticity is phrenic long-term facilitation (pLTF), a long-lasting increase in phrenic motor output elicited by acute exposure to intermittent hypoxia. Although, we know a great deal about the mechanism that underlies pLTF under normal circumstances, the mechanism that underlies pLTF in models of motor neuron death is not well understood. Determining the mechanism that underlies pLTF and how it can be enhanced in models of motor neuron death to restore breathing is the focus of the laboratory.

We utilize a multidisciplinary approach to elucidate these mechanisms. These include: 1) novel pharmacological injections to induce models of motor neuron death; 2) whole animal plethysmography to measure respiration in unanesthetized animals; 3) in vivo neurophysiology to measure spontaneous nerve output and nerve output in response to targeted drug delivery; and 4) immunohistochemical localization of neurotransmitter receptors and proteins of interest on individual neurons, astrocytes and microglia. Using these techniques, we have recently developed a novel model of motor neuron death that mimics aspects of ALS (amyotrophic lateral sclerosis) related to ventilatory function. Further, using this model, we can study the mechanism that underlies pLTF and how this plasticity can be enhanced following motor neuron death.

Jaume Padilla, Ph.D.

Office: 306 Gwynn Hall
Office: 573-882-7056
Email: padillaja@missouri.edu

Appointment: Assistant Professor Nutrition & Exercise Physiology

Research Description

Dr. Padilla's research primarily focuses on understanding the physiological and molecular mechanisms by which physical inactivity and obesity-associated insulin resistance leads to impaired vascular function. A particular area of interest is studying the effects of adipose tissue-derived cytokines, and their interactive effects with hemodynamic forces (e.g., shear stress), in modulating vascular cell phenotype and function. Dr. Padilla's research is integrative and incorporates in vitro cell and tissue culture models, in vivo studies in small and large animals, and experiments in humans.

Leona J. Rubin

Office Location: 210 Jesse Hall
Office Phone:(573) 884-1402
RubinL@missouri.edu

Education:
BA Temple University.
MS Rutgers University.
PhD University of Colorado Health Sciences Center.

Appointment: Associate Vice Chancellor for Graduate Studies & Associate Vice President of Academic Affairs & Graduate Education

Research

Dr. Rubin is a basic scientist whose research has always focused on understanding cellular signaling pathways. She began her research career at MU exploring second messenger signaling involved in control of cardiovascular function. Rodent models were employed to explore how immune mediators, released during inflammatory conditions (endotoxemia/sepsis) cause myocardial failure and/or vascular dysfunction through modulation of the activity of specific cellular targets such as potassium and calcium channels or contractile proteins. More recent work explored the signaling mechanism impacted by specific therapeutic modalities such as the anesthetic, ketamine which appears able to protect cardiovascular function during inflammatory states. Related studies utilized a swine model of atherosclerosis and the influence of gender to determine whether exercise had beneficial effects on receptor mediated signaling pathways and function of coronary smooth muscle. Studies that explored cellular signaling pathways involved in vascular metabolic vasodilation which is essential to match blood flow to tissue energy demands during exercise or disease impairment led the research to examine the role of AMP kinase, then a novel signaling pathway. Dr. Rubin's studies were the first to explore the role of AMPK in vascular smooth muscle function and metabolic vasodilation. These studies continue in the laboratory with the addition of the AMPK knock out mouse model. A serendipitous finding for this model was an interaction between AMPK alpha-1 KO and the C57Bl6 mouse strain which presents with significant cardiac hypertrophy that resembles physiologic hypertrophy. Current and future studies are directed at understanding the signaling pathway impacted by this interaction to better understand the cellular pathways that underlie exercise and disease-induced cardiac hypertrophy.

Steven S. Segal

Office: MA 415 HSC
Phone: 573-882-2553
E-mail: segalss@health.missouri.edu

Education:
PhD University of Michigan,
MA & BA University of California, Berkley

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

Our research is focused on understanding how oxygen delivery increases in response to metabolic demand. During exercise, the recruitment of skeletal muscle fibers (motor units) generates electrical and chemical signals in endothelial cells and smooth muscle cells of the microvessels that control the distribution and magnitude of muscle blood flow. Our experiments center on elucidating the cellular and molecular events which initiate these signals, how such signals are transmitted from cell to cell to orchestrate vasodilation and vasoconstriction in microvascular networks, and how these integrative processes are governed by the nervous system. Intravital video microscopy enables direct observations of blood flow control in the mammalian microcirculation. Histochemistry and vascular casting are used to quantify the architecture of neural and microvascular networks. Intracellular recording with dye labeling reveals cell-specific electrical signals which determine the contractile status of smooth muscle and its regulation by the endothelium. Calcium imaging provides unique insight into cellular responses which reflect the activity of ion channels and key regulatory enzymes. Complementary studies of isolated microvessels and their constitutive cells enable even greater resolution of specific regulatory processes. Pharmacology, immunolabeling, and Real-Time Polymerase Chain Reaction are used to resolve the functional expression of proteins which mediate cell-to-cell coupling through gap junctions and electrical signaling through ion channels.

Collaborative studies using transgenic mice afford unique insight into how particular signaling pathways affect control processes within the microcirculation. In turn, these basic relationships are athways affect control processes within the microcirculation. In turn, these basic relationships are being explored in light of how aging affects microvascular structure and function. Opportunities for graduate and postdoctoral training include: molecular physiology of vascular cells, electrical and optical monitoring of cell signaling, microsurgery and microdissection, intravital video microscopy, conventional and immunohistochemistry, evaluation of gene expression, and modeling the biophysical properties of cells and tissues.

Jim R. Sowers

Office: D109 Diabetes Center UHC
Phone: 882-0999
E-mail: sowersj@missouri.edu

Education:
MD University of Missouri-Columbia,
BS Central Missouri State University

Appointment: Director of the MU Diabetes and Cardiovascular Center, Professor, Department of Medicine, Professor, Department of Medical Pharmacology and Physiology

Research

Fifty million people have high blood pressure and are prone to developing Type 2 diabetes. Dr. Sowers, directs the MU Diabetes and Cardiovascular Center and is associate dean for clinical research. His work addresses the link between high blood pressure and diabetes to better understand how to prevent and cure the diseases, which are growing problems in the United States.

International Investigators

Nicola J. Brown, Ph.D.

Adjunct Dalton Investigator
Office Location: Department of Oncology, University of Sheffield, S10 2RX
Office Phone: 0114 2712789
N.J.Brown@sheffield.ac.uk

Education:
PhD: University of Sheffield
BS: University of Sheffield

Appointments: Professor of Microcirculation Biology, Head of Academic Surgical Oncology Unit, Head of Microcirculation Research Group, Department of Oncology, Faculty of Medicine Dentistry and Health

Research

My research interests are mechanisms of physiological and pathophysiological angiogenesis in wound healing and tumour progression and the role of anti-angiogenic and anti-vascular strategies for the treatment of angiogenesis dependent disorders.
The principal objective and research strategy of the Microcirculation Research Group is to investigate the mechanisms regulating tumour angiogenesis in preinvasive to invasive cancer progression and how this may be targeted for therapy. The facility contains state-of-the-art specialised fluorescent in vivo microscopy and multiphoton microscopy which allows real-time imaging of blood vessel development, blood flow, leucocyte-endothelial and tumour-endothelial interactions, in a variety of preclinical in vivo models, in addition to a panel of in vitro angiogenesis assays. The clinical study of human tissue, both normal and breast cancer are complemented by laboratory based modeling, both basic and applied.

Yoshiro Sohma

Office: 263 DCRC
Phone: 573-882-0938
E-mail: somay@missouri.edu

Education:
PhD and MD Osaka Medical College

Appointment: Visiting Professor, Department of Medical Pharmacology & Physiology

Research

I have a broad research interest that covers the molecular physiology and biophysics of ion channels and transporters, and their role in the physiological function of cells/tissue. I have studied the permeation and gating of a large-conductance, Ca2+-activated, voltage-dependent potassium (BKCa) channel. BKCa channels are ubiquitously expressed in many different tissues and are known to be sensitive to membrane potential, intracellular calcium, magnesium, pH. These channels are also activated by CO and sensitive to O2 associated with heme-oxygenase2 and, moreover, some slice-variants respond to membrane stretch. Based on these findings, BKCa channels are suggested to play an important role in integrating cell signals, a recently proposed new function of an ion channel. I am also involved in a Japanese-based collaborative project undertaking a comprehensive study of the GABAergic system in peripheral tissues (not the central nervous system) and work on GABAA receptor channels and GABAB-mediated modulation of K+ and Ca2+ channels in peripheral tissues. In the Dalton Cardiovascular Research Center, I study the molecular mechanism of gating and permeation of the CFTR chloride channel, a member of the ATP-Binding Cassette (ABC) transporter superfamily, in collaboration with Dr. Tzyh-Chang Hwang. Our work has made a significant contribution to the recent advances in the biophysical understanding of the ATP-dependent gating mechanism in CFTR, and has provided great insight into a possible common functional mechanism that can be applied to the whole ABC transporter superfamily. The structure of the membrane spanning domain (MSD) in CFTR is known to be similar to that in the P-glycoprotein drug efflux pump. We therefore believe that understanding MSD structure/function in CFTR will lead to a better understanding of multi-drug resistance (MDR) proteins. In addition to investigating the biophysics of the CFTR molecule itself, I am also interested in studying the functional interactions of CFTR with other membrane proteins and lipids. This should help in our understanding of the complicated regulatory mechanisms that underlie physiological functions in cell membrane. I have also employed computer modeling to simulate ion transport processes in epithelial cells (e.g., bicarbonate transport in pancreatic duct cells). This approach is very useful to bridge information from molecular biophysics and cell/tissue physiology to the research field of epithelial transport. I believe that by taking such a general and comprehensive approach to the study of different channels/transporters and channel/transporter-mediated physiological systems, induces a 'positive cooperative effect' which accelerates each research project, and which also gives us a novel scientific standing point of view for the channel sciences.

APPENDICES

PUBLICATIONS

PRESENTATIONS

SEMINARS

ACTIVE GRANTS & CONTRACTS

PROFESSIONAL SERVICE ACTIVITIES

Christopher Baines

Publications:

Gutierrez-Aguilar M, Baines CP. Structural Mechanisms of Cyclophilin D-Dependent Control of the Mitochondrial Permeability Transition Pore. Biochim Biophys Acta. 2015; 1850:2041-7.

Presentations:

10/15/15: "Using Proteomics to Understand Cardiac Myocyte Necrosis", First Annual Midwest Bioinformatics Symposium, University of Missouri Kansas City, Kansas City, MO.

7/17/15: "Molecular Composition of the Mitochondrial Pore", Symposium on Advances in the Biochemistry of Ischaemia and Hypoxia, University of Bristol, Bristol, Great Britain.

6/8/15: "The mitochondrial protein C1qbp binds to cyclophilin D and ATP synthase and regulates the mito chondrial permeability transition", International Society for Heart Research, North American Section meeting, Seattle, WA.

Active Grants:

R01HL094404 (PI: Baines) 12/2013-11/2018
NIH/NHLBI $1,250,000 direct costs
"Molecular Identity of The Cardiac Mitochondrial Pore"
Role: PI

Research Contract (PI: Baines) 6/2015-6/2016
Gilead Inc. $31,225 direct costs
"Cyclophilin and ASK1 inhibitors in regulation of mitochondrial permeability transition and cell death"
Role: PI

R01 (PI: Ding) 9/2015-8/2020
NIH/NINDS $1,250,000 direct costs
"Neuroprotection and mechanisms of PBEF in Cerebral Ischemia"
Role: Co-I

Professional Services

(Spr.): MIM/BioSci-9432 Molecular Biology II (2 lectures, 13 students)
(Spr): Veterinary Research Scholars Program (Co-Director, 39 students)
(Fall): V_BSCI-5506 Veterinary Cell Biology (Director, 17 lectures, 120 students)

Post Doctoral Supervisor
PhD Dissertation Advisor
PhD Dissertation Committee Member

Member, Biomedical Sciences Chair Search Committee
Member, Academic Research Advisory Board, College of Veterinary Medicine

Reviewer, 2015 Excellence in Education Awards
Member, 23rd Annual Cardiovascular Day Planning Committee
Member, Early Career Committee, Basic Cardiovascular Sciences Council, American Heart Association
Chair, Ischemia, Cardioprotection & Mitochondria Interest Group, North American Section of the International Society for Heart Research

Award Grading
American Heart Association, Scientific Sessions 2015.
Judge, Senior Young Investigator Award, North American Section of the International Society for Heart Research Annual Meeting.
American Heart Association, Scientific Sessions 2015.

Symposium Chair
"Interest Group 5 Session – Ischemia and Mitochondria", International Society for Heart Research North American Section meeting, Seattle, WA, June.

Grant Reviews:
2015: NIH Myocardial Ischemia and Metabolism (MIM) Study Section, Ad Hoc.
NIH Membrane Biology and Protein Processing (MBPP) Study Section, Mail Reviewer.

Professional Memberships:
American Heart Association
American Physiological Society
American Society for Biochemistry and Molecular Biology
Biophysical Society
International Society for Heart Research

Shawn Bender

Publications

Mueller KB, Bender SB, Hong K, Yang Y, Aronovitz M, Jaisser F, Hill MA, Jaffe IZ. Endothelial mineralocorticoid receptors differentially contribute to coronary and mesenteric vascular function without modulating blood pressure. Hypertension 66(5): 988-997, 2015.

Bender SB*, Castorena-Gonzalez JA*, Garro M, Reyes-Aldosoro CC, Sowers JR, DeMarco VG, Martinez-Lemus LA. Regional variation in arterial stiffening and dysfunction in western diet-induced obesity. Am J Physiol Heart Circ Physiol 309(4): H574-H582, 2015.
*Authors contributed equally to this work (joint first-authorship)
*Selected for Editorial Focus: Gooch KJ & Trask AJ. Tissue-specific vascular remodeling and stiffness associated with metabolic diseases. Am J Physiol Heart Circ Physiol 309(4): H555-H556, 2015.

DeMarco VG, Habibi J, Jia G, Aroor AR, Ramirez F, Martinez-Lemus LA, Bender SB, Garro M, Hayden MR, Sun Z, Meininger GA, Manrique C, Whaley-Connell A, Sowers JR. Low dose mineralocorticoid receptor blockade prevents western diet-induced arterial stiffening in female mice. Hypertension 66(1): 99-107, 2015.

Bostick B, Habibi J, DeMarco VG, Jia G, Domeier TL, Lambert MD, Aroor AR, Nistala R, Bender SB, Garro M, Hayden MR, Ma L, Manrique C, Sowers JR. Mineralocorticoid receptor blockade prevents western diet-induced diastolic dysfunction in female mice. Am J Physiol Heart Circ Physiol 308: H1126-H1135, 2015.
*Selected for Journal Podcast Series (at http://ajpheart.podbean.com – posted 4-7-2015)

Bender SB, DeMarco VG, Padilla J, Jenkins NT, Habibi J, Garro M, Pulakat L, Aroor AR, Jaffe IZ, Sowers JR. Mineralocorticoid receptor antagonism treats obesity-associated diastolic dysfunction. Hypertension 65: 1082-1088, 2015.
*Selected for Clinical Implications Section: Hypertension 65: 925, 2015.

Presentations

2015 – Annville to Columbia: a journey into academic cardiovascular research
Lebanon Valley College Department of Biology, Annville, PA

Active Grants

CDA-2 IK2 BX002030, Career Development Award-2, Dept of Veterans Affairs BLR&D (PI, 7/1/2013-6/31/2018); Mineralocorticoid receptor-mediated vascular insulin resistance. Total Award Amount: $891,620.

Professional Services

Grant Review
University of Missouri Research Board 2014-
American Heart Association Vascular BioBP BSci 3 Peer Review Committee Fall 2015-
Editorial Activities
2010 – Editorial Board (Review Editor) – Frontiers in Exercise Physiology
2011 – Trainee Advisory Consulting Editor – Journal of Applied Physiology

2011 – Editorial Board – CardioRenal Medicine
2014 – Guest Editor – American Journal of Physiology: Heart and Circulatory Physiology
Reviewer for Scientific Journals
American Journal of Cardiology
American Journal of Physiology – Endocrinology & Metabolism
American Journal of Physiology – Heart & Circulatory Physiology
American Journal of Physiology – Regulatory, Integrative and Comparative Physiology
Basic Research in Cardiology
British Journal of Pharmacology
CardioRenal Medicine
Cardiovascular Research
Diabetes
Endocrinology
European Journal of Applied Physiology
Experimental Biology and Medicine
Experimental Physiology
Journal of Applied Physiology
Journal of Cardiothoracic Surgery
Journal of Cardiovascular Pharmacology
Journal of Vascular Research
Journal of Visualized Experiments
Life Sciences
Medicine & Science in Sports & Exercise
Microcirculation
Microvascular Research
Molecular and Cellular Endocrinology
Obesity
Pflügers Archiv – European Journal of Physiology
Pharmacological Reports
Physiological Genomics
PLOS ONE
Vascular Pharmacology

2015- Vice Chair, Research & Development Committee, Truman VA Hospital

Frank Booth

Publications

Rapid Alterations in Perirenal Adipose Tissue Transcriptomic Networks with Cessation of Voluntary Running. Ruegsegger GN, Company JM, Toedebusch RG, Roberts CK, Roberts MD, Booth FW. PLoS One. 2015 Dec 17;10(12):e0145229. doi: 10.1371/journal.pone.0145229. eCollection 2015. PMID: 26678390

Effects of ovariectomy and intrinsic aerobic capacity on tissue-specific insulin sensitivity. Park YM, Rector RS, Thyfault JP, Zidon TM, Padilla J, Welly RJ, Meers GM, Morris ME, Britton SL, Koch LG, Booth FW, Kanaley JA, Vieira-Potter VJ. Am J Physiol Endocrinol Metab. 2015 Dec 8:ajpendo.00434.2015. doi: 10.1152/ajpendo.00434.2015. [Epub ahead of print] PMID: 26646101

Comparative adaptations in oxidative and glycolytic muscle fibers in a low voluntary wheel running rat model performing three levels of physical activity. Hyatt HW, Toedebusch RG, Ruegsegger G, Mobley CB, Fox CD, McGinnis GR, Quindry JC, Booth FW, Roberts MD, Kavazis AN. Physiol Rep. 2015 Nov;3(11). pii: e12619. doi: 10.14814/phy2.12619. Epub 2015 Nov 24. PMID: 26603455

AMPK-agonist AICAR delays the initial decline in lifetime-apex VO2peak while voluntary wheel running fails to delay its initial decline in female rats. Toedebusch RG, Ruegsegger GN, Braselton JF, Heese AJ, Hofheins J, Childs TE, Thyfault JP, Booth FW. Physiol Genomics. 2015 Nov 17:physiolgenomics.00078.2015. doi: 10.1152/physiolgenomics.00078.2015. [Epub ahead of print] PMID: 26578698

Western diet-induced hepatic steatosis and alterations in the liver transcriptome in adult Brown-Norway rats. Roberts MD, Mobley CB, Toedebush RG, Heese AJ, Zhu C, Krieger AE, Cruthirds CL, Lockwood CM, Hofheins JC, Wiedmeyer CE, Leidy HJ, Booth FW, Rector RS. BMC Gastroenterol. 2015 Oct 30;15:151. doi: 10.1186/s12876-015-0382-3. PMID: 26519296

Endurance Exercise and the Regulation of Skeletal Muscle Metabolism. Booth FW, Ruegsegger GN, Toedebusch RG, Yan Z. Prog Mol Biol Transl Sci. 2015;135:129-51. doi: 10.1016/bs.pmbts.2015.07.016. Epub 2015 Sep 5. PMID: 26477913

Reduced metabolic disease risk profile by voluntary wheel running accompanying juvenile Western diet in rats bred for high and low voluntary exercise. Ruegsegger GN, Toedebusch RG, Braselton JF, Roberts CK, Booth FW. Physiol Behav. 2015 Dec 1;152(Pt A):47-55. doi: 10.1016/j.physbeh.2015.09.004. Epub 2015 Sep 11. PMID: 26367453

Understanding the Cellular and Molecular Mechanisms of Physical Activity-Induced Health Benefits. Neufer PD, Bamman MM, Muoio DM, Bouchard C, Cooper DM, Goodpaster BH, Booth FW, Kohrt WM, Gerszten RE, Mattson MP, Hepple RT, Kraus WE, Reid MB, Bodine SC, Jakicic JM, Fleg JL, Williams JP, Joseph L, Evans M, Maruvada P, Rodgers M, Roary M, Boyce AT, Drugan JK, Koenig JI, Ingraham RH, Krotoski D, Garcia-Cazarin M, McGowan JA, Laughlin MR. Cell Metab. 2015 Jul 7;22(1):4-11. doi: 10.1016/j.cmet.2015.05.011. Epub 2015 Jun 11. Review. PMID: 26073496

Mu opioid receptor modulation in the nucleus accumbens lowers voluntary wheel running in rats bred for high running motivation. Ruegsegger GN, Toedebusch RG, Will MJ, Booth FW. Neuropharmacology. 2015 Oct;97:171-81. doi: 10.1016/j.neuropharm.2015.05.022. Epub 2015 Jun 1. PMID: 26044640

The erosion of physical activity in Western societies: an economic death march. Booth FW, Hawley JA. Diabetologia. 2015 Aug;58(8):1730-4. doi: 10.1007/s00125-015-3617-5. Epub 2015 May 8. PMID: 25952481

Postdinner resistance exercise improves postprandial risk factors more effectively than predinner resistance exercise in patients with type 2 diabetes. Heden TD, Winn NC, Mari A, Booth FW, Rector RS, Thyfault JP, Kanaley JA. J Appl Physiol (1985). 2015 Mar 1;118(5):624-34. doi: 10.1152/japplphysiol.00917.2014. Epub 2014 Dec 24. PMID: 25539939

Physiology of sedentary behavior and its relationship to health outcomes. Thyfault JP, Du M, Kraus WE, Levine JA, Booth FW. Med Sci Sports Exerc. 2015 Jun;47(6):1301-5. doi: 10.1249/MSS.0000000000000518. PMID: 25222820 Active Grants

Professional Services
Editorial Board, Journal of Applied Physiology, 1980-1993; 1996-1999; 2005-present
Editorial Board, American Journal of Physiology: Cell Physiology, 1994-present

SERVICE ON DEPARTMENTAL COMMITTEES
Departmental Faculty Promotions and Tenure Committee 2008-present
Editorial Board, American Journal of Physiology: Regulatory, integrative and Comparative Physiology
Editorial Board, Physiological Genomics, 2005-present
Editorial Board, Section III: Health and Disease, Scandinavian Journal of Medicine and Science in Sports, 2006-present

SERVICE ON COLLEGE OF VETERINARY MEDICINE (1999-PRESENT) COMMITTEES
Faculty Responsibility Committee, 2005-present

SPONSORSHIP OF CANDIDATES FOR POSTGRADUATE DEGREE

SPONSORSHIP OF POSTDOCTORAL FELLOWS

Douglas Bowles

Publications

Kilroy, J.P., A.H. Dhanaliwala, A.L. Klibanov, D.K. Bowles, B.R. Wamhoff and J.A. Hossack. Reducing Neointima Formation in a Swine Model with IVUS and Sirolimus Microbubbles. Annals of Biomed. Eng. 2015 Apr 17 [Epub ahead of print] PMID:25893508

Masseau, I. and D.K. Bowles. Carotid endothelial VCAM-1 is an early marker of carotid atherosclerosis and predicts coronary artery disease in swine. Journal of Biomedical Science and Engineering. 8:11, 2015.

Active Grants

Mizzou Advantage (Fay, PI; Bowles, Co-I) 4/1/10-3/31/15 2.5%
MU $50,000 ADC
Targeting Plasminogen Activator inhibitor-1 to Inhibit Neointimal Hyperplasia
Major goals: Develop a novel DES

T32 RR007004 (Franklin, PI; Bowles, Mentor) 7/1/11-6/30/16 *2%
NIH/NCRR $321,084 ADC
Postdoctoral Training in Comparative Medicine
Major goals: The major goal of this project is to provide graduate research training in comparative medicine

Presentations

Hay, M., M.T. Hamilton, A.K. Johnson, B. Xue and D.K. Bowles. Estradiol Effects on Inflammation Related Gene Expression in the Nucleus Tractus Solitarius. FASEB J. 21(5): 2015.

Yang, H.T., D.K. Bowles, M.H. Laughlin and R.L. Terjung. Prolonged walking attenuates hypersympathetic drive in swine with bilateral femoral artery occlusion. FASEB J. 21(5): 2015.

Tharp, D.L. and D.K. Bowles. KCa3.1 inhibition decreases size and alters composition of atherosclerotic lesions induced by low, oscillatory flow. FASEB J. 21(5): 2015.

Active Grants

*UM Research Board Grant (Bowles, PI) 1/1/15-12/31/15 *8%*
UM Office of Research $51,000 TDC
KCa3.1 in smooth muscle migration and atherosclerosis
Major goals: Determine the role of KCa3.1 in determining VSM migration and atherosclerotic lesion size and composition.
Role: PI

*CVM/COR Faculty Research Grant (Bowles, PI) 2/1/15-1/31/16*20%*
MU CVM $18,000 TDC
Contribution of vascular smooth muscle KCa3.1 to atherosclerosis.
Major goals: Use partial carotid ligation in a KCa3.1 transgenic mice to study the role of KCa3.1 in atherosclerosis.
Role: PI

Professional Service
National
Manuscript Review

Reviewer, Biology Open
Reviewer, PLOSOne
Reviewer, BioMed Research International
Reviewer, Hypertension
Reviewer, Atherosclerosis, Thrombosis and Vascular Biology
Reviewer, Circulation Research
Reviewer, Journal of Applied Physiology
Reviewer, Applied Physiology, Nutrition, and Metabolism

International
Editorial boards

Editorial Board of Scientifica

2015-present	Chair, Biomedical Sciences, University of Missouri
2013-2015	Associate Chair, Biomedical Sciences, University of Missouri
2010-present	Professor, Biomedical Sciences, University of Missouri
2010-present	Professor, Medical Pharmacology and Physiology, University of Missouri
2005-present	Director, MU Research Cath Lab, University of Missouri
1998-present	Investigator, Dalton Cardiovascular Research Center, University of Missouri

Nicola Brown

Publications

Is Breast Pain Greater in Active Females Compared to the General Population in the UK? Brown N, Burnett E, Scurr J. Breast J. 2015 Dec 14. doi: 10.1111/tbj.12547. [Epub ahead of print] PMID: 26661830

"Breasts are getting bigger". Where is the evidence? Brown N, Scurr J. J Anthropol Sci. 2015 Dec 11. [Epub ahead of print] No abstract available. PMID: 26656956

The prevalence and impact of heavy menstrual bleeding among athletes and mass start runners of the 2015 London Marathon. Bruinvels G, Burden R, Brown N, Richards T, Pedlar C. Br J Sports Med. 2015 Nov 26. pii: bjsports-2015-095505. doi: 10.1136/bjsports-2015-095505. [Epub ahead of print] No abstract available. PMID: 26612843

Professional Service

Medical Research Council Clinical Training & Fellowship Panel 2006 –
Editorial Board Journal of Vascular Research 2005 -
Photochemistry and Photobiology 2003-

Current Projects

Mechanisms of angiogenesis in preinvasive and invasive breast cancer
Role of neural guidance molecules in physiological and pathophysiological angiogenesis
Vascular targeting in breast cancer and sarcomas - funded by the Breast Cancer Campaign and Yorkshire Cancer Research
Role of angiogenesis in bone metastasis - funded by EU Framework VI consortium PROMET and Yorkshire Cancer Research
Vascular targeting and imaging - funded by Cancer Research UK/EPSRC/DOH
Role of stress proteins in the breast tumour microenvironment - funded by the Breast Cancer Campaign

Chandrasekar Bysani

Publications

Somanna NK, Yariswamy M, Garagliano JM, Siebenlist U, Mummidi S, Valente AJ, Chandrasekar B. Aldosterone-induced cardiomyocyte growth, and fibroblast migration and proliferation are mediated by TRAF3IP2. Cell Signal. 2015 Oct;27(10):1928-38.

Somanna NK, Valente AJ, Krenz M, Fay WP, Delafontaine P, Chandrasekar B. The Nox1/4 dual inhibitor GKT137831 or Nox4 knockdown inhibits Angiotensin-II-induced adult mouse cardiac fibroblast proliferation and migration. AT1 physically associates with Nox4. J Cell Physiol. 2015 (in press; PMID: 26445208).

Presentations

May 22, 2015. TRAF3IP2 in cardiac ischemic injury. Cardiology, UTHSCSA, San Abtonio, TX
June 18, 2015. RECK and adverse cardiac remodeling. Cardiology, University of Alabama, Birmingham.

Active Grants

VA Merit Review
(PI: Chandrasekar) 07/01/2014-6/30/2018 6 months
Veterans Administration $125,000/yr
Title: Interleukin-18 and post-infarct cardiac remodeling
The goal of this proposal is to investigate the causal role of interleukin-18 in post-infarct cardiac remodeling using cardiac-specific IL-18 knockout and cardiac-restricted IL-18 overexpressing mice
Overlap: None

1ROI HL086787
(PI: Chandrasekar) 12.1.2009-2.28.2015
(includes no-cost extension) 6 months
NIH/NHLBI $250,000/yr
Title: The role of Interleukin-18 in Myocardial Hypertrophy and Failure
The goal of this study is to investigate the causal role of IL-18 in pressure-overload induced myocardial hypertrophy and failure using loss-of-function and gain-of-function animal models
Overlap: None

Professional Services

EDITORIAL CONSULTANT

American Journal of Physiology, American Journal of Pathology, Arteriosclerosis, Thrombosis & Vascular Biology, Circulation, Cardiovascular Research, Cytokine, Journal of Molecular and Cellular Cardiology, Journal of Biological Chemistry
Oct 2009-to date Professor with tenure, Heart and Vascular Institute, Tulane University School of Medicine, New Orleans, LA
Oct 2009- to date Research Health Scientist, Southeast Louisiana Veterans Health Care System, New Orleans, LA
March 2009-to date Research Administrator, Southeast Louisiana Veterans Health Care System, New Orleans, LA
July 1, 2011- to date: Member, R & D Committee at Southeast Louisiana Veterans Health Care System – 629
September 2011 to date: Chair, IACUC at Southeast Louisiana Veterans Health Care System – 629

Lane Clarke

Publications

Liu, J, Walker, NM, Ootani, A, Strubberg, AM, Clarke, LL. Defective goblet cell exocytosis contributes to murine cystic fibrosis-associated intestinal disease. J. Clin. Invest. 125: 1056-1068, 2015.

Walker, NM, Liu, J, Stein, SR, Stefanski, CD, Strubberg, AM, Clarke, LL. Cellular chloride and and bicarbonate retention alters intracellular pH regulation in Cftr KO crypt epithelium. Am. J. Physiol. First published November 5, 2015; doi:10.1152/ajpgi.00236.2015

Presentations

Increased Functional AE2 Activity Does Not Compensate for Increased pHi in Cftr Knockout Enteroids, 115th

Symposium Speaker: Goblet Cell Dysfunction and Mucus Retention in the Cftr KO Intestine, 1st Cystic Fibrosis Foundation Research Conference: Pushing the Frontiers, Chantilly, VA. June 1, 2015.

Symposium Speaker: Human CFTR Mouse, Animal Models for CF Research and Drug Development, 1st Cystic Fibrosis Foundation Research Conference: Pushing the Frontiers, Chantilly, VA. June 1, 2015.

Abnormal Goblet Cell Function and Mucus Retention in the Cftr KO Intestine, Cystic Fibrosis Research Center, Department of Medicine, University of North Carolina-Chapel Hill, NC. April 24, 2015

Goblet Cell and Enterocyte Dysfunction in Intestinal Organoids from Cystic Fibrosis Mice, Department of Biomedical Sciences, University of Missouri, Columbia, MO. November 11, 2015.

Goblet Cell Dysfunction in Intestinal Organoids from Cystic Fibrosis Mice, Department of Anatomy and Physiology, Kansas State University, Manhattan, KS. November 16, 2015

Active Grants

National Institutes of Health R01 DK48816 – Years 15-19; "CFTR and Acid-Base Transporters in Regenerating Intestinal Crypts", 04/01/12-03/31/16 $1,518,290, PI.

Cystic Fibrosis Foundation; "Defective Goblet Cell Degranulation in Cystic Fibrosis Enteroids"; 04/01/2013-03/31/2015; $86,600, Sponsor.

Mizzou Advantage (Round 4); "Targeting Cell pH/Volume to Minimize Chemo-/Radiotherapy-Induced Intestinal Damage", 07/01/14-06/30/15, $58,000, Seed Grant, PI.

Phi Zeta Research Grant; "The Effect of High Bicarbonate on Small Intestinal Nutrient Absorption in a Cystic Fibrosis Mouse Model", 12/18/14-11/30/15, $1000, Sponsor.

Cystic Fibrosis Foundation, "Pathogenesis of Goblet Cell Dysfunction in the CF Intestine", 04/01/15-03/31/17,

Professional Services
Undergraduate Supervisor-High School Student
Master's Thesis Advisor
PhD Dissertation Advisor
PhD Dissertation Committee Member
Post Doctoral Supervisor

Search Committee for Chair of Biomedical Sciences Department, Member
CVM Curriculum Committee, Member

Interim Chair Professional Curriculum Committee, Member

NIH Study Section - Clinical, Integrative and Molecular Gastroenterology (CIMG), Regular member

Intestinal Disorders, Am. Gastroenterological Assoc., Abstract reviewer

Moderator, Animal Models for CF Research and Drug Development, 1st Cystic Fibrosis Foundation Research Conference: Pushing the Frontiers, May 31-June 3, 2015

Moderator, Workshop 2 "New Advances in CF Animal Models", 29th North American Cystic Fibrosis Conference, Phoenix, AZ, Oct. 8-10, 2015

George Davis

Active Grants
NIH-NHLBI- R01 R.T. Tranquillo-PI, G.E. Davis Coll. Investigator, "Biopolymer-guided human stem cell assembly for engineered myocardium. 9/05/11- 5/31/15., $65,000/ yr.

Professional Service
Professional Society Memberships
American Society for Investigative Pathology
American Association for the Advancement of Science
American Society for Cell Biology
North American Vascular Biology Organization

Ad hoc reviewer for the following granting agencies:
American Cancer Society
Spinal Cord Research Foundation
National Institutes of Health, Pathology A Study Section
American Heart Association, Western States Affiliate
ZRG1 CVRS-L Special Emphasis Panel
ZRG1 CVRS-B Special Emphasis Panel (Challenge grants)
ZRG1 VH C (02) Special Emphasis Panel

Ad hoc reviewer for the following journals:
American Journal of Physiology
Cancer
Experimental Cell Research
Journal of Virology
Clinical and Experimental Metastasis
Journal of Cell Biology
Science
Brain Research
Developmental Brain Research
Journal of Leukocyte Biology
Trends in Cardiovascular Medicine
FASEB Journal
American Journal of Pathology Journal of Cell Science
Atherosclerosis, Thrombosis and Vascular Biology
Molecular Biology of the Cell
Developmental Dynamics
Development
Proc. Natl. Acad. Sci. USA
Microcirculation
Blood
Current Biology
Arthritis and Rheumatism
BBA-Cancer
Cancer Research
Journal of Vascular Biology
Molecular and Cellular Biology

Michael J. Davis

Publications

Fairfax ST, Padilla J, Vianna LC, Holwerda SW, Davis MJ, and Fadel PJ: Myogenic responses occur on a beat-to-beat basis in the resting human limb. Am J Physiol 308(1):H59-67, 2015.

Chakraborty S, Davis MJ, Muthuchamy M. Emerging trends in the pathophysiology of lymphatic contractility. Seminars in Cell and Developmental Biology 2015 Jan 21. pii: S1084-9521(15)00007-5. doi: 10.1016/j.semcdb.2015.01.005.

Scallan JP, Hill MA and Davis MJ: Lymphatic Vascular Integrity is Disrupted in Diabetes: Dual Roles for Nitric Oxide. Cardiovascular Res 2015 Jul 1;107(1):89-97. doi: 10.1093/cvr/cvv117

Active Grants

NIH P01 HL-095486, Mechanisms of Microvascular Control in Health and Disease:
Project 2, Regulation of vascular smooth muscle Ca2+ and BK channels by the ECM-integrin-cytoskeletal axis; Davis MJ, Project Leader, $265,000/yr, 4/1/10-3/31/15, 25% effort

NIH P01 HL-095486, Mechanisms of Microvascular Control in Health and Disease
Project 1, Regulation of Microvascular Smooth Muscle Contraction by the ECM-Integrin-Cytoskeletal Axis; Davis MJ, Co-I (G. Meininger, P.I.), $256,000/yr, 4/1/10-3/31/15, 5% effort

NIH R01 HL-120867, Mechanisms of lymphatic valve and pump dysfunction in lymphedema;
Davis MJ, P.I., $250,000/yr, 8/1/14-7/31/18, 30% effort

NIH R01 HL-122608, Conduction within and along the lymphatic vascular wall; Davis MJ, P.I., $270,000/yr, 12/24/14 - 12/23/18, 25% effort

NIH U01 HL-123420, Transport Phenomena in the Lymphatic System; (D. Zawieja and J. Moore, Co-P.I.), $250,000/yr, 6/1/14-5/31/19, Davis MJ, Co-I; 5% effort

NIH R01 HL-117487, Lymphatic Vessel Abnormalities in CM-AVM; (Philip King, P.I.), $250,000/yr, 12/24/14 - 12/23/18, Davis MJ, Co-I; 5% Effort

Professional Services

2004- Associate Member, Smooth Muscle Research Group, University of Calgary, Alberta, Canada
2005- Margaret Proctor Mulligan Professor of Medical Research, Dept. of Medical Pharmacology & Physiology, University of Missouri, Columbia, MO

Vice-chair, Dept. of Medical Pharmacology & Physiology;
Adjunct Professor, Dept. of Internal Medicine;
Adjunct Professor, Dept. of Veterinary Biomedical Sciences;
Adjunct Professor, Dept. of Biological Engineering;
Investigator, Dalton Cardiovascular Research Center, University of Missouri

Microcirculatory Society: Member, 1983-
American Physiological Society: Member, 1986-
Biophysical Society: Member, 1990-
American Society for Biochemistry and Molecular Biology: Member, 2001-
North American Vascular Biology Organization: 1997-98, 2010-
The Physiological Society: 2011-

Editorial Boards:
American Journal of Physiology: Heart & Circulatory Physiology, 1991-99; 2001-10; 2013-
Journal of Vascular Research, 2001-
Frontiers in Vascular Physiology, 2010-

Shinghua Ding

Publications

Disruption of IP3R2-mediated Ca(2+) signaling pathway in astrocytes ameliorates neuronal death and brain damage while reducing behavioral deficits after focal ischemic stroke. Li H, Xie Y, Zhang N, Yu Y, Zhang Q, Ding S. Cell Calcium. 2015 Dec;58(6):565-76. doi: 10.1016/j.ceca.2015.09.004. Epub 2015 Sep 25. PMID: 26433454

Photothrombosis-induced Focal Ischemia as a Model of Spinal Cord Injury in Mice. Li H, Roy Choudhury G, Zhang N, Ding S. J Vis Exp. 2015 Jul 16;(101):e53161. doi: 10.3791/53161. PMID: 26274772

Active Grants

13GRANT17020004 (Grant-in-aid) SHINGHUA DING (PI) 07/01/2013-06/30/2015
American Heart Association-Midwest Affiliate Title: Mechanistic study of neuronal protective role of PBEF in cerebral ischemia The goal of this project is to study the mechanism of PBEF in neuronal protection in ischemia with focus on the role of PBEF in mitochondrial function and biogenesis. Role: PI R01NS069726 $143,000

SHINGHUA DING (PI) 05/15/2010-04/30/2015 NINDS/NIH Title: The Role of Gliotransmission in Cerebral Ischemia The goal of this project is to determine whether astrocytes play a role through gliotransmission in neuronal excitotoxicity and brain damage after ischemia using two-photon (2-P) microscopy, electrophysiology and immunocyto- and histo- chemistry. Role: PI $1,551,990

R01NS094539 SHINGHUA DING (PI) 09/30/2015-08/31/2020
Title: The role and mechanisms of PBEF in acute brain injury and long-term stroke outcomes after focal ischemic stroke The goal of this project is to determine the role of neuronal PBEF in brain protection, mitochondrial function, neuronal plasticity and behavioral outcomes after focal ischemic stroke. $1,593,766

Professional Service

June 15, 2009-present: Member of safety committee of Dalton Cardiovascular Research Center.
August 18, 2009-present: Member of safety committee of College of Engineering.
September 2011- present: Member, Library Committee, College of Engineering.
Member of Membership Committee, American Society for Neurochemistry. March 2011-March 2015.

Reviewers for multiple Journals
2013-Peer review study section member for BRAIN 5, American Heart Association (AHA).
2013-2016: Chair of the Membership Committee for American Society of Neurochemistry (ASN).
2011-2013: Member of the Membership Committee for American Society of Neurochemistry (ASN).

Paul J. Fadel

Publications

Augmented pressor and sympathetic responses to skeletal muscle metaboreflex activation in type 2 diabetes patients. Holwerda SW, Restaino RM, Manrique Acevedo C, Lastra G, Fisher JP, Fadel PJ. Am J Physiol Heart Circ Physiol. 2015 Nov 13:ajpheart.00636.2015. doi: 10.1152/ajpheart.00636.2015. [Epub ahead of print] PMID: 26566729

Influence of age on respiratory modulation of muscle sympathetic nerve activity, blood pressure and baroreflex function in humans. Shantsila A, McIntyre DB, Lip GY, Fadel PJ, Paton JF, Pickering AE, Fisher JP. Exp Physiol. 2015 Sep;100(9):1039-51. doi: 10.1113/EP085071. Epub 2015 Aug 18. PMID: 26154775

Role of habitual physical activity in modulating vascular actions of insulin. Padilla J, Olver TD, Thyfault JP, Fadel PJ. Exp Physiol. 2015 Jul 1;100(7):759-71. doi: 10.1113/EP085107. PMID: 26130183

Impact of prolonged sitting on lower and upper limb micro- and macrovascular dilator function. Restaino RM, Holwerda SW, Credeur DP, Fadel PJ, Padilla J. Exp Physiol. 2015 Jul 1;100(7):829-38. doi: 10.1113/EP085238. Epub 2015 Jun 10. PMID: 25929229

Autonomic adjustments to exercise in humans. Fisher JP, Young CN, Fadel PJ. Compr Physiol. 2015 Apr;5(2):475-512. doi: 10.1002/cphy.c140022. Review. PMID: 25880502

Myogenic responses occur on a beat-to-beat basis in the resting human limb. Fairfax ST, Padilla J, Vianna LC, Holwerda SW, Davis MJ, Fadel PJ. Am J Physiol Heart Circ Physiol. 2015 Mar 1;308(5):H554-5. doi: 10.1152/ajpheart.00012.2015. No abstract available. PMID: 25728961

Impaired dynamic cerebral autoregulation at rest and during isometric exercise in type 2 diabetes patients. Vianna LC, Deo SH, Jensen AK, Holwerda SW, Zimmerman MC, Fadel PJ. Am J Physiol Heart Circ Physiol. 2015 Apr 1;308(7):H681-7. doi: 10.1152/ajpheart.00343.2014. Epub 2015 Jan 16. PMID: 25599569

A cholinergic contribution to the circulatory responses evoked at the onset of handgrip exercise in humans. Vianna LC, Fadel PJ, Secher NH, Fisher JP. Am J Physiol Regul Integr Comp Physiol. 2015 Apr 1;308(7):R597-604. doi: 10.1152/ajpregu.00236.2014. Epub 2015 Jan 14. PMID: 25589014

Characterizing rapid-onset vasodilation to single muscle contractions in the human leg. Credeur DP, Holwerda SW, Restaino RM, King PM, Crutcher KL, Laughlin MH, Padilla J, Fadel PJ. J Appl Physiol (1985). 2015 Feb 15;118(4):455-64. doi: 10.1152/japplphysiol.00785.2014. Epub 2014 Dec 24. PMID: 25539935

Elevated peripheral blood mononuclear cell-derived superoxide production in healthy young black men. Deo SH, Holwerda SW, Keller DM, Fadel PJ. Am J Physiol Heart Circ Physiol. 2015 Mar 1;308(5):H548-52. doi: 10.1152/ajpheart.00784.2014. Epub 2014 Dec 19. PMID: 25527783

Adrenergic and non-adrenergic control of active skeletal muscle blood flow: implications for blood pressure regulation during exercise. Holwerda SW, Restaino RM, Fadel PJ. Auton Neurosci. 2015 Mar;188:24-31. doi: 10.1016/j.autneu.2014.10.010. Epub 2014 Oct 22. Review. PMID: 25467222

Myogenic responses occur on a beat-to-beat basis in the resting human limb. Fairfax ST, Padilla J, Vianna LC, Holwerda SW, Davis MJ, Fadel PJ. Am J Physiol Heart Circ Physiol. 2015 Jan 1;308(1):H59-67. doi: 10.1152/ajpheart.00609.2014. Epub 2014 Oct 31. PMID: 25362138

Acute inactivity impairs glycemic control but not blood flow to glucose ingestion. Reynolds LJ, Credeur DP, Holwerda SW, Leidy HJ, Fadel PJ, Thyfault JP. Med Sci Sports Exerc. 2015 May;47(5):1087-94. doi: 10.1249/MSS.0000000000000508. PMID: 25207931

Active Grants
"Impaired insulin-stimulated blood flow in diabetic patients: Underlying mechanisms", American Heart Association, Midwest Affiliate Grant in Aid, 10% effort, PI: Paul Fadel, 7/14-8/16, $143,000.

Professional Services
Associate Professor- Department of Medical Pharmacology and Physiology, University of Missouri, Columbia, MO (9/11-present)
Assistant Director for Research Training- MU Institute for Clinical and Translational Science (MU-iCATS), University of Missouri, Columbia, MO (9/11-present)

TEACHING EXPERIENCE:
Associate Professor- University of Missouri, Columbia, MO (9/05-present) Graduate Courses: Neural Control of the Circulation- 12 contact hours; Advanced Exercise Physiology- 3 contact hours; Skills in Biomedical Research- 6 contact hours, Respiratory Physiology Section of Veterinary Physiology- 3 contact hours.
Medical Courses: Problem Based Learning Tutor- Block 8. PEER REVIEW EXPERIENCE:
Ad-hoc Manuscript Reviewer- Medicine and Science in Sports and Exercise (6/01-present)
Experimental Physiology (2/03-present)
AJP: Regulatory, Integrative and Comparative Physiology (8/03-present)
Journal of Applied Physiology (12/03-present)
European Journal of Applied Physiology (9/04-present) AJP: Heart and Circulatory Physiology (12/04-present)
Journal of Physiology (5/05-present)
Experimental Biology and Medicine (10/06-present)
Hypertension (5/07-present)
Brain Research (8/09-present)
Annals of Neurology (12/09-present)
Expert Review of Cardiovascular Therapy (2/13-present)

Editorial Board- Experimental Physiology (11/12-present)
Journal of Applied Physiology (1/11-present)
AJP: Heart and Circulatory Physiology (1/11-present)
European Journal of Applied Physiology (1/10-present)
Frontiers in Exercise Physiology (7/10-present)

Ad-hoc Grant Reviewer- Alberta Heritage Foundation for Medical Research
University of Missouri Research Board
Natural Sciences & Engineering, Research Council of Canada

Shubra Gangopadhyay

Publications

Aaron J. Wood, Sagnik Basuray, Sangho Bok, Keshab Gangopadhyay, Shubhra Gangopadhyay, Sheila Grant, "Enhanced DNA Detection through the Incorporation of Nanocones and Cavities into a Plasmonic Grating Sensor Platform," IEEE Sensors, Accepted September 2015.

Haisheng Zheng, Yang Zhou and Shubhra Gangopadhyay, "Size-dependent work function and single electron memory behavior of pentacene non-volatile memory with embedded sub-nanometer platinum nanoparticles", Journal of Applied Physics, 117, 024504, 2015.

Haisheng Zheng, Mohamed Asbahi, Somik Mukherjee, Cherian J. Mathai, Keshab Gangopadhyayc, Joel K. W. Yang , Shubhra Gangopadhyay, "Room Temperature Coulomb Blockade Effects in AuNC/Pentacene Single Electron Transistors", Nanotechnology, 26 (35), 355204, 2015.

Biyan Chen, Avinash Pathak, Keshab Gangopadhyay, Peter V. Cornish, and Shubhra Gangopadhyay, "Single-molecule Detection in Nanogap-embedded Plasmonic Gratings", Nanobiomedicine, 2,8, 2015.

Aaron J. Wood, Biyan Chen, Sami Pathan, Sangho Bok, Cherian J. Mathai, Keshab Gangopadhyay, Sheila A. Grant, and Shubhra Gangopadhyay, "Influence of Silver Grain Size, Roughness, and Profile on the Extraordinary Fluorescence Enhancement Capabilities of Grating Coupled Surface Plasmon Resonance", RSC Advances, 5, 78534-78544, 2015.

Sagnik Basuray, Avinash Pathak, Biyan Chen, Drew Menke, Charles M. Darr, Keshab Gangopadhyay, Peter V. Cornish, Shubhra Gangopadhyay, "Single Molecule Oscillations of an RNA/DNA Duplex in a Plasmonic Nanocavity", J Nanomed Nanotechnol, 06, 03, 2015.

Haisheng Zheng, Somik Mukherjee, Keshab Gangopadhyay, Shubhra Gangopadhyay, "Ultrafine Pt nanoparticle induced doping/strain of single layer graphene: experimental corroboration between conduction and Raman characteristics",J Mater Sci: Mater Electron, 26, 4746–4753, 2015.

O Azizi, A El-Boher, JH He, GK Hubler, D Pease, W Isaacson, V Violante, S Gangopadhyay, "Progress towards understanding anomalous heat effect in metal deuterides," Current Science, 108(4), 565-573, February 2015.

GK Hubler, A El-Boher, O Azizi, D Pease, JH He, W Isaacson, S Gangopadhyay, V Violante, "Sidney Kimmel Institute for Nuclear Renaissance," Current Science, 108(4), 562-564, February 2015.

Steven C Hamm, L Currano and Shubhra Gangopadhyay, "Multilayer thin film capacitors by selective etching of Pt and Ru electrodes" Microelectronic Engineering, 133, 92-97, 2015.

VK Patel, JR Saurav, K Gangopadhyay, S Gangopadhyay and S. Bhattacharya, "Combustion characterization and modeling of novel nanoenergetic composites of Co3O4/nAl", RSC Advances 5 (28), 21471-21479, 2015.

Raja Thiruvengadathan, Clay Staley, Jordan Geeson, Steven Chung, Kristofer Raymond, Keshab Gangopadhyay, Shubhra Gangopadhyay, "Enhanced Combustion Characteristics of Bismuth Trioxide-Aluminum Nanocomposites Prepared through Graphene Oxide Directed Self-Assembly", Journal and Propellants, Explosives and Pyrotechnics, accepted, 2015.

Presentations
H Zheng and S Gangopadhyay, "Molecularly imprinted organic transistor-based sensor for selective trace chemical vapor detection," in 2015 Transducers - 2015 18th International Conference on Solid-State Sensors, Actuators and Microsystems (TRANSDUCERS), (June 21-25, 2015).

S Bok, S Pathan, A J Wood, B Chen, C J Mathai, K Gangopadhyay, S Grant, C McArthur, and S Gangopadhyay, "Highly sensitive plasmonic grating platform for the detection of a wide range of infectious diseases," in 2015 Transducers - 2015 18th International Conference on Solid-State Sensors, Actuators and Microsystems (TRANSDUCERS), (June 21-25, 2015).

Active Grants
IREX, $32,500: "Developing the concepts of entrepreneurship, technology transfer and curriculum development of a course in entrepreneurship in a university setting at UoT," Award period 8/1/2014-2/28/2015 Role Co-I

IREX, $39,500: "Training of professors from UoT in nanotechnology projects and equipment," Award period 8/1/2014-2/28/2015 Role: PI

Coulter Translational Partnership, $141,306: "Cost Effective Plasmonic Grating Platform for Detection of Mycobacterium Tuberculosis", Award period 7/1/2014-6/30/2015. Role: PI.

Professional Service
SCIENTIFIC AND PROFESSIONAL SOCIETY MEMBERSHIPS
American Physical Society
Institute of Electronics and Electrical Engineers
Material Research Society

Sep. 2003 - present LaPierre Chair Professor, Department of Electrical and Computer Engineering, University of Missouri, Columbia, Missouri (MU)

Sep. 2003 - present Joint Professor, Department of Physics and Astronomy, MU

Sep. 2003 - present Joint Professor, Department of Biological Engineering, MU

April 2006 - present Director, International Center for Nano/Micro Systems and Nanotechnology, MU

Kevin Gillis

Publications
Yao, J., Liu, X., Gillis, K.D. Two approaches for addressing electrochemical electrode arrays with reduced external connections. Analytical Methods 7:5760-5766, 2015.

Active Grants
NIH, R01, $543,546 (MU portion), "A scalable nxn electrochemical detector array platform with on-chip amplifiers for massively parallel recordings of quantal transmitter release events.", co-I (PI: M. Lindau, Cornell Univ.), 09/11-04/15

NIH / NIMH, R44 MH096650-03A1, Phase II SBIR (to ExoCytronics LLC), "Development of a prototype system for assaying exocytosis from individual cells", PI, $1,470,603 (subcontract to MU: $443,275), 09/15 – 07/18.

NIH / NIBIB, R01 EB020415-01A1, "Neuronal Imaging using Fluorescent Sensors for Neurotransmitters", co-I (PI: Tim Glass), $1,420,712, 12/2015 – 11/2019.

Professional Service
Journal Reviewer: Nature**,** Science, Neuron, EMBO Journal, Biophysical Journal, Journal of Theoretical Biology, Journal of Neuroscience

Undergraduate Graduate Student Advisor

Kenneth Gruber

Professional Service
Current: Founder and CEO/President, Tensive Controls, Inc., a biotech pharmaceutical company supported by grants from the NIH/NCI SBIR program, North Carolina State SBIR matching funds, and the IRS/HHS Qualifying Therapeutic Discovery Project program. Professor Emeritus of Biological Sciences, California State Polytechnic University, Pomona, CA; Adjunct Professor, Dalton Cardiovascular Research Center and Department of Medical Pharmacology and Physiology, University of Missouri, Columbia, MO

2004-Present Lytmos Group, Inc

American Physiological Society
Association Pour les Exchanges Scientifique
Internationaux (Honorary Member)

Li-Qun Gu

Publications

Zhang, X., Xu, X., Yang, Z., Burcke, A.J., Gates, K.S., Chen, S.J. and Gu, L.Q. Mimicking ribosomal unfolding of RNA pseudoknot in a protein channel. Journal of the American Chemical Society. Accepted for publishing (2015). http://pubs.acs.org/doi/abs/10.1021/jacs.5b07910

Zhang, X., Price, N.E., Fang, X., Yang, Z., Gu, L.Q. (Corresponding author) and Gates, K.S. (Corresponding author). Characterization of interstrand DNA-DNA cross-links using the α-hemolysin protein nanopore. ACS Nano. To be published ASAP (2015). http://www.ncbi.nlm.nih.gov/pubmed/26563913.

Wang,Y., Montana, V., Grubišić, V., Stout, R.F., Parpura, P. and Gu, L.Q. Nanopore sensing of botulinum toxin type B by discriminating an enzymatically cleaved peptide from a synaptic protein synaptobrevin 2 derivative. ACS Applied Materials and Interfaces 7, 184–192 (2015).

Wang, Y., Ritzo, B. and Gu, L.Q. Silver(I) ions modulate the stability of DNA duplexes containing cytosine, methylcytosine and hydroxymethylcytosine at different salt concentrations. RSC Advances 5, 2655–2658 (2015).

*Wang, Y. and Gu, L.Q. Biomedical diagnosis perspective of epigenetic detections using alpha-hemolysin nanopore. AIMS Materials Science 2, 448-472 (2015)

Active Grants

NIH R01 GM114204 PI 04/01/2015 – 3/31/2018
Nanopore single-molecule dielectrophoresis and biomedical detection

Total cost: $914,157
MU Research Board PI 2/1/2014 – 1/31/2015
Nanosensor for Counting MicroRNA Cancer Biomarkers

Direct cost: $35,000
Mizzou Advantage Co-I (PI: Mario Pennella) 8/2014 -8/2015
Undergraduate Research Teams (URT) educational program: Building Biological Systems that Operate in Living Cells

Professional Services

NIH/NCI IMAT R21 and R33 (2015), NIH IMST SBIR (2015), NIH EBIT (2015) Reviewer
Hong Kong Innovation and Technology Support Program (2015)
Engineering and Physical Sciences Research Council (2015)
Beckman Young Investigator Program (2015)
University of Missouri Research Board (2006, 2007, 2012, 2014, 2015)
Guest Editor (2015-present), Microchimica Acta, Special theme: Nanopores in Bioanalytical Sciences
Editor, Journal of Biosensors and Bioelectronics
University of Missouri Doctoral Faculty (2007-2017),
Chair of BE Faculty Search Committee (2015)
Traffic Appealing Committee (2015)
Judge of the Spring Undergraduate Research & Creative Achievements Forum (2010-2015)

Eileen Hasser

Publications

Matott MP, Ruyle BC, Hasser EM, Kline DD. Excitatory amino acid transporters tonically restrain nTS synaptic and neuronal activity to modulate cardiorespiratory function.
J Neurophysiol. 2015 Dec 30:jn.01054.2015. doi: 10.1152/jn.01054.2015. [Epub ahead of print]
PMID: 26719090

Coldren KM, Brown R, Hasser EM, Heesch CM. Relaxin increases sympathetic nerve activity and activates spinally projecting neurons in the paraventricular nucleus of nonpregnant, but not pregnant, rats. Am J Physiol Regul Integr Comp Physiol. 2015 Dec 15;309(12):R1553-68. doi: 10.1152/ajpregu.00186.2015. Epub 2015 Sep 23.
PMID: 26400184

King TL, Ruyle BC, Kline DD, Heesch CM, Hasser EM. Catecholaminergic neurons projecting to the paraventricular nucleus of the hypothalamus are essential for cardiorespiratory adjustments to hypoxia. Am J Physiol Regul Integr Comp Physiol. 2015 Oct;309(7):R721-31. doi: 10.1152/ajpregu.00540.2014. Epub 2015 Jul 8.
PMID: 26157062

Presentations

International Society for Autonomic Neuroscience, Stresa, Italy:
nTS ROS contribute to acute intermittent hypoxia-induced phrenic and sympathetic Long Term Facilitation

Active Grants

NIH	**07/15/10-12/31/15**
Plasticity of nTX output neurons in actue and chronic hypoxia	$970,507

Professional Service

Committees

- Departmental- GPAC, New Faculty Mentoring Committee, Outreach Committee
- College - Promotion and Tenure Committee
- University - Animal Care and Use Committee, Cardiovascular Day Organizing Committee

Manuscript Review

American Journal of Physiology (Heart Circulatory Physiology)
American Journal of Physiology (Regulatory Comp & Integ Physiology)
Journal of Physiology
Brain Research
Journal of Applied Physiology
Experimental Physiology
Journal of Neuroscience
Neuroscience

Grant Review

Research Board, University of Missouri

Cheryl Heesch

Publications:

Abstracts (National Meetings)

KM Coldren, DD Kline, EM Hasser, and CM Heesch. Chemical stimulation of the arterial chemoreflex activates neuroendocrine and pre-sympathetic corticotropin releasing hormone neurons in the paraventricular nucleus of the hypothalamus. FASEB J: 29: 653.6, 2015.

CM Heesch, JG Phaup, DD Kline, and EM Hasser. Acute hypoxia potentiates cardiorespiratory responses to blockade of the anti-oxidant enzyme, glutathione peroxidase, in the nucleus tractus solitarius. FASEB J: 29: 653.7, 2015.

BC Ruyle, KM Coldren, EF Palmieri, CM Heesch, and EM Hasser. Hypoxia activates neurons in the hypothalamic paraventricular nucleus that project to the nucleus tractus solitarii in the brainstem. FASEB J: 29: 653.9, 2015.

JD Magee, SH Deo, PJ Fadel and CM Heesch. Norepinephrine increases production of superoxide in cultured peripheral blood mononuclear cells and splenocytes isolated from rats. FASEB J: 29: 1059.5, 2015.

CM Heesch, KM Coldren, EM Hasser, and R Brown. Relaxin increases sympathetic nerve activity and activates spinally-projecting neurons in the paraventricular nucleus of nonpregnant, but not pregnant, rats. Auton Neuroscience: Basic & Clin: 192: 90 – 91, 2015. doi:10.1016/j.autneu.2015.07.118

D Ostrowski, CM Heesch, DD Kline, and EM Hasser. nTS ROS contribute to acute intermittent hypoxia-induced phrenic and sympathetic LTF. Auton Neuroscience: Basic & Clin: 192: 47, 2015. doi:10.1016/j.autneu.2015.07.398

Manuscripts (Peer Reviewed Journals)

King, TL, BC Ruyle, DD Kline, CM Heesch, EM Hasser. Catecholaminergic neurons projecting to the paraventricular nucleus of the hypothalamus are essential for cardiorespiratory adjustments to hypoxia. Amer. J. Physiol. (Regulatory, Integrative, & Comparative Physiol.) 309: R721 – R 731, 2015.

Coldren, KM, R Brown, EM Hasser, CM Heesch. Relaxin increases sympathetic nerve activity and activates spinally-projecting neurons in the paraventricular nucleus of nonpregnant, but not pregnant, rats. Amer. J. Physiol. (Regulatory, Integrative, & Comparative Physiol.), 309: R1553-1568, 2015. doi:10.1152/ajpregu.00186.2015

Book Chapters

Kline, DD, EM Hasser, and CM Heesch. Chapt. 34: Regulation of the Heart. Dukes Physiology of Domestic Animals, Edition 13, ed. Howard Erickson, Wiley, Pgs. 341 -351, 2015.

Heesch, CM, DD Kline, and EM Hasser. Chapt. 35: Control mechanisms of the circulatory system. Dukes Physiology of Domestic Animals, Edition 13, ed. Howard Erickson, Wiley, Pgs. 352 – 371, 2015.

Hasser, EM, CM Heesch, DD Kline and MH Laughlin. Chapt. 38: Special Circulations. Dukes Physiology of Domestic Animals, Edition 13, ed. Howard Erickson, Wiley, Pgs. 399 – 416, 2015.

Presentations
3/2015: Experimental Biology 2015;Boston; 3/28-4/1, 2015 (abstracts 1 – 4)

9/2015: International Soc. Autonomic Nervous System; Stresa, Italy (abstracts 5 & 6).

Active Grants

"Plasticity of nTS output neurons in acute and chronic hypoxia"	07/01/10-12/31/14 (NCE through 12/31/15)	$491,679 (total annual direct)

National Institutes of Health (R01 HL098602)
Multi-Principal Investigator Grant
Role: PI's = E.M. Hasser, C.M. Heesch, D.D. Kline [Heesch annual direct $163,512]
(1.8 Calendar months/ each)
15% effort – no salary recovery in NCE year

"Central nervous system plasticity in Sympathoinhibition in pregnancy	06/01/09- 09/30/15 (in NCE)	~$250,000 (annual direct)

NIH (R01 HL091164)
Role: PI
20% effort; no salary recovery in NCE years

"CNS role of the ovarian hormone Relaxin in maintenance of sympathetic Outflow in pregnancy"	03/14 – 03/16	$7,500

Univ. of Missouri, Research Council, #8008
Role: P.I.
No PI salary allowed

"Cell-type specific changes in CNS CV control-pregnancy"	11/01/15-10/31/16 (Award notice 5/20/15)	$59,988

UM Research Board #2411
Role: PI
No PI salary allowed

Professional Services
National/International
Manuscript Peer Review:
American Journal of Physiology/ Heart & Circ. Physiol
American Journal of Physiology/ Regulatory
Journal of Applied Physiology
Hypertension

Editorial Board:
06/10- present: Review Editor, Frontiers in Physiology: Frontiers in Integrative Physiology

Michael Hill

Publications

Mishra, R.C., Wulff, H., Hill, M.A. and Braun, A.P. Inhibition of myogenic tone in rat cremaster and cerebral arteries by SKA-31, an activator of endothelial KCa2.3 and KCa3.1 channels (J. Cardiovasc Pharmacol, Accepted for publication).

Chen, X., Zhang, H., Hill, M.A., Zhang, C. and Park, Y. Regulation of coronary endothelial function by interactions between TNF, LOX-1 and Adiponectin in ApoE Knockout mice. (J. Vasc. Res. (Accepted pending minor revision).

Scallan, J.P., Hill, M.A. and Davis, M.J. Lymphatic vascular integrity is disrupted in diabetes: Dual roles for nitric oxide Cardiovascular Research, In Press.

Hong, K., Lee, S., Li, R., Wu, J., Yang, Y. and Hill, M.A. Adiponectin receptor antagonist, AdipoRON, Causes Arteriolar Relaxation by a direct smooth muscle action. Microcirculation, In Press, 2015.

Vieira-Potter, V.J., Lee, S., Bayless, D.S., Scroggins, R.J., Welly, R.J., Fleming, N.J., Smith, T.N., Meers, G.M., Hill, M.A., Rector, R.S. and Padilla, J. Disconnect between adipose tissue inflammation and cardiometabolic dysfunction in Ossabaw pigs. (Obesity, In Press 2015).

Katelee Barrett Mueller, K., Bender, S.B., Hong, K., Yang, Y., Aronovitz, M., Jaisser, F., Hill, M.A. and Jaffe, I.Z. Endothelial mineralocorticoid receptors differentially contribute to coronary and mesenteric vascular function without modulating blood pressure. Hypertension (In Press, 2015).

Foote, C.A., Castorena-Gonzalez, J.A., Staiculescu, M.C., Clifford, P.S., Hill, M.A., Meininger, G.A. and Martinez-Lemus, L. Brief 5-HT exposure induces arteriolar inward remodeling in vivo via a mechanism involving transglutaminase activation and actin cytoskeleton reorganization. Am. J. Physiol., In Press.

Grants

National Institutes of Health 1 P01 HL095486-01A1
Project Title: Mechanisms of Microvascular Control and Coordination in Health and Disease Period: 5/2010 – 4/2015 Principal Investigator: Gerald A. Meininger, PhD. Co-Investigator Project 1 (10% time) Michael A. Hill, Ph.D. Co-Investigator Project 2 (5% time) Michael A. Hill, Ph.D. Director of Core C (10% time) Michael A. Hill, Ph.D.

NIH RO1HL085119 – 3/31/2016
Role of Cytokine-induced Inflammation in Endothelial Dysfunction in Diabetes Total Award (Current Period): $1,000,000 (Direct Costs) The major focus of this proposal is on vascular dysfunction in a rodent model of type 2 diabetes. Specifically focusing on how cytokines and immune cells contribute to abnormal function of endothelial cells and alter vasomotor responsiveness. Principal Investigator; Michael A. Hill, Ph.D., Effort 22.5%

Professional Service

2015- Present, Interim Director, Dalton Cardiovascular Research Center, Univesity of Missouri
2006 – 2015 Associate Director, Dalton Cardiovascular Research Center, University of Missouri

2006 – present Professor of Physiology (Tenured), Dalton Cardiovascular Research Center
Department of Medical Pharmacology and Physiology, University of Missouri

2007 – present Adjunct Professor, Department of Biological Engineering, University of Missouri

2011 – present Distinguished Research Fellow, External Joint Appointments (Current):RMIT University
Melbourne, Vic 3083.

2011 – present Visiting Professor, Luzhou Medical College, Luzhou, China

Associate Editor:

- Frontiers in Vascular Physiology (2010 – present)

Editorial Boards:

- American Journal of Physiology: Heart and Circulatory Physiology (1/96 - 12/98; 1/13 - present)
- Microcirculation (1/2002 – present)
- Journal of Vascular Research (2007 – present)
- Frontiers in Vascular Pharmacology (2010 – present)
- Frontiers in Vascular Physiology (2010 – present)

Professional Societies

Microcirculatory Society
American Physiological Society
Australian Physiological Society
Australian and New Zealand Microcirculatory Society
Biophysical Society
American Association for the Advancement of Science
Australian-American Fulbright Alumni

2012 – present	University of Missouri, Research Council
2011 – present	Coordinator of MOU/Exchange agreement between Suzhou Medical College, China and DCRC, University of Missouri.
2007 – present	Seminar Committee, Dep't of Medical Pharmacol. and Physiol. University of Missouri
2006 – present	Internal Review Committee, Dalton Cardiovascular Research Ctr
2012 - present	Joint Programming Committee Representative (APS, CV Section)
2012 - present	International Committee, American Physiological Society.
2011 – present	International Liaison Committee, Microcirculatory Society, USA.
2011 – present	Long Range Planning Committee, Chair, Microcirculatory Society, USA.
2010 – present	Steering Committee, CV Section, American Physiological Society
2010 – present	Development Committee, Microcirculatory Society, USA.

Virginia Huxley

Active Grants
Current:
NIH R01 DK095501-01A1 "Insulin as a Regulator of Microvascular Exchange Score 20; 9th percentile
06-01-2013 through 05-31-2017; $1,250,000 total direct

Professional Service
1996 - Director, Microvessel Core Facility
1999 - Adjunct Professor, Veterinary Biomedical Sciences, UM-Columbia School of Veterinary Medicine
2003 - Professor, Department of Medical Pharmacology & Physiology, UM-Columbia
2003 - Senior Investigator, Center for Diabetes and Cardiovascular Health
2005 - Director, National Center for Gender Physiology, UM-Columbia
2011 - Director, Pulmonary/Critical Care & Physiology Research Partnership
2011 - Professor, Department of Internal Medicine, UM-Columbia

PRIZES, AWARDS, FELLOWSHIPS:
2012-2015 Associate Editor, Journal of Physiology (London)
2010- Associate Editor, Frontiers in Vascular Physiology
Monthly Division Research Meeting/Journal Club (2011-)

Faculty of 1000, Integrative Physiology	2010-present
Associate Editor	
Frontiers in Vascular Physiology	2010-present

Editorial Board	
Journal of Vascular Research	1998-present
Asian Biomedicine	2007-present

Guest Reviewer	
American Journal of Physiology:	1983-present
Circulation Research	1986-
Biorheology	1989-
Biophys. Biochem. Acta	1989-
Journal of Applied Physiology	1991-
Journal of Physiology (London)	1991-
Hypertension	1996-
Microcirculation	2004 –
Cardiovascular Research	2005 –
Journal of Pharmacology and Experimental Therapeutics	2007 -
Arteriosclerosis, Thrombosis and Vascular Biology	2008 -
Journal of General Physiology	2009 -

Extra-mural Advisory Boards	
University of Arizona Training Grant Advisory Committee	2006-present
Center for Gender Physiology, Johns Hopkins University	2008-present

Tzyh-Chang Hwang

Publications

The Fifth Transmembrane Segment of Cystic Fibrosis Transmembrane Conductance Regulator Contributes to Its Anion Permeation Pathway. Zhang J, Hwang TC. Biochemistry. 2015 Jun 23;54(24):3839-50. doi: 10.1021/acs.biochem.5b00427. Epub 2015 Jun 10.

Gao X, Hwang TC. Proc Natl Acad Sci U S A. 2015 Feb 24;112(8):2461-6. doi: 10.1073/pnas.1420676112. Epub 2015 Feb 9. PMID: 25675504 PMID: 26024338

Modulation of CFTR gating by permeant ions. Yeh HI, Yeh JT, Hwang TC. J Gen Physiol. 2015 Jan;145(1):47-60. doi: 10.1085/jgp.201411272. Epub 2014 Dec 15. PMID: 25512598

Presentations

Active Grants

2013 – 2015 Cystic Fibrosis Foundation, $125,000
2013 – 2015 University of Missouri, School of Medicine Bridge Fund, $38,960
2014 – 2018 NIHR01, NIDDK, "Molecular pathophysiology of cystic fibrosis", $920,000
2014 – 2016 Vertex Pharmaceuticals, $134,012
2014 – 2015 AbbVie, $35,000

Professional Service

2004 – present Member, Graduate Educational Committee, Medical Pharmacology and Physiology
2004 - present Member, Tenure and Promotion committee, Dalton Cardiovascular Research Center
2011 – present, Member, Tenure and Promotion committee, School of Medicine
2010 – present Associate Editor, Frontier in Pharmacology of Nature Products
2003 - present Editorial Board, Journal of General Physiology
2010 – present Member of College of CSR Reviewers, NIH
2014 – present Ad hoc member, ZRG1 F10A (Physiology and Pathobiology of Cardiovascular and Respiratory Systems) Study Section, NIH

Graduate and PostDoctoral Student Supervisor

Journal Reviewer: Journal of General Physiology
Grant Reviewer: Cystic Fibrosis Foundation, Italian Telethon Foundation, NIH (ZRG1 F10A, ad hoc reviewer).

Memberships:

Biophysical Society
Society of General Physiologist

Salman Hyder

Presentations

Cook, M. T., Mafuvadze, B., Besch-Williford, C.; Ellersieck, M., Goyette, S. and Hyder, S. M. (2015) Luteolin Prevents Development of Progestin-accelerated 7,12- dimethylbenz(a)anthracene (DMBA)-induced Mammary Tumors in Sprague-Dawley Rats. 97th Annual Endocrine Society Meeting, San Diego, Abstract # SAT-329

Goyette, S., Mafuvadze, B., Cook, M. T., Liang, Y., Hyder, S. M. (2015) Enrichment of CD44high stem-cell-like cells as a possible mechanism of progestin-dependent progression of human breast cancer. Phi Zeta Day

Liang, Y., Mafuvadze, B., Zou, X., Besch-Williford, C., and Hyder , S. M. (2015) Inhibition of oxidosqualene cyclase blocks proliferation and survival of prostate cancer cells. 106th Annual American Association of Cancer Research Meeting, Philadelphia, USA, Cancer Res 2015; 56 (Part 1): Abst 5422

Goyette, S., Mafuvadze, B., Cook, M. T., Liang, Y., Hyder, S. M. (2015) Enrichment of CD44high stem-cell-like cells as a possible mechanism of progestin-dependent progression of human breast cancer. 106th Annual American Association of Cancer Research Meeting, Philadelphia, USA, Cancer Res 2015; 56 (Part 1):Abstract #1867.

Cook, M. T., Liang, Y., Goyette, S., Mafuvadze, B., Besch-Williford, C. and Hyder, S. M. (2015). Therapeutic Effects of Luteolin Against Progestin-Dependent Breast Cancer Involves Induction of Apoptosis, and Suppression of both Stem-Cell-Like Cells and Angiogenesis. 106th Annual American Association of Cancer Research Meeting, Philadelphia, USA, Cancer Res 2015; 56 (Part 1): Abst 4159

Liang, Y., Aebi, J. and Hyder, S.M. (2015) Inhibitors of oxidosqualene cyclase block growth and survival of both hormone-dependent and independent breast cancer cells. Proceedings of the 20th World Congress on Advances in Oncology, Athens, Greece. Abstract # 328

Cook, M. T., Liang, Y. and Hyder , S. M. (2015) Luteolin inhibits growth and metastasis of hormone-dependent and triple-negative breast cancer cells and reduces their stem-cell like characteristics. 3rd Congress on Steroid Research, Chicago, IL. P015

Hyder, S. M., Cook, M. T., Besch-Williford, C. and Liang, Y. (2015) Luteolin inhibits progestin-dependent VEGF Induction, stem-cell like characteristics, and tumor progression of human breast cancer cells. San Antonio Breast Cancer Conference, San Antonio, TX. P1-16-06

Refereed Journal Articles

Cook, M.T., Liang, Y., Besch-Williford, C., Goyette, S., Mafuvadze, B. M. and Hyder, S. M. (2015) Luteolin inhibits progestin-dependent angiogenesis, stem cell-like characteristics, and growth of human breast cancer xenografts. SpringerPlus 4: 444

Cook, M. T., Mafuvadze, B., Besch-Williford, C., Ellersieck, M. R., Goyette, S. and Hyder, S. M. (2015) Luteolin suppresses development of medroxyprogesterone acetate-accelerated 7,12-dimethylbenz(a)anthracene-induced mammary tumors in Sprague-Dawley rats. Oncology Reports, In Press.

Active Grants

Hyder, S.M. (PI) "Treatment and prevention of breast cancer using multi-functional inhibitors of cholesterol biosynthesis" Dept of Defense Breast Cancer Pgm $500,000-direct cost 6/1/12-5/31/15

Hyder, S. M. (PI) "Blocking androgen receptor activity with an inhibitor of cholesterol synthesis: a novel means of suppressing prostate cancer" Dept of Defense Prostate Cancer Pgm $75,000 (direct cost) 9/2014-8/2016

Hyder, S. M. (PI) "Preventing development and metastasis of triple-negative breast cancer by reactivation of mutant p53 protein and disruption of tumor blood vessels" College of Veterinary Medicine, University of Missouri-Columbia (2/15-1/16) $18,000

Hyder, S. M. (Mentor) Franklin, C (PI) NIH T32 RR07004 "Post-doctoral Comparative Medicine Training Grant" Date: 07/11-06/16 (Direct cost: $169, 266/yr)

Hyder, S.M. (consultant) (Clarke, L PI) "CFTR and acid-base transporters in regenerating intestinal crypts" NIH-RO1 $1,250,000/direct cost (3/12-2/16)

University/College/Departmental Committees

Chancellor's Advisory Committee (MU, Chancellor Brady Deaton)
Member, Promotion and Tenure Committee, Dept of Biomedical Sciences, University of Missouri-Columbia
Chair, Appointment and Promotion Committee, DCRC
Member, Dalton Cardiovascular Research Center Scientific Programs Committee
Faculty Responsibility Committee (member), College of Vet Med, MU

National/International Service

National/International Service

Editorial Board: Histology & Histopathology (2002-present)
Honorary Visiting Professorship, Luzhou Medical College, China (2011-2016)
Awards & honors

Editorial Board:
Histology & Histopathology (2002-present)
International Journal of Oncology (2005-present)

Co-chair. Session: Breast Cancer, 20th World Congress on Advances in Oncology, Athens, Greece (2015)
Honorary Visiting Professorship, Luzhou Medical College, China (2011-2016)
Member, External Advisory Committee member for NIH Botanical Center grant (Director; Dr. Dennis Lubahn)

Ad-hoc reviewer

Manuscripts:

Current Cancer Drug Targets	Nutrition and Cancer
Endocrine Related Cancer	Mini-Reviews in Medicinal Chemistry

Other:

Abstract Reviewer: Annual Endocrine Society Meeting 2015
Judge, Presidential Award Poster, Endocrine Society Meeting, San Diego , 2015
External P & T Reviewer, Oregon State University (2015)

Grant Review
Reviewer, Fellowships, International Union Against Cancer
Invited Reviewer, American Medical Association (Neoplastic Study Section)
Reviewer, International Foundation for Science (Natural Products Area), Sweden
Reviewer, Cancer Association of South Africa, Mowbray, South Africa

David Kline

Publications

Excitatory amino acid transporters tonically restrain nTS synaptic and neuronal activity to modulate cardiorespiratory function. Matott MP, Ruyle BC, Hasser EM, Kline DD. J Neurophysiol. 2015 Dec 30:jn.01054.2015. doi: 10.1152/jn.01054.2015. [Epub ahead of print] PMID: 26719090

Catecholaminergic neurons projecting to the paraventricular nucleus of the hypothalamus are essential for cardiorespiratory adjustments to hypoxia. King TL, Ruyle BC, Kline DD, Heesch CM, Hasser EM. Am J Physiol Regul Integr Comp Physiol. 2015 Oct;309(7):R721-31. doi: 10.1152/ajpregu.00540.2014. Epub 2015 Jul 8. PMID: 26157062

Active Grants

1 R01 HL085108-01 (Kline DD) 4/01/2008-03/30/2015
NIH/NHLBI $250,000 direct/yr Adaptation of brainstem circuits to chronic hypoxia

R01 HL098602 (Kline DD) 04/01/2010 –12/31/2015
NIH/NHLBI Direct: $499,999 direct/yr Plasticity of nTS output neurons in acute and chronic hypoxia
Role: PI (MultiPI: Kline/Hasser/Heesch)

Professional

Committee/mentorship

Teaching Activity: Didactic and Clinical Teaching Departmental

2013-pres. Director of Graduate Studies (DGS)
2009-pres. Member, Research Advisory Committee (RAC)

College

2013-pres. Member, Non-Tenure Track Promotion Committee (term ends 2016)
2013-pres. Member, Computer Committee (term ends 2016)
2008-pres. Member, Animal Resources Committee (term ends 2015)
2014 Reviewer, CVM Faculty Research Awards

University

2009-pres. Member, Appointment and Promotions Committee, Dalton Cardiovascular Research Center
2008-pres. Member, Animal Issues Response Team (AIRT), Office of Research

National

2013-pres. Joint Programming Committee (JPC) representative, Hypoxia Interest Group of the American Physiological Society.
2011-pres. Reviewing Editor: Frontiers in Integrative Physiology

International

Editorial Board: Frontiers in Integrative Physiology
Reviewing Editor: Frontiers in Integrative Physiology, 2011-pres.

Reviewer: Journal of Physiology (London), Journal of Neurophysiology, Journal of Neuroscience, Neuroscience, Neuroscience Letters, Journal Neuroscience Methods, Journal of Applied Physiology, Brain Research, American Journal of Physiology (Regulatory, Integrative and Comparative Physiology; Cell), Respiration Physiology and Neurobiology, British Journal of Pharmacology, Experimental Physiology, Neurogastroenterology and Motility

Professional Service

Editorial Advisory Boards:

American Journal of Physiology: Heart and Circulatory Physiology; 2005 – present
Cardiovascular Research; 2008 – present
Circulation Research; 2010 - present

Associate Editor: Frontiers in Vascular Physiology: 2010 – present
PLoS One; 2013 -- present

Professor and Chair, Department of Medical Pharmacology and Physiology, University of Missouri-Columbia, Columbia, MO 65211 (2004 - Present)

George L. and Melna A. Bolm Distinguished Chair in Cardiovascular Health, Department of Medical Pharmacology and Physiology, University of Missouri-Columbia, Columbia, MO 65211 (2013 - Present)

Editorial Boards:
American Journal of Physiology: Heart and Circulatory Physiology, 1987-1999; 2005-present
Circulation Research 2010-present

Associate/Academic Editor:
Frontiers in Vascular Physiology: 2010 – 2015
PLoS One; 2013 -- 2015

External Grant Application Referee for:
Novo Nordisk Fonden, 2015

PROFESSIONAL SOCIETY MEMBERSHIPS:

American Physiological Society
American Society for Pharmacology and Experimental Therapeutics
United States Microcirculatory Society
European Society for Microcirculation
American Heart Association
American Gastroenterological Society
American Association for the Advancement of Science
Nitric Oxide Society
North American Vascular Biology Organization
International Society for Heart Research
Research Society on Alcoholism
Society of Free Radical Biology and Medicine

Maike Krenz

Publications

The Q510E mutation in Shp2 perturbs heart valve development by increasing cell migration. Edwards MA, Crombie K, Schramm C, Krenz M.Journal of applied physiology (Bethesda, Md. : 1985). 2015; 118(1):124-31.PubMed [journal]PMID: 25359717 PMCID: PMC4281644

Elevated Ca2+ transients and increased myofibrillar power generation cause cardiac hypercontractility in a model of Noonan syndrome with multiple lentigines. Clay SA, Domeier TL, Hanft LM, McDonald KS, Krenz M. American journal of physiology. Heart and circulatory physiology. 2015; 308(9):H1086-95.PubMed [journal] PMID: 25724491 PMCID: PMC4551123

Somanna NK, Valente AJ, Krenz M, Fay WP, Delafontaine P, Chandrasekar B. e Nox1/4 dual inhibitor GKT137831 or Nox4 knockdown inhibits Angiotensin-II-induced adult mouse cardiac broblast proliferation and migration. AT1 physically associates with Nox4. J Cell Physiol. 2015 (in press; PMID: 26445208)

Active Grants

1R01HL116525-01, Research Project Grant (R01), NIH/NHLBI, Krenz (PI) Title: SHP2 controls cardiac stress adaptation 07/24/2013 – 06/30/2017 (total cost $1,437,357) Role: PI

NIH/ NCATS, TRND program Use of Rapamycin for the Treatment of Hypertrophic Cardiomyopathy in Patients with LEOPARD Syndrome (Key Investigator Kontaridis) 06/01/2013 – 05/31/2017 Role: Co-Investigator, 0% effort

Professional Service

2014 – present Graduate and Undergraduate Mentor
2012 – present Dissertation Committee Member
2009 – present Member, Dalton Safety Committee
2011 – present Member, Dalton Science Display Committee
2011 – present Member, MPP Graduate Education Committee
2011 – present MPP representative, School of Medicine Faculty Affairs Council
1999 – present Member, International Society for Heart Research (ISHR), North American Section
2010 – present Member, American Heart Association
2010 – present Member, American Physiological Society

Reviewer: American Journal of Physiology, Heart and Circulation Physiology, American Journal of Physiology, Endocrinology and Metabolism, Basic Research in Cardiology, Biochimica et Biophysica Acta (BBA), Molecular Cell Research, Circulation Research, Coronary Artery Disease, FEBS Letters, Frontiers in Integrative Physiology, International Journal of Clinical Cardiology, Journal of Applied Physiology, Journal of Cardiac Failure, Journal of Molecular and Cellular Cardiology, Journal of Vascular Research, Nature, PLOS ONE, Proceedings of the National Academy of Sciences, Trends in Cardiovascular Medicine, Yonsei Medical Journal

Grant Reviewer:

Jan 2015 Congressionally Directed Medical Research Program (CDMRP), Review Panel member
Mar 2015 NIH/NHLBI Special Emphasis Panel ZHL1 CSR-H (M3) 1 (R13/U13), member
Oct 2015 AHA Study Section CV Dev BSc1, member
Dec 2015 Congressionally Directed Medical Research Program (CDMRP), Review Panel Chair
Oct 2015 AHA Study Section CV Dev BSc1, member

Harold Laughlin

Publications

Credeur DP, Holwerda SW, Restaino RM, King PM, Crutcher KL, Laughlin MH, Padilla J, and Fadel PJ. Characterizing rapid onset vasodilation to single muscle contractions in the human leg. J Appl Physiol (1985). 2015 Feb 15;118(4):455-64

Laughlin MH, Padilla J, Jenkins NT, Thorne PK, Martin JS, Rector RS, Akter S, Davis JW. Exercise-induced differential changes in gene expression among arterioles of skeletal muscles of obese rats. J Appl Physiol 2015 Sep 15;119(6):583-603.

Laughlin MH, Padilla J, Jenkins NT, Thorne PK, Martin JS, Rector RS, Akter S, Davis JW. Exercise training causes differential changes in gene expression in diaphragm arteries and 2A arterioles of obese rats. J Appl Physiol (1985). 2015 Sep 15;119(6):604-16.

Sheldon RD, Padilla J, Jenkins NT, Laughlin MH, Rector RS. Chronic NOS inhibition accelerates NAFLD progression in an obese rat model. Am J Physiol Gastrointest Liver Physiol 2015 Mar 15;308(6):G540-9. PMID: 25573175

Bender SB, and Laughlin MH. Modulation of endothelial cell phenotype by physical activity: impact on obesity-related endothelial dysfunction. Am J Physiol Heart Circ Physiol. 2015 Jul 1;309(1):H1-8. PMID: 25934096

Crissey JM, Padilla J, Vieira-Potter VJ, Thorne PK, Koch LG, Britton SL, Thyfault JP, Laughlin MH. Divergent role of nitric oxide in insulin-stimulated aortic vasorelaxation between low- and high-intrinsic aerobic capacity rats. Physiol Rep.. 2015 2015 Jul;3(7). pii: e12459. doi: 10.14814/phy2.12459. PMID: 26197933

Linden MA, Lopez KT, Fletcher JA, Morris EM, Meers GM, Siddique S, Laughlin MH, Sowers JR, Thyfault JP, Ibdah JA, Rector RS. Combining metformin therapy with caloric restriction for the management of type 2 diabetes and nonalcoholic fatty liver disease in obese rats. Appl Physiol Nutr Metab. 2015 Oct;40(10):1038-47. PMID: 26394261

Thijssen DH, Schreuder TH, Newcomer SW, Laughlin MH, Hopman MT, Green DJ. Impact of 2-Weeks Continuous Increase in Retrograde Shear Stress on Brachial Artery Vasomotor Function in Young and Older Men. J Am Heart Assoc. 2015 Sep 28;4(10). pii: e001968. doi: 10.1161/JAHA.115.001968.. PMID: 26416875

Laughlin MH. Physical activity-induced remodeling of vasculature in skeletal muscle: role in treatment of type 2 diabetes. J Appl Physiol In Press. 2015

Presentations

Laughlin, MH: Mechanisms Responsible for Beneficial Effects of Physical Activity on Atherosclerosis and Coronary Artery Disease. Keynote speaker at the Annual Indiana Physiological Society meeting. Marian University, Indianapolis IN February 21, 2015.

Laughlin, MH: Physical activity-induced remodeling of vasculature in striated muscle: treatment of T2D. Annual Experimental Biology Meeting, Boston MA, March 25-April 2, 2015.

Laughlin, MH: How does physical activity produce beneficial effects on atherosclerosis and coronary heart disease? Texas A&M Distinguished Lecture Series, April 7-8, 2015.

Active Grants
National Institutes of Health, R01; "Training: Muscle Blood Flow and Capillary Dynamics; Amount: $1,000,000 total ($250,000 for 2010); Duration: 5/1/1981-4/30/2015; 25% effort; PI. (Submitted as a new RO1 October, 2015)

Service Activity:
Manuscript Review for Journals:

1980-Present Avia. Space Environ. Med	1980-Present J. Applied Physiol
1980-Present Med. Sci. Sports Exercise	1981-Present Am. J. Physiol.
1985-Present Hypertension	1990-Present Microvascular Research
1993-Present Circulation	1993-Present Circulation Research
1994-Present Microcirculation	1994-Present Microcirculation
2015-Present Europ J Prevent. Cardiol.	

2013-2016 Elected to Council of the American Physiological Society
2015 Scholar Award from the Health and Kinesiology Department at Texas A&M University.
2015 Adolph Lecture, presented at EB meetings 2015.
2015-Present Europ J Prevent. Cardiol.
2013-2016 Elected to Council of the American Physiological Society
2015 Scholar Award from the Health and Kinesiology Department at Texas A&M University.
2015 Adolph Lecture, presented at EB meetings 2015.
2015 Aging Systems and Geriatrics (ASG) Study Section, National Institutes of Health Special Emphasis Review Group 2015/05, ASG meeting, 03/04/2015 - 03/05/2015.

GRANT REVIEW COMMITTEES:
7/1/1990-Present National Institutes of Health Reviewer reserve
2015 Skeletal Muscle Exercise Physiology (SMEP) Study Section, National Institutes of Health Special Em phasis Review Group 2015/10, SMEP meeting, 10/08/2015 - 10/09/2015.

Editorial Board

1987-Present	Journal of Applied Physiology
2005-Present	Medicine and Science in Sports & Exercise
2011-present	Journal of Geriatric Cardiology (JGC)

Yayun Liang

Active Grants
Liang, Yayun (Co-I), Hyder, S. M (PI), "Blocking Androgen Receptor Activity with an Inhibitor of Cholesterol Synthesis: A Novel Means of Suppressing Prostate Cancer" Department of Defense Breast Cancer Pgm. Total cost: $151500.00 (6/1/14-5/30/15).

Professional services
Member of The Science Advisory Board (Since 2014). Editorial Board of Journal of Heart Health (Since 2014)
Active member of American Association for Cancer Research (1997-present)
Active member of Women in Cancer Research (2002-present)
Active member of Minorities in Cancer Research (2009-present)

Luis Martinez-Lemus

Publications

Jia, G., A.R. Aroor, L.A. Martinez-Lemus and J.R. Sowers, 2015. Mitochondrial functional impairment in response to environmental toxins in the cardiorenal metabolic syndrome. Archives of toxicology 89: 147-153

Jia, G., L.A. Martinez-Lemus, and J.R. Sowers, 2015. Interaction of Adipogenesis and Angiogenesis in Dietary-Induced Obesity. Diabetes 64: 2326-2328.

DeMarco, V.G., J. Habibi, G. Jia, A.R. Aroor, F.I. Ramirez-Perez, L.A. Martinez-Lemus, S.B. Bender, M. Garro, M.R. Hayden, Z. Sun, G.A. Meininger, C. Manrique, A. Whaley-Connell, J. R. and Sowers, 2015. Low-Dose Mineralocorticoid Receptor Blockade Prevents Western Diet-Induced Arterial Stiffening in Female Mice. Hypertension 66: 99-107.

Bender, S.B., J.A. Castorena-Gonzalez, M. Garro, C.C. Reyes-Aldasoro, J.R. Sowers, V.G. DeMarco, and L.A. Martinez-Lemus, 2015. Regional variation in arterial stiffening and dysfunction in Western diet-induced obesity. Am. J. Physiol. 309:H574-582. (Featured in Editorial)

Jia, G., A.R. Aroor, V.G. DeMarco, L.A. Martinez-Lemus, G.A. Meininger, and J.R. Sowers, 2015. Vascular stiffness in insulin resistance and obesity. Front. Physiol. 6:231.

Jia, G., J. Habibi, V.G. DeMarco, L.A. Martinez-Lemus, L. Ma, A.T. Whaley-Connell, A.R. Aroor, T.L. Domeier, Y. Zhu, G.A. Meininger, K.B. Mueller, I.Z. Jaffe, and J.R. Sowers, 2015. Endothelial Mineralocorticoid Receptor Deletion Prevents Diet-Induced Cardiac Diastolic Dysfunction in Females. Hypertension. doi 10.1161/HYPERTENSIONAHA.115.06015

Foote, C.A., J.A. Castorena-Gonzalez, M.C. Staiculescu, P.S. Clifford, M.A. Hill, G.A. Meininger, and L.A. Martinez-Lemus, 2015. Brief serotonin exposure initiates arteriolar inward remodeling processes in vivo that involve transglutaminase activation and actin cytoskeleton reorganization. Am J Physiol Heart Circ Physiol:ajpheart 00666 02015. doi 10.1152/ajpheart.00666.2015

Book Chapters

Martinez-Lemus, L.A. and M.H. Laughlin. Microcirculation, Lymph and Edema. In Dukes' Physiology of Domestic Animals. 13th Edition. Ed: William O. Reece. Wiley-Blackwell, Iowa, p. 372-385, 2015.

Presentations

Vascular Smooth muscle cell stiffness and arterial remodeling. University of Southern Denmark. Odense, Denmark (June 16, 2015).
Vascular Smooth muscle cell stiffness and arterial remodeling. Danish Cardiovascular Research Academy, 2015 Summer Meeting at The Sandbjerg Estate. Sonderborg, Denmark (June 11, 2015).
Local mechanisms of resistance artery remodeling. University of Calgary. Calgary, Canada. (February 18, 2015).

Active Grants

National Science Foundation (NSF). "Collaborative Research: Sens-and Act Systems for Substance Release Modeling Drug Delivery Triggered by Immune-Sensing Based on Nanostructured Electrodes." Multi-Investigator Grant. Principal Investigator, Evgeny Katz. Co-Principal Investigator Luis A. Martinez-Lemus (5% Effort), $440,541.00 for 2014-2017.

National Institutes of Health (NIH). "Mechanisms of Microvascular Remodeling Progression." Principal Investigator, Luis A. Martinez-Lemus (40% Effort), $1,250,000.00 for 2009-2014. One year No-Cost extension to 2015. Scored at 120 (3.5%).

Professional Service

- European Society for Microcirculation
- American Physiological Society
- Microcirculatory Society
- Poultry Science Association
- Appointed member of the subcommittee for animal studies at the Harry S. Truman Memorial Veterans Hospital, 800 Hospital Dr. Columbia, MO 65201 (December 2013-2016)
- International Society for Resistance Arteries (ISRA) 2014 Tri-annual Conference Organization Commit tee (2013-2014)
- American Physiological Society-Cardiovascular Section: NIH Liaison Committee (2012-2015)
- Appointed member of the Dalton Cardiovascular Research Center Safety Committee (2009-Present).
- Appointed member of the Appointment and Promotions Committee for non-tenure track faculty within the Dalton Cardiovascular Research Center, University of Missouri-Columbia (2006-Present).

Editorship

- Editorial Board Member for "CardioRenal Medicine," 2012-Present.
- Review Editorial Board for "Frontiers in Vascular Physiology," 2010-Present.
- Editorial Board member for "Microcirculation," 2010-2015

Reviewer

- Journal of Vascular Research
- Arteriosclerosis, Thrombosis, and Vascular Biology
- American Journal of Physiology
- Heart and Circulatory Physiology
- Regulatory, Integrative and Comparative Physiology
- Clinical and Experimental Medicine
- Experimental Physiology
- Clinical and Experimental Pharmacology and Physiology
- Poultry Science
- The Anatomical Record
- PLOS-One
- Anatomical Record
- Hypertension
- Microcirculation
- Hospital Practice
- BioMed Central Cell Biology

2009-Current Teaching advance imaging techniques (Fuorescence, Confocal and Multiphoton Microcopy) to graduate students at the Department of Medical Pharmacology and Physiology, University of Missouri-Columbia as part of the course entitled "Skills in Biomedical Research" (course # MPP8420).
2010-Current Coordinating the Journal Club for graduate students at the Department of Medical Pharmacology and Physiology, University of Missouri-Columbia (course # MPP9422).
2008-Current Teaching Microvascular Function to graduate students in the Department of Medical Pharmacology and Physiology, University of Missouri-Columbia as part of the course entitled "Microvascular Circulatory Function" (course # MPP9434).

Gerald Meininger

Publications

Sehgel N.L., Z. Sun, Z.Hong, W.C. Hunter, M.A. Hill, D.E. Vatner, S.F. Vatner, G.A. Meininger. Augmented Vascular Smooth Muscle Cell Stiffness and Adhesion when Hypertension is Superimposed on Aging. Hypertension, 65(2): 370-377, 2015. (Shared Senior Authorship and Corresponding Author) DOI: 10.1161, PMID: 23709594, PMCID: PMC3840243.

Hong Z., K.J. Reeves, Z. Sun, Z. Li, N.J. Brown, G.A. Meininger. Vascular smooth muscle cell stiffness and adhesion to collagen I modified by vasoactive agonists. PLOS ONE, 10(3):1-12, 2015. DOI: 10.1371, PMID: 25745858

DeMarco, V.G., J. Habibi, G. Jia, A.R. Aroor, F. Ramirez, L. Martinez-Lemus, S.B. Bender, M. Garro, M.R. Hayden, Z. Sun, G.A. Meininger, C. Manrique, A. Whaley-Connell, and J.R. Sowers. Very Low Dose Mineralocorticoid Receptor Blockade Prevents Western Diet-induced Arterial Stiffening in Female Mice. Hypertension, 66:00-00, 2015. DOI: 10.1161 (In Press)

Domeier, T.L., M.E. Nance, J.T. Whitfield, Y. Zhu, A.M. Gibson, L.M. Hanft, K.S. Campbell, G.A. Meininger, K.S. McDonald1, and S.S. Segal. Attenuated sarcomere lengthening of the aged murine left ventricle observed using two-photon fluorescence microscopy. American Journal of Physiology: Heart and Circulatory Physiology, 2015. (In Press)

Jia G., J. Habibi, V.G. DeMarco, L.A. Martinez-Lemus, L. Ma, Adam T. Whaley-Connell, G.A. Meininger, K. Barrett Mueller, I.Z. Jaffe and J.R. Sowers. Endothelial mineralocorticoid receptor signaling contributions to cardiac diastolic dysfunction in females, Hypertension, 66:00-00, 2015. DOI: 10.1161 (In Press)

Foote C.A., J.A. Castorena-Gonzalez, M.C. Staiculescu, M.A. Hill, G.A. Meininger and L.A. Martinez-Lemus. Transient serotonin exposure induces arteriolar inward remodeling in vivo via a mechanism contingent on transglutaminase activation and actin cytoskeleton reorganization. American Journal of Physiology: Heart and Circulatory Physiology, 2015. (In Press)

Zhao X., D.E. Vatner, T. McNulty, G. Lee, Y. Tian, S.Bishop, Z. Sun, G.A. Meininger, S.F. Vatner. Importance of abdominal over thoracic aortic stiffness with aging in primates. Atherosclerosis, Thrombosis, and Vascular Biology, 2015. (In Press)

Sun Z., S. Huang, Z. Li and G.A. Meininger. A mechanotransducing role for N-cadherin in vascular smooth muscle. Journal of Physiology, 2015. (In revision)

Wang X., L. Nichols, E.A. Grunz-Borgmann, Z. Sun, G.A. Meininger, J.M. Catania, R.C. Burghardt, A.R. Parrish. The role of the age dependent loss of fascin 2 in increased acute kidney injury. American Journal of Physiology: Renal Physiology, 2015. (In revision)

Jia G., J. Habibi, A.R. Aroor, L.A. Martinez-Lemus V. G. DeMarco, F. I Ramirez-Perez, Z. Sun, G.A. Meininger, K.B. Mueller, I.Z. Jaffe, and J.R. Sowers. Endothelial mineralocorticoid receptor mediates diet induced aortic stiffness in females. Circulation Research 2015 (Submitted)

"Mechanoadaptive Properties of Vascular Smooth Muscle: Does this change our view of contractile activation?". Department of Medical Physiology and Cardiovascular Research Institute, College of Medicine, Texas A&M University, College Station, TX, May 12-14, 2015.

Hill, M.A., S.R. Ella, A. Stupica, P. Bagher, Z. Nourian, M. Collins, C.J. Garland, P.E. Clifford, L. Martinez-Lemus, K.A. Dora and G.A. Meininger. Image-based Approaches for Probing the Complex Structural and Functional Arrangement of the Arteriolar Wall. Methods, 2015. (In preparation)

Hong Z., J.P. Trzeciakowski, M. Jin, Z. Li, Z. Sun, N.L. Sehgel, S.F. Vatner, G.A. Meininger. A method to analyze the spontaneous oscillations in cell stiffness and adhesion measured using AFM. Nanomedicine, 2015. (In Preparation)
Hodara V., V. Saroja Voruganti, Q. Meng, A.G. Comuzzie, R. Baker, K. Rice, Z. Hong, G.A. Meininger, J.L. Vande-Berg, Q. Shi. Mononuclear Cell Activation Elicited by Diets High in Simple Carbohydrates in Conjunction with Saturated Versus Unsaturated Fat in Baboons. 2015 (In Preparation)

Presentations
"The mechanoadaptive properties of vascular smooth muscle". Inserm Unit Laboratory of Vascular Biology, Georges Pompidou European Hospital, Paris, France, February 3, 2015.

"The mechanoadaptive properties of vascular smooth muscle". UMR Inserm U, Faculty of Medicine, Laboratory, Platforms and Clinical Research Center, Nancy, France, February 4, 2015.

Active Grants
National Institutes of Health-National Heart, Lung and Blood Institute, P01, Program Project Grant, "Mechanisms of Microvascular Control and Coordination in Health and Disease", Project Director and Principal Investigator, G.A. Meininger (30% effort), with MJ Davis, and RJ Korthuis as project leaders, $5,549,287 direct ($8,471,385 total) for 04/01/2010-03/31/2015.

As Co-Investigator National Institutes of Health-National Heart, Lung and Blood Institute, R01, Intrinsic vascular smooth muscle cell stiffness, Principal Investigator, S. Vatner, Co-Investigator, G.A. Meininger (5%), $213,827 (total for 5 year sub-contract period, direct and indirect), 04/01/10-03/31/15.

National Institutes of Health-National Heart, Lung and Blood Institute, R01, SFRP2, cell survival, and coronary vascular angiogenesis, Principal Investigator, D.E. Vatner (Rutgers University), Co-Investigator, G.A. Meininger (5%), $310,670 for 07/01/2013-08/30/2018.

Professional Service

World Congress for Microcirculation
International Liaison Committee, Chair-Elect, 2014-2015; Chair, 2015-2022.
Core Scientific Committee for organization of the World Congress for Microcirculation in Kyoto, Japan, 2015.
Awards Committee, Member, 2015.

Professional Committee of Qi-Blood for World Federation of Chinese Medicine Societies
Adviser Professional Committee Council, Modern Traditional Chinese Medicine, 2014-Present.

18th European congress of the ESCHM to be held in Lisbon in 5th -8th June 2016
International Advisory Committee, 2015-Present.

Scientific Journals
Editor-in-Chief for Frontiers in Vascular Physiology, January 2010-present.
Co-Editor (US) for Journal of Vascular Research, August 1999-Present.

Editorial Boards
Editorial Board Member for Frontiers in Cardiovascular and Smooth Muscle Pharmacology, 2010-present.
Editorial Board Member for International Journal of Physiology, Pathophysiology and Pharmacology, 2010-present.
Editorial Board Member for Nanotechnology, Science and Applications, 2007-present.
Editorial Board Member for Microcirculation, 1993-1999; 2003-2009; 2010-2012.
Member of the Strategic Planning and Resource Advisory Council (SPRAC), Office of the Chancellor, University of Missouri, July 2012-June 2015.
Member of the Internal Medicine Research Council (IMRC), Department of Internal Medicine, School of Medicine, University of Missouri, September 2012-present.
Member of Advisory Committee Meeting for T90/R90 Clinical Biodetective Training Grant, March 2009-present.
Member of School of Medicine Advisory Committee on Research Space, November 2010-present.
Member of Graduate and Doctoral Faculty at the University of Missouri-Columbia, September 2005-Present.
Member Council of Chairs, School of Medicine, University of Missouri-Columbia, September 2005-Present.
Member Center Directors Council, Office of Research, University of Missouri-Columbia, October 2005-Present.
Member Core Imaging Facility, Dalton Cardiovascular Research Center, University of Missouri-Columbia, September 2005-Present.
12th Annual meeting of Mechanisms of Vasodilation Symposium to be held at the Mayo Clinic, Rochester, MN 7th-9th November 2016
Scientific Committee, 2015-2016**.**
Co- Organizer and Co-Chair of Symposium on Vascular Stiffness, a novel therapeutic approach for hypertension for the American Society of Pharmacology and Experimental Therapeutics, San Diego, CA, April 2015.
Co-Organizer and Co-Chair Symposium on Mechanobiology: Roles of Cellular and Non-Cellular Contributors for the 10th World Congress for Microcirculation, Kyoto, Japan, September 25-27, 2015.
Physiology-Medical Student Teaching

2007-2015 Problem based learning facilitator
Microcirculatory Function – A graduate student course.

2009-2015: Atomic Force Microscopy as a biological research tool.

Cellular and Molecular Light and Electron Microscopy Course – A graduate student course
2009-2015; Atomic Force Microscopy as a biological research tool.

Mark Milanick

Professional Services
Graduate Student Committees

Journal Reviewer: Journal of Physiology

Red Cell Club, 1979-present
National Association of Biology Teachers, 2011-present

2010-present Chair, Campus Minority Affairs Committee
2008-present Departmental Doctoral Faculty Review Committee
2012-present MU Status of Women Committee

Nicole Nichols

Publications

Nichols, N.L., Satriotomo, I., Harrigan, D.J. and Mitchell, G.S. (2015). Acute intermittent hypoxia induced phrenic long-term facilitation despite increased SOD1 expression in a rat model of ALS. Exper. Neurol. 273: 138-150.

Nichols, N.L., Vinit, S., Bauernschmidt, L. and Mitchell, G.S. (2015). Respiratory function after selective respiratory motor neuron death from intrapleural CTB-saporin injections. Exper. Neurol. 267: 18-29

Satriotomo, I., Nichols, N.L., Dale, E.A., Emery, A.T., Dahlberg, J. and Mitchell, G.S. Repetitive acute intermittent hypoxia increases growth/neurotrophic factor expression in non-respiratory motor neurons. In revision at Neuroscience.

Presentations

N. Nichols. Novel Strategies to improve respiratory function in ALS. March 2015. Featured Topic Symposium on Respiratory related disorders in aging and neurodegeneration, Experimental Biology International Meeting, Boston, MA.

N. Nichols. Trying to breathe with ALS. December 2015. Saturday Morning Science lecture series. University of Missouri. Columbia, MO.

Active Grants

2015-present	NIH K99/R00 Pathway to Independence Grant – NHLBI (R00)
2014-2015	NIH K99/R00 Pathway to Independence Grant – NHLBI (K99)

Professional services

2015	Ad hoc reviewer for J. of Physiology
2015	Ad hoc reviewer for Respiratory Physiology & Neurobiology
2015-present	Member of the American Thoracic Society Assembly on Sleep and Respiratory Neurobiology Program Committee

American Physiological Society
American Thoracic Society
Society for Neuroscience

Jaume Padilla

Publications

Montero D, Pierce GL, Stehouwer CDA, Padilla J, Thijssen DHJ. The impact of age on vascular smooth muscle function in humans. J Hypertens 33(3):445-53, 2015.

Fairfax ST, Padilla J, Vianna LC, Holwerda SW, Davis MJ, Fadel PJ. Myogenic responses occur on a beat-to-beat basis in the resting human limb. Am J Physiol Heart Circ Physiol 308(1):H59-67, 2015.

Credeur DP, Holwerda SW, Restaino RM, King PM, Crutcher KL, Laughlin MH, Padilla J, Fadel PJ. Characterizing rapid onset vasodilation to single muscle contractions in the human leg. J Appl Physiol 118(4):455-64, 2015.

Vieira-Potter VJ, Padilla J, Park J, Welly R, Scroggins R, Britton S, Koch L, Jenkins NT, Crissey J, Zidon T, Morris M, Meers G, Thyfault JP. Female rats selectively bred for high intrinsic aerobic fitness are protected from ovariectomy-associated metabolic dysfunction. Am J Physiol Reg Integ Comp Physiol 308(6):R530-42, 2015.

Sheldon RD, Padilla J, Jenkins NT, Laughlin MH, Rector RS. Chronic NOS inhibition accelerates NAFLD progression in an obese rat model. Am J Physiol Gastrointest Liver Physiol 308(6):G540-9, 2015.

Bender BS, De Marco VG, Padilla J, Jenkins NT, Habibi J, Garro M, Pulakat L, Aroor AR, Jaffe IZ, Sowers JR. Minerolocorticoid receptor antagonism treats obesity-associated cardiac diastolic dysfunction. Hypertension 65(5):1082-8, 2015.

Montero D, Walther G, Diaz-Cañestro C, Pyke KE, Padilla J. Microvascular dilator function in athletes: a systematic review and meta-analysis. Med Sci Sports Exerc 47(7):1485-94, 2015.

Professional services

2013-Present Assistant Professor, Department of Nutrition & Exercise Physiology, University of Missouri, Columbia

2013- Present Joint Assistant Professor, Departments of Child Health and Dalton Cardiovascular Research Center, University of Missouri, Columbia, MO

Journal Reviewer (Ad hoc)
2007-Present British Journal of Sports Medicine
2008-Present Free Radical Biology and Medicine
2008-Present European Journal of Applied Physiology
2008-Present Journal of Applied Physiology
2009-Present Journal of the American College of Nutrition
2009-Present American Journal of Physiology Heart Circulatory Physiology
2010-Present Medicine and Science in Sports and Exercise
2010-Present Clinical Physiology and Functional Imaging
2010-Present Hypertension

2011-Present Experimental Physiology
2011-Present Hypertension Research
2011-Present American Journal of Hypertension
2012-Present Journal of Physiology
2012-Present Clinical Science
2012-Present Vascular Medicine
2014-Present Journal of Physiology and Pharmacology
2014-Present Journal of Biological

Luis Polo-Parada

Publications
Jeremy B. Essner, Charles H. Laber, Sudhir Ravula, Luis Polo-Parada and Gary A. Baker. (2015). Pee-dots: biocompatible fluorescent carbon dots derived from the upcycling of urine. Green Chem., 2016,18, 243-250 DOI: 10.1039/C5GC02032H.First published online 21 Sep 2015

Active Grants
5/2014-5/2015 $120,000 Mizzolar: A Hub for Research & Training in sustainable Carbon Based Solar Energy. Co-PI, 33% FTE The Mizzou Advantage imitative.

Professional Service
Advisor Ph. D. Students
Advisor Post-Doctoral

- School of Medicine Research Council. 2012-2015
- MU PREP (Post-baccalaureate Research Education Program. 2008-present

Committee on Committees 2013-2016
School of Medicine Research Council. 2012-2015
American Heart Association 2004-present
Society for Neuroscience 1999-present
Biophysical Society 1994-present

Advisory Committees
The National Academies. Advisers to the Nation on Science, Engineering and Medicine.
Americana Heart Association.
International Mentoring Programs
American Heart Association.

Editorial Board
Medicinal Chemistry: Current Research
Journal of Biochips & Tissue Chips
Journal of Modern Physics

Ad Hoc reviewer
Journal of Neuroscience Methods
Journal of Neuroendocrinology
Journal of Applied Physiology
Toxins
American Journal of Physiology –Heart and Circulatory Physiology
RSC Analyst

Steven Segal

Publications

Socha MJ, Boerman EM, Behringer EJ, Shaw RL, Domeier TL and Segal SS. Advanced age protects microvascular endothelium from aberrant Ca2+ influx and cell death induced by hydrogen peroxide. J Physiol 593.9: 2155–2169, 2015.

Boerman EM and Segal SS. Depressed perivascular sensory innervation of mouse mesenteric arteries with advanced age. J Physiol 2015: Accepted Article; DOI: 10.1113/JP270710.

Behringer EJ and Segal SS. Membrane potential governs calcium influx into microvascular endothelium: Integral role for muscarinic receptor activation. J Physiol 293.20: 4531-4548, 2015.

Segal, SS. Integration and Modulation of Intercellular Signalling Underlying Blood Flow Control. J Vasc Res 52:136-157, 2015

Presentations

University of Western Ontario, London, Ontario, Canada (04/16/2015)
"Integration and Modulation of Intercellular Signaling Underlying Blood Flow Control" (AC Burton invited lectureship)
University of California, San Diego CA (09/28/2015)
"Modulating Intercellular Signaling Underlying Blood Flow Control: Roles for Nerves and K+ Channels"
University of Missouri, Biological Sciences (10/28/2015)
"Mechanisms Underlying Neuromodulation of Blood Flow Control"

Active Grants

R37 HL041026; National Institutes of Health/NHLBI (Segal, PI: MERIT Award)
Title: "Intercellular Coordination of Blood Flow Control" Project Period: 09/01/2008-02/28/2019.

R01 HL-122608; National Institutes of Health/NHLBI (Davis MJ, PI). Title: "Conduction within and along the lymphatic vascular wall" Project Period: 01/01/15 - 12/31/18. Role: Co-Investigator

Professional Service

Editorships
Journal of Physiology: Reviewing Editor
Editorial Boards
American Journal of Physiology: Heart and Circulatory Physiology
Journal of Vascular Research
Institutional
Doctoral Faculty: Medical Pharmacology and Physiology; Biomedical Sciences
Faculty Review Committee, MU School of Medicine
Fellow, MU Interdisciplinary Center on Aging
Promotion and Tenure Committee: Medical Pharmacology and Physiology

Yoshiro Sohma

Active Grants
2013.4 – 2016.3 $160,000., $130,000. Japan Society for the Promotion of Science. Grant-in-Aid for Scientific Research (B): 25293049, PI, "Mechanism of ABC transporters studied by direct observations using high-speed Atomic Force Microscopy".

Professional Services
Society of General Physiologist (USA)
Biophysical Society (USA)
The Physiological Society (UK)
Physiological Society of Japan
Biophysical Society of Japan
The Japanese Pharmacological Society

Editorial Board: 2006.4 – present Member, Editorial Board, Journal of the Physiological Society of Japan.

James Sowers

Publications

Caveolin-1 in Cardiovascular Disease: A Double-Edged Sword. Jia G, Sowers JR. Diabetes. 2015 Nov;64(11):3645-7. doi: 10.2337/dbi15-0005. No abstract available. PMID: 26494216

Endothelial Mineralocorticoid Receptor Deletion Prevents Diet-Induced Cardiac Diastolic Dysfunction in Females. Jia G, Habibi J, DeMarco VG, Martinez-Lemus LA, Ma L, Whaley-Connell AT, Aroor AR, Domeier TL, Zhu Y, Meininger GA, Barrett Mueller K, Jaffe IZ, Sowers JR. Hypertension. 2015 Dec;66(6):1159-67. doi: 10.1161/HYPERTENSIONAHA.115.06015. Epub 2015 Oct 5. PMID: 26441470

Combining metformin therapy with caloric restriction for the management of type 2 diabetes and nonalcoholic fatty liver disease in obese rats. Linden MA, Lopez KT, Fletcher JA, Morris EM, Meers GM, Siddique S, Laughlin MH, Sowers JR, Thyfault JP, Ibdah JA, Rector RS. Appl Physiol Nutr Metab. 2015 Oct;40(10):1038-47. doi: 10.1139/apnm-2015-0236. Epub 2015 Jun 24. PMID: 26394261

Quantification of histone modifications by parallel-reaction monitoring: a method validation. Sowers JL, Mirfattah B, Xu P, Tang H, Park IY, Walker C, Wu P, Laezza F, Sowers LC, Zhang K. Anal Chem. 2015 Oct 6;87(19):10006-14. doi: 10.1021/acs.analchem.5b02615. PMID: 26356480

Vascular stiffness in insulin resistance and obesity. Jia G, Aroor AR, DeMarco VG, Martinez-Lemus LA, Meininger GA, Sowers JR. Front Physiol. 2015 Aug 14;6:231. doi: 10.3389/fphys.2015.00231. eCollection 2015. Review. PMID: 26321962

The VASP Road to NAFLD: A Macrophage Detour. Lastra G, Manrique C, Jia G, Sowers JR. Diabetes. 2015 Aug;64(8):2711-3. doi: 10.2337/db15-0551. No abstract available. PMID: 26207036

Interaction of Adipogenesis and Angiogenesis in Dietary-Induced Obesity. Jia G, Martinez-Lemus LA, Sowers JR. Diabetes. 2015 Jul;64(7):2326-8. doi: 10.2337/db15-0202. No abstract available. PMID: 26106192

Regional variation in arterial stiffening and dysfunction in Western diet-induced obesity. Bender SB, Castorena-Gonzalez JA, Garro M, Reyes-Aldasoro CC, Sowers JR, DeMarco VG, Martinez-Lemus LA. Am J Physiol Heart Circ Physiol. 2015 Aug 15;309(4):H574-82. doi: 10.1152/ajpheart.00155.2015. Epub 2015 Jun 19. PMID: 26092984

Low-Dose Mineralocorticoid Receptor Blockade Prevents Western Diet-Induced Arterial Stiffening in Female Mice. DeMarco VG, Habibi J, Jia G, Aroor AR, Ramirez-Perez FI, Martinez-Lemus LA, Bender SB, Garro M, Hayden MR, Sun Z, Meininger GA, Manrique C, Whaley-Connell A, Sowers JR. Hypertension. 2015 Jul;66(1):99-107. doi: 10.1161/HYPERTENSIONAHA.115.05674. Epub 2015 May 26. PMID: 26015449

Role of perivascular adipose tissue on vascular reactive oxygen species in type 2 diabetes: a give-and-take relationship. Padilla J, Vieira-Potter VJ, Jia G, Sowers JR. Diabetes. 2015 Jun;64(6):1904-6. doi: 10.2337/db15-0096. No abstract available. PMID: 25999534

Active Grants

2012-2016 BLR&D, Interactions of the RAAS and a Western Diet on Insulin Metabolic Actions, James R. Sowers (PI), 650,000

2011-2016 NHLBI, Ang II and Overnutrition and Insulin resistance in Cardiovascular Tissue, James R. Sow ers (PI), $1,250,000.

Professional Service

Professional consultation (other patient care)

- Public presentations as an expert in endocrinology, metabolism and hypertension and vascular medicine
- No consulting to public agencies, foundations, or professional associations

Journal Editorial activity

Editor In Chief –

Cardiorenal Medicine 2010-present

Associate Editor – Diabetes, Journal of Hypertension 2011-present

Editorial Board Memberships

- 2006-present Journal of Hypertension
- 2006-present Journal of American Society of Hypertension
- 2004-present Cardiovascular Drug Reviews
- 2002-present American Journal Clinical Hypertension
- 2002-present American Journal Hypertension
- 1994-present Journal of Human Hypertension
- 1991-present Metabolism
- 1991-present Hypertension

Editorships - Reviewer

New England Journal of Medicine, Hypertension, Journal of Biological Chemistry, Circulation, Circulation Research, American Journal of Physiology, Diabetes, Journal of Clinical Investigation, Journal of Clinical Endocrinology and Metabolism, Endocrinology, American Journal of Medicine, Archives of Internal Medicine, Annals Internal Medicine, Journal American Medical Association, and others. Other professionally related service

- NIH, VA Merit Board, American Heart National Reviews Committees
- Department of Veterans Affairs (VA) Joint Biomedical Laboratory Research and Development and Clini cal Science Research and Development Scientific Merit Review Board
- VCMB (Vascular Cell Molecular Biology) Study Section - NIH
- Data Monitoring Committee "Combination Angiotensin Receptor Blocker and Angiotensin converting Enzyme Inhibitor for Treatment of Diabetic Nephropathy. Cooperative Studies Program Coordinating Center. VA Connecticut Healthcare System
- Microcirculation Study Section – NIH

National and International Boards and Committees

- Positions held in professional associations
- Educational Committee - High Blood Pressure Council
- Scientific Awards Committee - American Society of Hypertension
- External Advisory Board – COBRE grant "Hypertension and Cardiorenal Disease Research Center, John Hall, University of Mississippi

State and Local Boards and Committees

- University of Missouri Internal Medicine Research Council, Chiar – 2011-present
- SOM Administrators Research Council (ARC) 2004-present
- Truman VA Research and Development Committee 2004-present
- R and D committee; ACCORP Truman VA 2004-present

COMMITTEES AND STUDY SECTIONS
Charter Member VCMB Study Section – NIH – 2010-present

04/2007-Present Director, Center for Diabetes and Cardiovascular Research, Columbia, Missouri
04/2007-Present Director, Division of Endocrinology, Diabetes & Metabolism, University of Missouri-Columbia, Columbia, Missouri
04/2007-Present Vice-Chair for Research, Department of Internal Medicine, University of Missouri-Columbia, Columbia, Missouri

04/2007-Present Professor of Medicine, Physiology & Pharmacology, University of Missouri-Columbia, Missouri
04/2007-Present Staff Physician, Medical Service, Truman VA, Columbia, Missouri

Major Professional Societies

American Society Clinical Investigation
Alpha Omega Alpha, Honor Medical Society
American Physiology Society
Society of Vascular Medicine
American Federation of Clinical Research
Fellow, High Blood Pressure Council
American College of Physicians
Endocrine Society
American Diabetes Association
American Society of Hypertension
American College of Physicians (Fellow)
Southern, Western, and Central Society of Clinical Investigation
International Society of Hypertension
International Society of Hypertension in Blacks
Inter-American Society of Hypertension

Zhe Sun

Publications

Low-Dose Mineralocorticoid Receptor Blockade Prevents Western Diet-Induced Arterial Stiffening in Female Mice. DeMarco VG, Habibi J, Jia G, Aroor AR, Ramirez-Perez FI, Martinez-Lemus LA, Bender SB, Garro M, Hayden MR, Sun Z, Meininger GA, Manrique C, Whaley-Connell A, Sowers JR. Hypertension. 2015 Jul;66(1):99-107. doi: 10.1161/HYPERTENSIONAHA.115.05674. Epub 2015 May 26. PMID: 26015449

Vascular smooth muscle cell stiffness and adhesion to collagen I modified by vasoactive agonists. Hong Z, Reeves KJ, Sun Z, Li Z, Brown NJ, Meininger GA. PLoS One. 2015 Mar 6;10(3):e0119533. doi: 10.1371/journal.pone.0119533. eCollection 2015. PMID: 25745858

Augmented vascular smooth muscle cell stiffness and adhesion when hypertension is superimposed on aging. Sehgel NL, Sun Z, Hong Z, Hunter WC, Hill MA, Vatner DE, Vatner SF, Meininger GA. Hypertension. 2015 Feb;65(2):370-7. doi: 10.1161/HYPERTENSIONAHA.114.04456. Epub 2014 Dec 1. PMID: 25452471

Active Grants

1P01HL095486 (G. Meininger, PI) $ 1,515,000 04/01/2010~03/31/2015
Project Title: Mechanisms of Microvascular Control in Health and Disease
National Institutes of Health- National Heart, Lung and Blood Institute
Role: Co-Investigator, 20% effort

Professional Service

Assistant Research Professor, Dalton Cardiovascular Research Center, University of Missouri-Columbia, November 2005- present Supervisor: Dr. Gerald A. Meininger

Director of the Atomic Force Microscopy Core, Dalton Cardiovascular Research Center, University of Missouri-Columbia, November 2008- present. Supervisor: Dr. Gerald A. Meininger

Sigma Xi, the Scientific Research Society
Microcirculation Society
American Physiological Society
Ad Hoc Reviewer:
American Journal of Physiology: Heart and Circulatory Physiology
Journal of Vascular Research
Journal of Neuroscience Methods
Nature Nanotechnology
Nano-Medicine

Xiaoqin Zou

Publications

Chengfei Yan, Sam Z. Grinter, Benjamin Ryan Merideth, Zhiwei Ma, Xiaoqin Zou*. Iterative knowledge-based scoring functions derived from rigid and flexible decoy structures: Evaluation with the 2013 and 2014 CSAR benchmarks. Journal of Chemical Information and Modeling, Special Issue on CSAR (invited), DOI: 10.1021/acs.jcim.5b00504 [Epub ahead of print, Oct. 2015].

Zhe Wang, Tao Yu, Jian-Ping Sang, Xian-Wu Zou, Chengfei Yan, Xiaoqin Zou*. Computation and simulation of the structural and functional characteristics of the kidney urea transporter. Journal of Physical Chemistry B, 119: 5124–5131, 2015 (featured in the front cover of the issue).

Chengfei Yan, Xiaoqin Zou*. Predicting peptide binding sites on protein surfaces by clustering chemical interactions. Journal of Computational Chemistry. 36:49-61, 2015 (featured in the inside cover of the first issue of 2015, the free issue that is distributed by the journal for advertisement).

Presentations

Chengfei Yan, Xiaoqin Zou*. Predicting Peptide Binding Sites on Protein Surfaces by Clustering Chemical Interactions. Biophysical Society Annual Meeting, Baltimore, Maryland, 2015.

Yayun Liang, Benford Mafuvadze, Xiaoqin Zou, Cynthia Besch-Williford, Salman M Hyder. American Association for Cancer Research (AACR) Annual Meeting, Philadelphia, PA, 2015.

Active Grants

American Chemical Society National Meeting, Symposium on "Molecular Biophysics: Revealing the interplay between different forces and effects in biochemical processes", Boston, Massachusetts, 2015. Funding Agency: NIH Grant # R01GM109980

Project Title: Database and software development for protein-nucleic acid structure prediction Funding Period: February 1, 2015 to November 30, 2019 Total Amount: $1,461,940 Funding Agency: NIH Grant # R01 HL126774 (Role: co-PI)

Project Title: Manipulating IKs as a therapeutic approach to cardiac arrhythmias Funding Period: July 1, 2015 to June 30, 2019 Total Amount: $3,106,906 (My share: $736,800) Leading Principal Investigator: Jianmin Cui Funding Agency: Cystic Fibrosis Foundation Grant # HWANG15G0 (Role: co-PI)

Project Title: Structure-based drug design for cystic fibrosis Funding Period: September 1, 2015 to August 31, 2017 Total Amount: $216,000 Principal Investigator: Tzyh-Chang Hwang (My share: $80000) NSF Grant # 1429294 (role: co-I)

Project Title: MRI: Acquisition of Instrument for Data-intensive Applications with Hybrid Cloud Computing Needs Funding Period: September 1, 2014 – July 31, 2017 Total Amount: $600,408 Principal Investigator: Chi-Ren Shyu

Funding Agency: NSF Grant # 0953839 Project Title: CAREER: A computational approach to template-based structure selection for protein-protein interactions Funding Period: August 1, 2010 to July 31, 2015 Total Amount: $734,016 Principal Investigator: Xiaoqin Zou

Funding Agency: American Heart Association (Midwest Affiliate) Grant # 13GRNT16990076 Project Title: Structure-based screening and discovery of potentiators for KCNQ1 and IKs channels Funding Period: July 1, 2013 – June 30, 2015 Total Amount: $143,000 Principal Investigator: Xiaoqin Zou

Professional Services
Campus Services - Serve as a member in the Campus Parking and Transportation Committee (2011-present).

Member of the Advisory committee for Oversea Outstanding Faculty Recruitment, College of Life Sciences, Zhejiang University, 2014-present.

o NIH study section ad hoc member, 2014, 2015
o Served as a judge for the poster competition of Informatics Symposium, 2012 -2015.
o Served as a judge for Undergraduate Research & Creative Achievements Forum of the University of Missouri-Columbia in 2007 (Physical Science & Engineering Division), 2010, 2013 and 2015 (Life Sciences Division).