Dalton Cardiovascular Research Center 2012
Front picture is a nighttime shot of Dalton Cardiovascular Research Center. Images at top and bottom of picture are from Dalton Investigators.
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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 investigators have $27.3 million in active research funding, have published 242 manuscripts in nationally recognized journals and books and gave 83 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.
DCRC Committees

The Internal Advisory Committee:

Dr. Gerald A. Meininger, Chair
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Dr. Alan Jones
Dr. Ed Blaine
Dr. Virginia Huxley
Dr. Ron Terjung
Dr. Kevin Gillis

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Dr. Luis A. Martinez-Lemus
Dr. David Kline

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Dr. Eileen M. Hasser
Dr. Kevin Gillis

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Dr. Kevin Gillis
Dr. Lane Clarke

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Dr. Christopher Baines, Chair
Dr. Shinghua Ding
Dr. Maike Krenz
Dr. Min Li
Dr. Luis Martinez-Lemus
Laura McClaskey
Director's Office and Administrative Contacts

Director, Dr. Gerald Meininger  
Phone: 573-882-9662 E-mail: meiningerg@missouri.edu

Associate Director, Dr. Michael Hill  
Phone: 573-884-4604 E-mail: hillmi@missouri.edu

Development, Dr. Edward Blaine  
Phone: 573-882-9014 E-mail: blainee@missouri.edu

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

Administrative Associate, Bin Ke  
Phone: 573-882-9502 E-mail: keb@missouri.edu

Grants and Contracts Administrator, Abdelrahman Elhadi, Pre/Post Awards  
Phone: 573-882-7433 E-mail: elhadia@missouri.edu

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

Administrative Assistant, Karen McVay  
Phone: 573-882-7588 E-mail: mcvayk@missouri.edu

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

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

User Support Analyst, John Donahue  
Phone: 573-882-3546 E-mail: donahuejt@missouri.edu

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

Assistant Lab Animal Technician, 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, Meiningher, Milanick, Polo-Parada, Segal, Zou, Sun

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

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

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

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

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

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

Cardiac Muscle, Development & Disease
Investigators: Baines, Krenz, Meiningher, Sun, Polo-Parada, Rubin
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.
Associate Director

Michael A. Hill
Office: 133A DCRC
Phone: 573-884-4601
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.
Principal 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.
Research

Currently, the primary focus of my laboratory is 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 are presently working 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 involves 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 are conducted in conscious, freely moving animals to obtain data that is not compromised by anesthesia or restraint. We are also studying changes in renal morphology, especially distribution of the loops of Henle, using immunohistochemistry techniques.

We continue our interest in neuroendocrine regulation of fluid balance and cardiovascular function, with emphasis on hypertension and heart failure. We are particularly interested in the actions of angiotensin II and vasopressin on brain cardiovascular centers. Our recent 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 is to map the distribution of heightened nervous system activity in the peripheral vascular beds that is associated with chronic angiotensin hypertension. We are also investigating receptor regulation and post receptor signaling associated with angiotensin infusion.

We are 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.
Silvia G. Bompadre

Office Location: 359
Office Phone: 573-884-9474
E-mail: BompadreS@missouri.edu

Education:
PhD in Physics from University of Washington

Appointment: Assistant Professor, Department of Physics

Research
Research in my laboratory focuses on understanding how the CFTR (Cystic Fibrosis Transmembrane conductance Regulator) chloride channel works at the molecular level and how mutations in CFTR cause defective functions. The final goal is to apply the acquired knowledge in therapeutic design for cystic fibrosis. Combining electrophysiology and fluorescence spectroscopy techniques with molecular biology and molecular modeling, studies are set to study CFTR's structure/function which in turn can help design specific compounds for specific disease-associated mutants.

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

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

The first question being posed is: what are the aging mechanisms of decreased proliferation and differentiation of satellite cells, the adult stem cells in skeletal muscle? Experiments are concerned with regulation of p21Cip1/WAF1, p27Kip1, p53, FoxO3a, Sirt1 and other proteins as they regulate proliferation and differentiation.

The second question being posed is: by what mechanisms does physical inactivity trigger metabolic dysfunction? When rats that have voluntarily ran in wheels cease running, specific intra-peritoneal fat masses increase, insulin sensitivity in specific skeletal muscles falls, and enhanced vasodilatation of the aorta is lost. Research is under way to determine molecules responsible for these inactivity effects.
Doug Bowles

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

Education:
PhD University of Texas-Austin

Appointments: Associate Director of the National Center for Gender Physiology
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 in 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.
Research
Our laboratory investigates electrolyte and nutrient transport across epithelial tissues (airway, reproductive and intestinal) during health and disease. The major focus is to understand the role of the cystic fibrosis transmembrane conductance regulator protein (CFTR) in the regulation of acid-base and nutrient transport across alimentary epithelia. CFTR is the protein product of the gene that is mutated in cystic fibrosis (CF) and normally functions in epithelial cells as a cyclic AMP-regulated anion channel. Present studies investigate the role of anion exchange proteins that work with CFTR in promoting bicarbonate transport or that work with Na+ transport proteins for NaCl absorption across intestinal epithelium. Most studies involve either measurements of acid-base or nutrient transporter activity using fluorescent dyes to monitor intracellular pH by microfluorimetry or electrophysiological recordings in Ussing chambers of native mucosa and cell lines derived from gene-targeted (“knockout”) mice. In addition to the cystic fibrosis mice, the laboratory maintains colonies of mice with gene-targeted deletion of other acid-base transporting proteins, including Na+/H+ exchangers, Cl-/HCO3- exchangers and Na+/K+/2Cl- cotransporters. Molecular studies in the laboratory involve the measurements of gene expression in the mice (quantitative real-time PCR, Northern blots and microarrays) and cloning of specific murine transporters for functional expression studies in heterologous cell systems. In addition to the above methods, other techniques employed in the laboratory include cell culture, retroviral and adenoviral gene transfer, pH stat/isotopic flux studies, laser capture microdissection and PCR based genotyping.

Appointments: Associate Professor, Department of Physical Medicine and Rehabilitation. Professor, Department of Biomedical Sciences
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)?
Appointments: Associate Department Head and Professor, Department of Medical Pharmacology and Physiology

Research
My general area of research is on the mechanisms of mechano-transduction by blood vessels: How does the smooth muscle layer of a blood vessel detect changes in pressure? What cellular proteins and signaling pathways are involved in this process? Currently, our focus is on a signaling axis linking the extracellular matrix with integrin receptors, the cytoskeleton, and ion channels. We focus on two ion channels: the L-type, voltage-gated calcium channel and the large-conductance, calcium-activated (BK) potassium channel that are acutely regulated by integrin signaling in vascular cells.

The experimental approaches used in my laboratory include isolated, perfused microvessel methods and single-cell electrophysiology. We combine these with a variety of imaging methods, including confocal, atomic force, and TIRF microscopy. We also use molecular analyses, such as site-directed mutagenesis and co-immunoprecipitation, to identify and test which integrin-associated proteins are involved in modulation of ionic channels. A related research interest is the role that collecting lymphatic vessels play in the regulation of extracellular fluid balance.

These vessels exhibit spontaneous, phasic contractile behavior that propels lymph centrally. Lymphatic function is dramatically different than that of arterioles and venules and they express isoforms of contractile proteins that suggest lymphatic smooth muscle is a hybrid between cardiac muscle and vascular smooth muscle. We investigate their mechanical properties using servo-controlled systems to study isolated lymphatics from rats and mice under isobaric, isometric and isotonic conditions.
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.
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.
Research Interests
- 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.
Research Interests

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, spherical 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.
Kevin D. Gillis

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

Education:
DSc, MSEE & BSEE Washington State 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 protein kinases. We are presently using multiple biophysical approaches to assay dynamic aspects of secretion from individual adrenal chromaffin cells. We have found that activation of protein kinase C (PKC) enhances depolarization-induced exocytosis many fold while actually decreasing the calcium current which triggers release. Using several different protocols, we have shown that PKC enhances secretion by increasing the size of the "readily releasable pool" of secretory granules. On the other hand, our experiments with caged Ca2+ show that PKC does not shift the Ca2+-sensitivity of the final step in secretion. Since protein kinases 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. In the future, we plan on further characterizing the kinetic steps modulated by protein kinases. For example, does PKC increase the size of the readily releasable pool by increasing the "filling" rate or does it stabilize vesicles in the "readily releasable" state? We also plan to examine the targets of kinase action at the molecular level.

Our research also has a strong engineering component with particular emphasis on developing or refining electrical and optical techniques for studying secretion. Techniques in use in the lab include patch-clamp electro-physiology with membrane capacitance measurements as an assay of involved with is investigating the effects of cardiovascular deconditioning which occurs after a period of bed rest or spaceflight. Deconditioning is associated with several changes in cardiovascular regulation including increased incidence of orthostatic hypotension, which is the inability to maintain blood pressure when a person stands up, and a reduced ability to maintain blood pressure during hemorrhagic events. Since females have higher incidence of problems following bed rest or spaceflight, we are evaluating gender differences in autonomic control of the circulation following cardiovascular deconditioning. Defining the central nervous system mechanisms that account for the changes in autonomic nervous system function during these states will increase our understanding and enhance our ability to treat problems associated with pregnancy and cardiovascular deconditioning, exocytosis/ endocytosis, amperometric detection of catecholamine secretion with carbon fiber electrodes, photometric measurement of membrane turnover and intracellular Ca2+ concentration with indicator dyes, and photo-release of intracellular Ca2+ from caged compounds.
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.
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.
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.
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 Taiwan 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.
Allan Jones  
Office Location: MA 415 Medical Sciences  
Office Phone: 573-882-8029  
E-mail: JonesA@missouri.edu  
Education:  
PhD University of Pennsylvania  

Research Description  
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.
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.
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 posts ischemic 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 posts ischemic 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.
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.
M. Harold Laughlin

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

Education: PhD University of Iowa, BA Simpson College

Appointments: Chair of the Department of Biomedical Sciences, 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 arteries and veins, and alterations in factors 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.
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.
Mark Milanick

Office Location: 360 Dalton Cardiovascular Research Center
Office Phone: 573-882-4741
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).


Approaches for resource poor areas

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.
Educational innovations

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

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 Exchanger
   - 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.
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.
Leona Rubin
Office: E102 VMED
Phone: 573-882-5903
E-mail: rubinl@missouri.edu

Education:
BA Temple University, MS Rutgers University,
PhD University of Colorado Health Science Center

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

Research
Dr. Rubin’s research is focused on understanding cellular pathways that regulate cardiovascular function during health and disease states. There are three major projects within the laboratory: 1.) How do immune mediators, released during inflammatory conditions (endotoxemia/sepsis, atherosclerosis) cause myocardial and/or vascular failure? Investigations focus on alterations in second messenger system(s) and cellular targets such as potassium and calcium channels. Therapeutic modalities also are probed as a means to identify affected pathways. 2.) What are the cellular pathways that mediate vascular hypoxic vasodilation? Matching of blood flow to meet tissue substrate needs is a fundamental property of the vasculature. However, the signals and vascular mechanisms responsible for dilation are unknown. We have targeted three sites for involvement in hypoxic vasodilation, AMP-activated kinase, Akt and voltage-dependent potassium channels. 3.) What is the role of sex hormones in modulating cardiovascular function? Specifically, do sex hormones alter expression of voltage-dependent potassium channels in either vascular smooth muscle or the myocardium? Myocardial studies examine both intrinsic (potassium currents of cardiac myocytes) and extrinsic (heart rate variability) control of heart rate. Methodologies include those needed to: 1.) measure contraction, Ca2+, and ionic currents of isolated ventricular myocytes; 2.) measure intracellular second messenger molecules, their substrates and products (gel electrophoresis ion chromatography, high performance liquid chromatography, gas chromatography and mass spectrometry), 3.) in vitro physiology of vascular function and 4.) in vivo assessment of heart rate variability. Our animal models include a swine model of sex hormone replacement (estrogen or testosterone), guinea pig and rat models of endotoxemia and genetically modified mouse models lacking components of signaling pathways that regulate cardiovascular function.
James C. Schadt
Office: 323 DCRC
Phone: 573-882-7366
E-mail: schadtj@missouri.edu

Education:
PhD Texas Tech University, MS Indiana State University,
MS & BS Northern Illinois University

Appointment: Associate Professor, Department of Biomedical Sciences

Research
Traumatic injury is the leading cause of death for individuals under 40 years of age. While head trauma is
the most common cause of death, blood loss is number two, and the effects of head injury are exacerbated
by blood loss.

The goal of my research is to learn more about how the brain and endocrine systems control the heart and
blood vessels during traumatic blood loss.
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 always 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.
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 hemeoxygenase2 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.
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.

Ronald Terjung

Office: E101VMED
Phone: 882-2635
E-mail: terjungr@missouri.edu

Education:
PhD University of Iowa, MA San Jose State College, BS Wheaton College

Appointment: Associate Dean of Research, Department of Biomedical Sciences, Professor, Department of Biomedical Sciences, Adjunct Professor, Department of Medical Pharmacology and Physiology

Research
Adenine nucleotide (And = ATP + ADP + AMP) metabolism occupies a pivotal role in cell regulation, particularly for skeletal muscle where contractile activity increases ATP hydrolysis rate manyfold. The control of [ATP] in contracting muscle depends upon: 1.) the control of energy supply pathways; 2.) And degradation reactions; 3.) And synthesis reactions from precursors; and 4.) contractile activity which determines the rate of ATP hydrolysis. He is evaluating differences in And metabolism among skeletal muscle fiber types, critical responses to exercise, and adaptations induced by chronic exercise. Enhanced physical activity represents an important treatment for persons with peripheral arterial insufficiency and leads to meaningful adaptations that
increase exercise tolerance. These adaptations include neovascular development to improve a) blood/tissue exchange properties within muscle (enhanced capillarity) and b) flow capacity to active muscle (collateral vessel expansion). The exercise-induced increase in collateral blood flow likely involves the angiogenic growth factors (e.g. bFGF, VEGF). These potent cytokines stimulate neovascularization in experimental ischemia in vivo. His working hypothesis is that neovascularization occurs in response to tissue "need" established by flow deficits (ischemia) and/or by increased demands for vascular support (exercise). His research is evaluating: 1.) the interactions between ischemia, exercise and exogenously infused recombinant angiogenic growth factors; 2.) the functional significance of the vascular adaptations; and 3.) the tissue events related to neovascularization.

Xiaoqin Zou
Office: 222D DCRC
Phone: 573-882-6045
E-mail: xiaojinz@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.
APPENDICES

PUBLICATIONS

PRESENTATIONS

SEMINARS

ACTIVE GRANTS & CONTRACTS

PROFESSIONAL SERVICE ACTIVITIES
Christopher Baines

Publications:

Peer-Reviewed:

McGee AM, Baines CP. Phosphate is not an absolute requirement for the inhibitory effects of cyclosporin A or cyclophilin D deletion on mitochondrial permeability transition. Biochem J. 2012; 443:185-91.


Book Chapters:

Abstracts:

Gutierrez-Aguilar M, Baines CP. Mapping the Role of the Mitochondrial Phosphate Carrier in Cardiomyocyte Life and Death. Presented at the AHA Scientific Sessions meeting, November 2012.


Presentations


“RIPing Holes in Mitochondria: trying to decipher programmed necrosis.” Cardiovascular Center, University of Iowa, Iowa City, IA. 10/12/12.
Baines continued:

“Regulation of the Mitochondrial Permeability Transition Pore.” International Society for Heart Research, North American Section Annual Meeting, Banff, Canada. 5/28/12.

“Complement 1q Binding Protein, Or Why a Cardiac Guy is Looking at Cancer.” Center for Translational Medicine and Department of Pathology and Cell Biology, Thomas Jefferson University, Philadelphia, PA. 4/12/12.

**Active Grants:**

Current:
- R01HL094404 (PI: Baines) 12/2008-11/2013
  NIH/NHLBI $1,250,000 direct costs
  “Molecular Identity of The Cardiac Mitochondrial Pore” Role: PI

  AHA Midwest Affiliate $52,000
  “Investigating Hexokinase-2-mediated cardioprotection” Role: Sponsor

- MU-iCATS Pilot Grant (PI: Emter) 1/2012-12/2012
  University of Missouri $50,000 direct costs
  “The Effects of Cyclophilin Inhibition on Cardiomyocyte Cell Death and Ventricular Remodeling in Heart Failure” Role: Co-PI

- Prostate Cancer Development Award (PI: Baines) 9/2012-9/2013
  US Department of Defense $75,000 direct costs
  “Inhibiting Mitophagy as a Novel Mechanism to Kill Prostate Cancer Cells” Role: PI

- Research Council Award (PI: Baines) 4/2012-3/2013
  University of Missouri $7,500 direct costs
  “Identifying C1qbp binding drugs as potential anti-cancer agents” Role: PI

- Sponsored Research Agreement (PI: Baines) 7/2012-6/2013
  Stealth Peptides, Inc. $17,765
  “Mechanisms of Cytoprotection by SPI-20 and MTP-131 peptides” Role: PI
Baines continued

**Professional Service**

Department/Center:

Chair, Dalton Cardiovascular Research Center Safety Committee

Director, Biomedical Sciences Seminar Series

Member, Biomedical Sciences Graduate Program Admissions Committee

Member, Biomedical Sciences Assistant/Associate Professor Search Committee

College:

Member, Research Committee, College of Veterinary Medicine

Member, VOLUM Committee, College of Veterinary Medicine

University:

Panel member, Summer Faculty Grant Writing Institute

National/International:

Editorial Boards: American Journal of Physiology, Heart and Circulatory Physiology, Bioenergetics
Edward Blaine

Professional Service

Committee on Committees, 2009-2010, Chair 2010-present
Faculty Grievance Oversight Committee, 2009-present
Honorary Degree Committee, University of Missouri, 2008-present
Graduate Education Committee, Dept. Med. Pharm./Phys, 2004-present
Campus Planning Committee, University of Missouri, 2005-present
Health Outcomes in Athletes, 2009-

Problem-based Learning Curriculum
  Continuous teaching contributions since 1992

2011-2012 Tutor Blocks 2,3,4,5,7, M4 mentor
2012-2013 Tutor Blocks 1,2,3,5,8, M4 mentor

  Microcirculation (Graduate Course)
  Renal Physiology (Graduate Course)
  Neurohumoral Control of the Circulation (Graduate Course)
  Salt and Water Homeostasis (Undergraduate Course)
  Herpetology, Physiological Ecology (Undergraduate Course)
Silvia Bompadre

Active Grants

Fluorescence microscopy studies of CFTR channels $23,000 (9/1/2012 – 8/31/2013) PI: Silvia G. Bompadre
MU Research Board Grant

Single-molecule studies of CFTR channels $14,500 PI: Silvia G. Bompadre MU Summer Research Fellowship

Professional Service

Reviewer

University of Missouri Research Board
Research Grants Council (Hong Kong)
Frontiers in Pharmacology
Computational Biology

Member of the Arts & Sciences Diversity Committee (2012-present).


**Active Grant**

**Douglas Bowles**

**Publications**


**Presentations**


Invited Symposium for AALAS Conference on “Swine models of atherosclerosis”, Minneapolis, Minnesota, November, 2012

**Active Grants**

CVM/COR Faculty Research Grant (Bowles, PI) 1/1/12-12/31/12  *2% NIH/NHLBI: SBIR $17,953 ADC Role of KCa3.1 in driving coronary smooth muscle phenotype Major goals: Perform loss-of-function/gain-of-function experiments of KCa3.1 in porcine coronary arteries.

R44 HL097485-01 (Chen, PI; Bowles, Co-I) 9/1/11-7/31/13 10% NIH/NHLBI: SBIR $237,000 ADC Improved long-term biocompatibility of coronary stents by plasma coating process Major goals: Develop a superior plasma coating for stents

P01 HL52490 (Laughlin, PI; Bowles, Project Leader) 1/1/06-12/31/12 (NCE) 25% NIH/NHLBI $187,472 ADC (Project 1) Ion channel regulation of coronary smooth muscle phenotype.
Bowles continued

Major goals: Determine the role of ion channels in coronary smooth muscle regulation of tone following exercise training. Treadmill endurance exercise and dietary high fat interactions are studied in miniature swine P01 HL52490 (Laughlin, PI; Bowles, Core Leader) 1/1/06-12/31/112 (NCE) 5\% NIH/NHLBI  $167,681 ADC (Core C)Vascular Biochemistry and Molecular BiologyMajor goals: Provide investigators with access to routine as well as state-of-the-art molecular and biochemical tools, expertise, and techniques.

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

MU Life Sciences Trust Fund Research Grant (Fay, PI; Bowles, Co-PI) 1/1/09-11/30/12 (NCE) 10\% Missouri Life Sciences Research Board $815,625 ADC Targeting Plasminogen Activator Inhibitor-1 to Inhibit Restenosis

Professional Service

Appointments
1998-present Graduate Faculty, University of Missouri-Columbia
1998-present Doctoral Faculty, University of Missouri-Columbia
1998-present Biomedical Sciences Faculty, College of Veterinary Medicine, UMC
2006-present Medical Pharmacology and Physiology Faculty, College of Medicine, UMC

*Course Director, Veterinary Cell Biology 5506/7333
Faculty evaluator, Veterinary Physiology 8420
Course Director, Multidisciplinary Approaches to Biomedical Sciences 8085

Graduate Committee Member
Kurt Marshall, 2011-pres. Department of Biomedical Sciences, University of Missouri
Ashlee Williams, 2011-2012, Department of Biomedical Sciences, University of Missouri
Zeev Swartz, DVM, 2011-pres. Departments of Surgery, Biomedical Sciences, University of Missouri
Joe Company, 2008-pres. Department of Biomedical Sciences, University of Missouri
Kyle McCommis, 2008-pres. Department of Biomedical Sciences, University of Missouri
Luise King, 2009-pres. Department of Biomedical Sciences, University of Missouri

2012-pres. Chair, Biomedical Sciences Doctoral Faculty Nomination Committee
2010-pres. Biomedical Sciences Faculty Mentoring Committee (Dr. Emter)
2012-2015 CVM Promotion and Tenure Committee
2012 Chair, Associate Dean for Academic Affairs Search Committee
2011-2012 Chair, CVM Faculty Policy Committee
2009-2013 CVM Faculty Policy Committee
2010-2012 Chair, CVM Administrator Evaluation Committee
2005-present CVM Graduate/Resident Training Committee
2003-present Research Mentor, Comparative Medicine Training Program
2012 Member, Professional Science Masters Task Force
2012-2015 Member, Graduate Faculty Senate
Bowles continued

2012-2013 Graduate Faculty Senate Awards Committee
2012-pres. Member, Life Sciences Fellowship Awards committee
2006-pres. Trustee, Health Activity Center M. Harold Laughlin Scholarship Fund

Manuscript Review
2009-pres. Reviewer, Hypertension
2009-pres. Reviewer, Atherosclerosis, Thrombosis and Vascular Biology
2007-pres. Reviewer, Circulation
2000-pres. Reviewer, American Journal of Physiology: Cell
1995-pres. Reviewer, Circulation Research
1998-pres. Reviewer, Medicine & Science in Sports & Exercise
2011-14 Nominating Committee, CV Section, American Physiological Society
2012 Sponsor for Visiting Professor, Dr. David Fisher, University of Western Cape, Capetown, South Africa during October/November. Purpose of Dr. Fisher’s visit was to continue collaborative project on cellular mechanisms of herbal-based medical therapies.

Grant Review Board

2012 Dunhill Medical Trust Review (London)

Editorial boards

2012-pres. Editorial Board of Scientifica

Manuscript Review

2001-pres. Reviewer, Cardiovascular Research
Publications

Professional Journals


Published Abstracts


Presentations

*Cftr and the Biology of Regenerating Crypts in Intestinal Organoids. St. Jude Children's Research Hospital, Memphis, TN – April 5, 2012


*Epithelial-Specific Pathology Revealed in Intestinal Organoids from the Cystic Fibrosis Mouse, West Virginia University Health Sciences Center, Morgantown, WV – October 18, 2012

Poster presentations


Active Grants

01/12-11/12 MU Research Board, Modulation of Cftr to minimize crypt damage during chemotherapy”, $50,000, PI

01/12-12/12 Mizzou Advantage, “Regenerating Intestinal Crypts for Biomedical Research”, $50,000, PI.

04/11 - 3/13 Cystic Fibrosis Foundation; “Abnormal Regulation of Goblet Cells in the Cystic Fibrosis Intestine”, $194,400, PI.
Clarke continued

07/11-06/12 National Institutes of Health; “CFTR and Acid-Base Transporters in Regenerating Intestinal Crypts”, $74,946, NIH R56, PI.

05/12-04/16 National Institutes of Health; “CFTR and Acid-Base Transporters in Regenerating Intestinal Crypts”, $1,619,473, R01 DK 48816–14–17, PI.

Professional Service

National/International

-Cystic Fibrosis Foundation, Research and Training Committee (Grant Reviews), Charter Member

-CIMG NIH Study Section, Charter Member

-South Carolina INBRE Bioinformatics Pilot Projects


College

NAVMEC/Education Committee, Member

Department

Graduate Policy Advisory Committee, Biomedical Sciences - Member
Publications


Invited Lectures/ Presentations

2012 (October) Developmental Vascular Biology Workshop V- Navbo Conference, Monterey, CA, Invited Speaker

2012 (August) 5th Mayo Clinic Angiogenesis Conference, Minneapolis, MN, Invited Speaker

2012 (July) Signal Transduction by Engineered Extracellular Matrices-Gordon Conference, Biddeford, ME, Invited Speaker
George Davis continued

2012 (June) International Vascular Biology Meeting 2012, Wiesbaden, Germany, Invited Speaker
2012 (April) UCLA Vascular Biology Seminar Series, Los Angeles, CA
2012 (March) Program in Genomics of Differentiation Seminar Series, National Institutes of Health, Bethesda, MD
2012 (March) Harvard Medical School Vascular Biology Seminar Series, Boston, MA

Active Grants


NIH-NHLBI- R01 G.E. Davis- PI, “Hematopoietic stem cell cytokine control of developmental vascularization” 1/01/11- 12/31/14. $250,000/ yr.

NIH- NHLBI- R01 G.E. Davis- PI, M.J. Davis Coll. Investigator, “Pericyte proteinase inhibitors and EC tube stabilization” 1/12/10- 11/30/13, $250,000/ yr.

NIH- NHLBI- R01 G.E. Davis- PI, M.J. Davis Coll. Investigator, D.C. Zawieja- Coll. Investigator, “Molecular control of EC lumen formation by MT1-MMP” 1/01/08- 12/31/12, $250,000/ 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
American Journal of Pathology Journal of Cell Science
Trends in Cardiovascular Medicine
Atherosclerosis, Thrombosis and Vascular Biology
Journal of Vascular Biology
FASEB Journal
Molecular and Cellular Biology
Cancer Research
BBA-Cancer
Arthritis and Rheumatism
Current Biology
Blood
Microcirculation
Proc. Natl. Acad. Sci. USA
Development
Developmental Dynamics
Molecular Biology of the Cell
Michael J. Davis

Publications


Manuscripts in Press


Scallan JP, Wolpers HJ, Davis MJ: Rapid lymphatic constriction in response to elevated output pressure is conducted across inter-lymphangion valves (J Physiology, Dec. 10, 2012) (Highlighted article)

Book Chapters


Li Qin Zhang, Dilyara Cheranova, Margaret Gibson, Shinghua Ding, Daniel P. Heruth, Deyu Fang, and Shui Qing Ye. RNA-seq reveals novel transcriptome of genes and their isoforms in human pulmonary microvascular endothelial cells treated with thrombin. PloS ONE 7:e31229, 2012.


Knockout of type 2 IP3 receptor (IP3R2) enhances cell proliferation in the subgranular zone (SGZ) in dentate gyrus of hippocampus. 42th Annual Meeting of Society for Neuroscience, October 13-17, 2012, New Orleans. Wenting Chang, Hailong Li, Nannan Zhang, Shinghua Ding.


Xiaozhen Wang, Hailong Li, Jinglu Tan, Shinghua Ding Knockout of type 2 IP3 receptor (IP3R2) enhances neuronal proliferation. Life Science Week, University of Missouri-Columbia. April 16-20, 2012.

Wenting Chang, Hailong Li, Nannan Zhang The effect of inhibition of group 1 mGluRs on brain damage after ischemia. Life Science Week, University of Missouri-Columbia. April 16-20, 2012. Hailong Li, Nannan Zhang, Shinghua Ding.


Knockout of type 2 IP3 receptor (IP3R2) enhances neuronal proliferation. Cardiovascular Day, University of Missouri-Columbia. February 21, 2012. Wenting Chang, Hailong Li, Nannan Zhang

“PBEF plays a critical role in brain protection after ischemia”. Cardiovascular Day, University of Missouri-Columbia, February 21, 2012. Shinghua Ding (Invited talk)
Ding continued

Brain protection by PBEF in cerebral ischemia. Children's Mercy Hospitals and Clinics, School of Medicine, University of Missouri-Kansas City, January 6, 2012.

**Active Grants**

Astrocyte-mediated neuronal excitation (0735133N)* AHA (National SDG grant) $260,000 7/1/2007- 6/30/2012 PI 24% effort Non-cost extension for the year 7/01/2011- 6/30/2012

Role of Gliotransmission in ischemia (R01NS069726) NIH $1,591,689 5/15/2010- 4/30/2015 PI 40% effort

**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, Organizing Committee; Cardiovascular Day 2012; University of Missouri-Columbia. February 21, 2012.

Judge, Undergraduate Research & Creative Achievements Forum; University of Missouri. April 24, 2012

Member of Membership Committee, American Society for Neurochemistry. March 2011-March 2015.

Reviewers for multiple Journals

Session chair on the Colloquium titled “Glial dysfunction and role in brain ischemia” at 43rd Annual American Society for Neurochemistry (ASN) meeting in Baltimore, March 3-7, 2012.
Paul J. Fadel

Publications


Presentations

Impaired Nitric Oxide Signaling in the Brain, Not the Periphery, Contributes to Increased Cardiovascular Risk. APS Conference, Omaha, Nebraska (7/12).

Influence of Sex and Ovarian Hormones on Arterial Baroreflex Control during Exercise in Humans. Brazil

Control of the Sympathetic Nervous System in Humans: A Role for Insulin. Department of Biomedical Sciences-Veterinary Medicine, University of Missouri, Columbia (2/12).

Active Grants

“Aging, Sex, and Neural Cardiovascular Control during Dynamic Exercise,” National Institutes of Health, R01 HL093167, 30% effort, PI: Paul Fadel, 8/08-8/13, $1,250,000.

“Exercise and Health: Integration from molecule to patient,” National Institutes of Health, T32-AR048523, PI: Ronald Terjung, Mentor: Paul Fadel, 7/09-6/14, $1,000,000.


“Age- and sex- related alterations in arterial baroreflex control of blood pressure during dynamic exercise,” American College of Sports Medicine Foundation Research Grant, PI: Areum Kim (Graduate Student), Advisor: Paul Fadel, 7/10-3/12, $5,000.
Professional Services

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)

Editorial Board- Experimental Physiology (11/12-present)

Journal of Applied 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

University of Missouri, School of Medicine Scientific Peer Review Committee (1/08-present)

Medical Pharmacology and Physiology Seminar Committee (9/08-present)
William Fay

Publications


Presentations

Dept. of Cellular and Molecular Physiology, Univ. of Nevada Reno (Sept. 2012); Topic: Regulation of ion channels by integrin-ECM signaling (Ion Channel Regulation course

March 2012  Cardiovascular Day, University of Missouri, Columbia, MO
May 2012  Dept. of Toxicology & Pharmacology, University of Mississippi, Jackson, MS
October 2012  Dept. of Physiology & Cell Biology, University of Nevada, Reno
November 2012  Dept. of Anatomy & Physiology, Kansas State University, Lawrence, KS
2012  Co-chair, MCS-BMS meeting, Oxford, UK

Active Grants

NIH R01
“Roles of Plasminogen Activator Inhibitor-1 and Vitronectin in Failure of Coronary Revascularization”
PI: W. Fay (25% effort)
9/1/10-8/31/14. $250,000 annual direct costs.
Impact/Priority Score: 12; %ile score: 1.0

NIH/NHLBI Program Project Grant
“Molecular Genetics of Coagulation Disorders” PI: D. Ginsburg
Dr. Fay is Co-Investigator (10% effort) on Project 3: (“Thrombosis and Hemostasis in Host Defense from Bacterial Infection”, PI: H. Sun)
4/1/09-3/31/14 ($223,863 annual direct costs of Project 3)

NIH R44
“Improved long-term biocompatibility of coronary stents by plasma coating process”
PI: M. Chen. Dr. Fay is Co-Investigator (10% effort)
NIHR44
“Prevention of Vein Graft Intimal Hyperplasia with Human Apyrase”
PI: R. Chen. Dr. Fay is Co-Investigator (10% effort) 0/1/12-9/30/13

Professional Services

Specialty Board Review


2010- School of Medicine Advisory Committee on Research Space
2011- Blue Ribbon Commission, School of Medicine
2012-2013 Research Portfolio Taskforce Committee

Vice-Chair of Department, 2006-present
Administrative Advisory Committee, 2005-present
   Space Committee, 2005-present
   RIF Committee, 2005-present

2012 FASEB Summer Research Conference: Smooth Muscle, Snowmass, CO-Session Chair

Journal Review

American Journal of Physiology: Advances in Physiology Education
American Journal of Physiology: Cell Physiology
American Journal of Physiology: Heart and Circulatory Physiology
American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology
Circulation Research
Journal of Applied Physiology
Journal of Vascular Research
Microcirculation
PLoS ONE
Shubra Gangopadhyay

Publications


An, Woo-Jin; Wang, Wei Ning; Ramalingam, Balavinayagam; Mukherjee, Somik; Daubayev, Batyrbek; Gangopadhyay, Shubhra; Biswas, Pratim, "Enhanced Water Photolysis with Pt Metal Nanoparticles on Single Crystal TiO2 Surfaces”, Langmuir, in press (2012).


Gangopadhyay continued


Active Grants

NSF ECCS-123217, $ 360,000: ”Trace Vapor Detection of Explosives using Molecularly Imprinted Organic Field Effect Transistors and Metal Nanoparticles”, Award Period: 9/1/2012-5/31/2015. Role: PI.

THE BOEING COMPANY $70,000: ”Window repair”, Award Period: 5/1/2012-7/31/2012.


NANOTECHNOLOGY ENTERPRISES INC/US Army $299,712: Large scale production of advanced nanoenergetic materials as primer for propellant ignition 08/01/2011-07/30/2012 Role: Co-I

NEMS/MEMS Works, LLC./NANOTECHNOLOGY ENTERPRISES INC/US Army $150,000: Prototype development of remote memory disruption systems for portable electronics 08/01/2011-07/30/2012 Role: PI


CROSSLINK , LLC., NTEC/US Army $100,000: Solid State High Power and Energy Density Supercapacitors Using Nanostructured Conductive Polymer Composites 08/01/11-07/30/2012 Role: PI


NEMS/MEMS Works, LLC/US Army $85K: MEMS Based Systems Development for Contaminant Analysis Role: PI. Award Period: 8/09-8/12. The objective of this research is experimentation with PDMS fiber and nanoporous particulate films for efficient contaminant analysis with specificity.
Gangopadhyay continued

NSF ECCS-0901566 GOALI $372K: Nanothermite Based Micro Shockwave Generators and Nanoparticles for Targeted and Efficient Gene/Drug Delivery Role: PI. The objective of this research is to develop a novel digitally controlled micro shockwave generator by integrating nanothermites with MEMS. Award Period: 8/09-7/13.

NSF ECCS-0801753 $324K: Collaborative Research: High Density Metal and Semiconductor Nanoparticles for Memory and Photonic Applications. Role: PI. The major goal of this project is to prepare high density metal and semiconductor nano-particles for fabricating memory and photonic devices. Award Period: 6/08-NEMS/MEMS Works, LLC US Army $50K: Development of a Micro-Device for Remote Disruption of Electronic Hardware. Role: PI Award Period: 11/10-09/11


Office of Naval Research N00014-11-C0392 $1.6M: University of Missouri Railgun Program. Role: Co-I. Award Period: 03/11-03/13

Professional Service

American Physical Society

Institute of Electronics and Electrical Engineers

Material Research Society

American Chemical 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
Publications


Research Funding (all dollar amounts indicate direct costs)
Present
NIH, SBIR (to ExoCytronics LLC), $671,743 (subcontract to MU: $212,101), “Development of a prototype system for assaying exocytosis from individual cells”, PI, 09/11 – 05/13.

NIH, R01, $543,546 (MU portion), "A scalable n×n 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, R01, $1,016,600, “Programmable multi-target detection using an aptamer-integrated nanopore”, co-I (PI: L.-Q. Gu), 02/07 – 01/12

Completed
University of Missouri Research Board, $40,000, “Combined optical and electrical measurement of exocytosis from single vesicles”, PI, 9/10 – 2/12.

Professional Service


Current graduate students:
Jia Yao, PhD student in BE, 08/06 – present
Jaya Ghosh, PhD student in BE, 08/07 – present
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


Presentations


(Invited speech) Gu, L.Q. Nanopore in personalized medicine –from Sequencing to Epigenetics Biophotonics Workshop, University of Missouri, 2012

Gu continued

**Active Grants**

Programmable multi-target detection using aptamerintegrated nanopore NIH $1,270,750 02/01/2007 –1/31/2012 (non-cost extension) PI

Coulter Foundation Bridging Fund at MU Coulter Foundation $111,800 (+$25,000 for commercialization strategy) 07/01/2012-06/30/2013 PI
Eileen Hasser

Publications


Presentations

Activation of nucleus tractus solitarii (nTS) neurons that project to the rostral ventrolateral medulla (RVLM) or hypothalamic paraventricular nucleus (PVN): Role of acute hypoxia (AH). Ruyle BC, Heesch CM, King TL, Kline DD, Hasser EM. FASEB J., 2012

Caudal ventrolateral medulla (CVLM) activation by acute hypoxia (AH) is independent of changes in arterial blood pressure (ABP). King, TL, Heesch CM, Friskey SA, Ruyle BC, Kline DD, Hasser EM

Cardiovascular deconditioning augments baseline breathing as well as peripheral and central chemoreflex responses. Zhou Z, King TL, Kline DD, Heesch CM, Hasser EM

Hydrogen peroxide (H2O2) modulates membrane properties in second-order nucleus tractus solitarii (nTS) neurons. Ostrowski TD, Hasser EM, Heesch CM, Kline DD

Expression of ROS catabolic enzymes in the medial nucleus tractus solitarii (nTS) of rats and upregulation during acute hypoxia. Ostrowski TD, Barr SL, Dantzler HS, Hasser EM, Kline DD, Heesch CM

Active Grants

RO1 HL55306 04/01/07-03/31/11 No-cost extension Cardiovascular Regulation in Hindlimb Unweighted Rats National Institutes of Health Annual: $225,000/yr. Total: $900,000 Role: PI

RO1 HL098602 07/15/2010-06/30/2014 Plasticity of nTS output neurons in acute and chronic hypoxia National Institutes of Health Role: (Multi PI with Cheryl M. Heesch and David D. Kline) Annual: $491,679 Total Direct/yr ($163,283.00-EMH Direct): Total: 2,830,531

RO1 HL091164 (Cheryl M. Heesch, PI) Central nervous system plasticity in sympathoinhibition in pregnancy National Institutes of Health Role: Co-I Annual: $250,000 Direct costs
Hasser continued

**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
- National

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

King, T.Luise; Heesch, Cheryl M.; Friskey, Sarah; Ruyle, Brian; Kline, David D.; Hasser, Eileen M. Caudal ventrolateral medulla (CVLM) activation by acute hypoxia (AH) is independent of changes in arterial blood pressure (ABP). FASEB J. 26: 702, 2012.


Zhou, Zijin; King, T. Luise; Kline, David D.; Heesch, Cheryl M.; Hasser, Eileen M. Cardiovascular deconditioning augments baseline breathing as well as peripheral and central chemoreflex responses. FASEB J. 26: 702, 2012.

Ruyle, Brian C.; Heesch, Cheryl M.; King, T. Luise; Kline, David D.; Hasser, Eileen M. Activation of nucleus tractus solitarii (nTS) neurons that project to the rostral ventrolateral medulla (RVLM) or hypothalamic paraventricular nucleus (PVN): Role of acute hypoxia (AH). FASEB J. 26: 702, 2012.


Tim D. Ostrowski, Stacy L. Barr, Heather A. Dantzler, Eileen M. Hasser, David D. Kline, Cheryl M. Heesch. Expression of ROS catabolic enzymes in the medial nucleus tractus solitarii (nTS) of rats and upregulation during acute hypoxia. Presented American Physiological Society Meeting, Omaha, Nebraska, Fall 2012


Active Grants

“Central nervous system plasticity in 06/01/09- 03/31/13
Sympathoinhibition on pregnancy” ~$250,000
National Institutes of Health (R01 HL091164) (annual direct)
(Role: Principal Investigator, 20% effort)
Heesch continued

“Plasticity of nTS output neurons in acute and chronic hypoxia” 07/01/10-06/30/15 $491,679 National Institutes of Health (R01 HL098602) Multi-Investigator Role: PD/PI = E.M. Hasser, C.M. Heesch, D.D. Kline [CMH direct $163,512] (1.8 Calendar months/ each)

“Cardiovascular regulation in hindlimb unweighted Rats” 04/01/07-03/31/13 $225,000 (no cost extension) NIH-R01-HL53306 (E. Hasser, P.I.) (Role: Co-Investigator, 10% effort)

“Adaptation of brainstem circuits to chronic hypoxia” NIH R01 HL085108-01 04/15/08-04/14/13 $250,000 (annual direct) (D.D. Kline, P.I.) (Role: Co-Investigator, 1 person/month effort)

Presentations (*) invited presentations

4/2012: Experimental Biology 2012; San Diego, CA; April 2012 (see abstracts above)

* 8/2012: Tim D. Ostrowski, Stacy L. Barr, Heather A. Dantzler, Eileen M. Hasser, David D. Kline, Cheryl M. Heesch. Expression of ROS catabolic enzymes in the medial nucleus tractus solitarii (nTS) of rats and upregulation during acute hypoxia. Presented American Physiological Society Conference, Omaha, Nebraska, Fall 2012

Professional Service

Departmental:
02/06- present: Junior Faculty Mentoring Committees, Dept. Biomed Sci.
Ileana Constantinescu (Committee Chair)
Kathy Kuel-Kovarik (Committee Member)

2012 Biomedical Science: Conflict of Interest Committee

College:
01/09 – 9/30/12 Committee on Faculty Responsibility
12/10 – present CVM COR Grant Review Committee
8/11 – 7/14 CVM Faculty Policy Committee (Ex Officio)
Heesch continued

University:
09/03-present  Executive Committee, Interdisciplinary Neuroscience Program
8/11 – 7/14 College of Vet Med Representative to Faculty Council
8/11 – 7/14  Faculty Affairs Committee, Univ. of Missouri Faculty Council


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

Grant Application Reviews: 3/ 2012 External Consultant, “Sex and Gender in Women's Cardiovascular Disease,” University of California, Davis.

Committee/mentorship listing:
8/2011 – present (Kevin) Max Coldren (Ph.D., Adviser, Dr. Cheryl Heesch)
2008 -- present Luise King (Ph.D., Adviser, Dr. Eileen Hasser)

Dept. of Medical Pharmacology & Physiology, School of Medicine, Univ. of Missouri
2008 –2012 Areum Kim (Ph.D., Adviser, Dr. Paul Fadel)
2012 – present Shenghua Yuan (Ph.D., Adviser, Dr. Steven Segal)

Dept. of Physics, University of Missouri
9/11 – present Chris Owens (Ph.D., Adviser, Dr. Gabor Forgacs)

Interdisciplinary Neuroscience Program
7/2010 – present Randall Brown (M.S., Adviser, Dr. Cheryl Heesch)

Honors College, Senior Honors Research Project
1/2012 – present Sean McCalmon (Adviser, Cheryl Heesch)
8/2012 – present Joshua Abernathy (Adviser, Cheryl Heesch)

Veterinary Research Summer Program (VRSP)
4/2012 – 8/2012 Lance Serbousek (Adviser, Cheryl Heesch)

Teaching Activity:

Wtr 2012 VBSCI 9467 (MPP 9437): Neural Control of the Circulation, 3 credit hr. course (14 contact hrs)
Wtr 2012 VBSCI 400: Multipisciplinary Problems Course 1 credit hr. course (2 lec/contact hrs.)
Wtr 2012 VBSCI 5508: Veterinary Pharmacology, Course Director, 2 credit hr. course (32 contact hrs)

Fall 2012 VBmS 5507: Veterinary Pharmacology (Diuretics, 2 lec/contact hrs)
Fall 2012 Vet Neuroscience 5100: Autonomic Nervous System (1 lec/contact hr)
Publications


Presentations


Hill, M.A. Mechanotransduction in arterioles. Australian Vascular Biology Society, Sanctuary Cove, Qld, Australia (September 2012).

Active Grants

“Signaling Mechanisms Underlying Myogenic Tone in Arterioles of Skeletal Muscle: Role of BKCa”
Total Award: $1,000,000 (Direct Costs) Period: 7/2009 – 6/2013. This project focuses on the role of the large conductance K+ channel (BKCa) in regulation myogenic tone in skeletal muscle arterioles. Specific emphasis is placed on heterogeneity in BKCa regulation in differing vascular beds.
Principal Investigator: Michael A. Hill, Ph.D., Effort 27.5%

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.

Professional Service

External Joint Appointments (Current):

2011 – present Visiting Professor
Luzhou Medical College
Luzhou, China

2006 – present Lecturer/discussion leader in Microcirculation graduate course (topics relating to endothelial cell/smooth muscle interactions)

2008 – present Problem Based Learning Facilitator (University of Missouri) for medical student education (Years 1 and 2). Block content related to metabolism, endocrinology, cell biology and cardiovascular.

2009 – present Advanced Imaging Techniques, Skills in Biomedical Research graduate course

Graduate Students/Residents Supervised

Srijita Dhar 2010 – present (University of Missouri)
Tanyi Qui 2012 – present (University of Missouri)
Kwangseok Hong 2012 – present (University of Missouri)
Postdoctoral/Research Fellow Trainees Supervised

Yan Yang (M.D., Luzhou Medical College, China)  2008 - present
Zahra Nourian (Ph.D., Aarhus University, Denmark)  2010 – present
Mozow Zuidema (M.D., Ph.D., University of Missouri)  2011 – present
Sewon Lee (Ph.D., University of Missouri)  2011 - present
Melissa Collins (Ph.D., Texas A&M University)  2012 - present

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)
•  Member of the organizing committee for the combined British and US Microcirculatory Societies Meeting, Oxford, July 2012.
•  Planning group for combined Microcirculatory Society/NAVBO Meeting (October 2013).

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

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.
2012 – 2013  President, Microcirculatory Society, USA (effective 5/2012).
2011 – 2012  President-elect, Microcirculatory Society, USA.
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.
2009 – 2012  Nominating Committee, American Physiological Society (CV Section), USA.
Chairperson, 2010 - present
Virginia Huxley

Publications


Presentations

2012

2013
Department of Pharmacology and Toxicology Seminar Series, Medical College of Georgia,

Active Grants

Current:
NIH R21 HL093068-01A2 “Sexual Dimorphism of Skeletal Muscle Microvascular Function”; Score 22; 10th percentile July 1, 2010 - June 30, 2012; $275,000 total direct No-cost extension through June 30, 2013

NIH R01 HL103151-01
“Sexual dimorphism in microvascular and lymphatic function: a role for insulin”; Score 29; 17th percentile 04-01-2010 through 03-31-2015; $1,250,000 total direct NIH R01 HL103425-01

“Sexual Dimorphism of Collecting Lymphatic Permeability” Score 28; 17th percentile 07-01-2010 through 06-30-2015 $1,250,000 total direct NIH R01 HL105328-01

“Integration of vascular network structure and function by quantitative microscopy analysis”; Multi PI; Palaniappan, Kannappan and Virginia H. Huxley Score 39 06/01/2010-05/31/2015; $3,199,806.46 total direct
Huxley continued

**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
2008 - MPP Journal Club
2008 - MPP Graduate student Skills
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
2008-12 Member NIH Hypertension & Microcirculation (HM) Study Section Medical Students (since inception of the PBL curriculum):
Respiration (3 lectures) (2011- )
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 -
NIH Hypertension and Microcirculation (HM) Study Section 2008-2012

**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


Active Grants

2008 – 2013  NIHR01, NIDDK, “Molecular pathophysiology of cystic fibrosis”, $1,014,155

2011 – 2012  Cystic Fibrosis Foundation, $75,000

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
**Publications**

Li, R., Ren, M., Chen, Ni., Hyder, S. M. And Wu, J. (2012) Depletion of platelets in B16/F10 melanoma tumor bearing mice prevents maturation of newly formed tumor blood vessels. 19th Annual Cardiovascular Day, University of Missouri, MO. (Feb 2012)


**Presentations:** Indicate invited presentations with an asterisk (*)

*Invited Presentation UMKC, Department of Pharmacology and Toxicology: VEGF: A therapeutic molecular target for treatment and prevention of hormone-dependent breast cancer

*Invited presentation at the 17th World Congress on Advances in Oncology. Crete, Greece. Title: Apigenin: a plant derived nutraceutical with preventive and therapeutc potential against progestin-dependent breast cancer.


Awards & honors

Editorial Board: Histology & Histopathology (2002-present)
International Journal of Oncology (elected to editorial board, 2005)
Honorary Visiting Professorship, Luzhou Medical College, China (2011-2016)

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
Chair, 2013 Cardiovascular Research Day Program Committee
Judge, Health Sciences Research Day, MU School of Medicine (2012)

National/International Service

Grant Review
Invited Reviewer, Florida Health Grants (Bankhead-Coley Cancer Research Program) (Follows NCI process for review)
Invited Reviewer, American Medical Association (Neoplastic Study Section)
Reviewer, Fellowship, International Union Against Cancer
Reviewer (ad hoc) University of Missouri Botanical Center Pilot Projects
Reviewer (ad hoc), University of Missouri internal LOIs to select one for Mary Kay

Foundation grant submission

Manuscript Review
BBA-Molecular Cell Research
Carcinogenesis
Endocrine Related Cancer
Environmental Health Perspective
J Agriculture & Food Chemistry
J Clinical Endocrinology & Metabolism
Oncogene


Book Chapter.

Abstracts.
King TL, Heesch CM, Friskey S, Ruyle BC, Kline DD, and Hasser EM (2012) Caudal ventrolateral medulla (CVLM) activation by acute hypoxia (AH) is independent of changes in arterial blood pressure (ABP). FASEB J March 29, 26:702.6

Zijin Zhou, King TL, Kline DD, Heesch CM, and Hasser EM (2012) Cardiovascular deconditioning augments baseline breathing as well as peripheral and central chemoreflex responses. FASEB J March 29, 26:702.14

Ruyle BC, Heesch CM, King TL, Kline DD, and Hasser EM (2012) Activation of nucleus tractus solitarii (nTS) neurons that project to the rostral ventrolateral medulla (RVLM) or hypothalamic paraventricular nucleus (PVN): Role of acute hypoxia (AH). FASEB J March 29, 26:702.15


Ostrowski TD, Barr SL, Dantzler HA, Hasser EM, Kline DD, and Heesch CM (2012) Expression of ROS catabolic enzymes in the medial nucleus tractus solitarii (nTS) of rats and upregulation during acute hypoxia. FASEB J March 29, 26:702.4


Austgen JA and Kline DD (2012) Serotonin 2A receptors augment synaptic transmission in the nucleus tractus solitarii (nTS).FASEB J March 29, 26:702.17
Active Grants

1R01 HL085108-01 (Kline DD) 4/01/2008-03/30/2013 NIH/NHLBI $250,000 direct/yr Adaptation of brainstem circuits to chronic hypoxia Role: PI

R01HL098602 (Kline DD) 04/01/2010 –03/31/2015 NIH/NHLBI Direct: $499,999 Plasticity of nTS output neurons in acute and chronic hypoxia Role: PI (MultiPI: Kline/Hasser/Heesch)

12POST11670002 (Ostrowski) 07/01/2012-06/30/2014 American Heart Association $46,000/yr Reactive Oxygen Species in Nucleus Tractus Solitarii Output Neurons: Neuronal Properties Following Intermittent Hypoxia

Professional Service

Committee/mentorship listing
Michael P. Matott, PhD Post-doctoral mentor
Tim D. Ostrowski, PhD Post-doctoral mentor
T. Luise King Committee member, Dept. of Biomedical Sciences
Kevin “Max” Coldren Committee member, Dept. of Biomedical Sciences
Randall Brown Committee member, Interdisciplinary Neuroscience Program
Nicholas P. Cooley Committee member, Dept. of Chemistry
Tim Pale Committee member, Dept. of Biological

Teaching Activity: Didactic and Clinical Teaching

Veterinary Physiology 5504, Co-Course Director
Veterinary Physiology 5504, Didactic teaching, 11 lecture hours, 18 laboratory hours, 12 examination hours, 4 review hours, 45 total contact hours, 126 students

Neural Control of the Circulation 9467 (Co-listed MPP 9437), Course Director
Neural Control of the Circulation 9467 (Co-listed MPP 9437), Didactic teaching, 5 lecture hours, 10 presentation hours, 4 examination hours, 19 total contact hours, 5 students
Publications


Presentations


“Molecular signaling pathways in ethanol preconditioning”. Department of Pharmacology, Tulane University, New Orleans, LA February 2012.

Active Grants

NIH; HL-095486, Program Project Grant, "Mechanisms of Microvascular Control and Coordination in Health and Disease"; Project Director, Project 3, “Microvascular Dysfunction: Impact of Ischemia-Reperfusion on ECM-Vascular Cell Interaction”; $262,000 per year, April 2010- March 2015.

Professional Service

1. American Physiological Society, Committee on Committees, 2011-2013

2. American Physiological Society, Cardiovascular Section Steering Committee, 2002-2013

3. Association of Physiology Department Chairs, 2004 – present

4. Association of Pharmacology Department Chairs, 2004 - present

6. Editorial Advisory Boards:
   - American Journal of Physiology: Heart and Circulatory Physiology; 2005 – present
   - Cardiovascular Research; 2008 – present
   - Circulation Research; 2010 - present

Publications


Presentations at Meetings

Growth signaling in the ventricular wall Experimental Biology April 22nd 2012, San Diego, CA

19th Annual Cardiovascular Day, Columbia, February 21, 2012 A model of hypertrophic cardiomyopathy induced by a protein tyrosine phosphatase Shp2 mutation demonstrates increased myofibrillar function. KS McDonald, LM Hanft, M Krenz


Myofilament Meeting 'Myofilament proteins as structural scaffolds and mediators of function', Madison, June 2 – 5, 2012. A model of hypertrophic cardiomyopathy induced by a protein tyrosine phosphatase Shp2 mutation demonstrates increased myofibrillar function. LM Hanft, TL Domeier, KS McDonald, M Krenz

Active Grants

RB 12-09, Research Board Grant, Krenz (PI)
Title: Role of Akt signaling in heart valve development and disease
02/01/12 – 01/31/13 (total cost $52,000)

URC 12-011, Research Council Grant, Krenz (PI)
Title: Regional microRNA profiling in hypertrophic-obstructive cardiomyopathy
11/1/11 – 10/31/12 (total cost $7319)
Krenz continued

**Professional Service**

April 2012   2012 Experimental Biology Meeting, Chair of Symposium “A Complex Interplay Coming together to Build the Heart”, April 22nd 2012

June 2012   Abstract Grader for Scientific Sessions 2012

June/July 2012   Abstract Session Builder for Scientific Sessions 2012

ad hoc Reviewer

2000 – present Basic Research in Cardiology
  Circulation Research
  Coronary Artery Disease
  FEBS Letters
  Journal of Applied Physiology
  Journal of Cardiac Failure
  Journal of Molecular and Cellular Cardiology
  Trends in Cardiovascular Medicine
  Yonsei Medical Journal

Feb / Mar 2012 Netherlands Organization for Scientific Research, Research Council for Earth and Life Sciences

July 2012 Congressionally Directed Medical Research Program (CDMRP), Tuberous Sclerosis Complex Research Program (TSCRP)

2011 - present Review Editor, Frontiers in Integrative Physiology
Harold Laughlin

Publications


Laughlin continued


Presentations


Active Grants

National Institutes of Health, R01; “Training: Muscle Blood Flow and Capillary Dynamics; Annual Direct $225,000; Duration: 7/15/10-4/20/14; 25% effort; PI.

Professional Service

Administrative Activity:
Chair of Biomedical Sciences
Curators’ Professor

Service Activity:
Member of the Editorial Board, Medicine and Science in Sports & Exercise. 2005 - Present
ASSOCIATE EDITOR: Journal of Applied Physiology. March 1, 2008 – Present

Manuscript Review for Journals:
1980-Present Avia. Space Environ. Med
1980-Present J. Applied Physiol
1981-Present Am. J. Physiol.
1985-Present Hypertension
1990-Present Microvascular Research
1993-Present Circulation
1993-Present Circulation Research
1994-Present Microcirculation

GRANT REVIEW COMMITTEES:
7/1/1990-Present National Institutes of Health Reviewer reserve
Publications


Presentations

Invited Presentation by the School of Pharmaceutical Sciences, Medical University of Herbei with title “Pre-clinical targeted therapy for combating advanced breast cancer” in Herbei, China on Oct. 2012

Active Grants


Liang, Yayun (Co-I), Hyder, S. M (PI), “Treatment of Breast Cancer and its Metastasis with APR-246 (PRIMA-1MET)” APREA AB $86,205 direct cost (2/1/12-7/31/12).

Liang, Yayun (Co-I), Hyder, S. M (PI), “Treatment and prevention of prostate cancer with novel inhibitors of cholesterol biosynthesis” College of Veterinary COR Award, $18,000 (2/1/12-12/31/12)

Professional services

9/2012-Present Adjunct Research Associate Professor, Dept. of Biomedical Sciences, College of Veterinary Medicine, University of Missouri, MO, USA
Editorial Board of Chinese Journal of Clinicians (International) (2011-2014)

Invited Presentation by the School of Pharmaceutical Sciences, Medical University of Herbei with title “Pre-clinical targeted therapy for combating advanced breast cancer” in Herbei, China on Oct. 2012

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


Presentations


Active Grants

National Institutes of Health (NIH) RO1. “Mechanisms of Microvascular Remodeling Progression.” Principal Investigator, Luis A. Martinez-Lemus (40% Effort), $2,250,000.00 for 2009-2014. Scored at 120 (3.5%).

National Institutes of Health (NIH) PO1. “Mechanisms of Microvascular Control and Coordination in Health and Disease.” Principal Investigator, Gerald A. Meininger. Core Director, Luis A. Martinez-Lemus (15% Effort), $8,470,000.00 for 2010-2014

Professional Service

American Physiological Society-Cardiovascular Section: NIH Liaison Committee (2012-2015)

Microcirculatory Society: Councilor (2011-2014)
Martinez-Lemus continued

Microcirculatory Society: Membership Committee (2010-2013)

Appointed member of the School of Medicine Research Council, University of Missouri-Columbia (2009-2012).

Appointed member of the Dalton Cardiovascular Research Center Safety Committee (2009-Present).

Appointed member of the Gender and Racial Diversity Equity Council Representation. School of Medicine, University of Missouri-Columbia (2009-2012).

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).

**Scientific Journals**

Editorial Board Member for “CardioRenal Medicine,” 2012-Present.


Editorial Board member for “Microcirculation,” 2010-2015.

*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*

*The Anatomical Record*

*Experimental Physiology*

*Poultry Science*

*BioMed Central Cell Biology*

*Hospital Practice*

*Microcirculation*

*Hypertension*

*Clinical and Experimental Pharmacology and Physiology*
Gerald Meininger

Publications


Clark, C.G., Z. Sun, G.A. Meininger and J.T. Potts. Atomic force microscopy to characterize binding properties of 7-containing nicotinic acetylcholine receptors on NK1-expressing medullary respiratory neurons. Experimental Physiology, 2012. (Epub ahead of print)


Amini, S., Z. Sun, A. Juriani, G.A. Meininger and K.E. Meissner. QD embedded microspheres as an evanescent light source for nearfield imaging. (In preparation for Nature Methods), 2012. {Shared senior authorship, Co-Corresponding Authors}


Seminars

“Biology at the tip of an atomic force microscope”, Annual Experimental Biology Meeting, San Diego, CA, April 2012.

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.

Active Grants

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, Public Health Service Grant, R01, “Acute mechanisms of vascular remodeling”, Principal Investigator, Luis A. Martinez-Lemus, Co-Investigator, G.A. Meininger (10% effort), $1,250,000 (direct) ($1,825,523 total) for 07/01/09-06/30/14.
Meininger continued

National Science Foundation; Evanescent Field-Enabled Atomic Force Microscopy for Nanoscale Imaging of Cell Membrane Dynamics. Principal Investigator, K Meissner, Co-Investigator, G.A. Meininger (8%), $200,000 (total) 09/01/2009-08/31/2011. No cost extension until 08/31/12.

Professional Service

Scientific Journals
Editorships
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.

Poster Judge at combined meeting of the British and US Societies for Microcirculation, Keble College, Oxford University, June 4-6, 2012.

Member Search Committee for Director of Office of Grant Writing and Publications, January-June 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.
Mark Milanick

Publications


Active Grants

2012-2014 NIH R21 DK09186 Developing a non-invasive monitoring system using NIR dyes inside erythrocytes 233,719

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
Luis Polo-Parada

Publications


Active Grants

Nanostructured High Surface Area Sensor Systems for Enhanced Detection. PI-Sheila Grant, Ph D. Co-PI, 10% Responsibility and credit. Funded by NSF. 6/2011-6/2012 $320,000


Professional Service

Problem Based Learning (PBL) Module 5 University of Missouri- Medical School -2012.

Advisor Ph. D. Students:

- Francisco Ramirez. Department of Biological Engineering Ph. D. Student.
- Jorge Gonzalez Castorena. Department of Biological Engineering. Ph. D. student.
Polo-Parada continued

Advisor Post-Doctoral:

- Dr. Asur Guadarrama Santana. University of Mexico. CYCADET. 2012-2013
- School of Medicine Research Council. 2012-2015
- Hearnes Center Committee. 2011-2013
- MU PREP (Post-baccalaureate Research Education Program). 2008-present

This is a program funded by NIH to increase research skills of BA/BS graduates from underrepresented minority or disadvantaged populations or with disabilities in order to enable them to enter and successfully complete PhD programs in the biomedical sciences.

National Chair and Co-Chair Services Experimental Biology Meeting. 2012

Study Section Participant

Advisory committee Member of the University of Missouri Doctoral Faculty (2009-2014).
# Leona Rubin

## Professional Service

**2011-Pres**  
Faculty/Provost Task Force on eLearning/Online Academic Programs: Chair. Develop roadmap and policies for development of online courses including issues of quality, intellectual property and impact on face to face courses.

**2011-Pres**  
University of Missouri System Retirement and Staff Benefits Committee: Chancellor appointed.

**2011-Pres**  
Strategic Plan Progress Committee: Tasked with maintaining a “living” web version of the campus strategic plan by identifying and collecting action plan updates and data for plan objectives.

**2011-2012**  
Faculty Grievance process Oversight Committee, Chair

**Service as Part of MU Faculty Council on University Policy**

**2006-2012**  
MU Faculty Council on University Policy: College of Veterinary Medicine Elected Representative
**Publications**


Westcott EB, Goodwin EL, Segal SS and Jackson WF. Function and expression of ryanodine receptors and inositol 1,4,5 trisphosphate receptors in smooth muscle cells of murine feed arteries and arterioles J Physiol 590.8: 1849-1869, 2012. PMID: 22331418

Behringer EJ and Segal SS. Tuning electrical conduction along endothelium of resistance arteries through Ca2+-activated K+ channels. Circ Res 110:1311-1321, 2012. PMC3467972

Socha MJ, Domeier TL, Behringer EJ and Segal SS. Coordination of Intercellular Ca2+ Signaling in Endothelial Cell Tubes of Mouse Resistance Arteries. Microcirculation 19:715-770, 2012. (Cover Illustration) PMC3502682

**Presentations**


Behringer E and S Segal. Tuning electrical conduction along endothelial cell tubes via Ca2+-activated K+ channels. FASEB J 26:1058.12, 2012.


Behringer E and S Segal. Aging impairs electrical conduction along endothelial tubes via activation of KCa channels by reactive oxygen species. Joint meeting of British and USA Microcirculatory Societies, Oxford, England, 07/12.
Segal continued

Seminars

Physiological Soc. 2012 (07/2012, Edingburg, Scotland; Session: Blood Flow Regulation: From Rest to Maximal Exercise) “Spreading the Signal for Vasodilation”


Symposium celebrating the 20th Anniversary of the Robert M. Berne Cardiovascular Research Center, University of Virginia (11/2012; Charlottesville, VA) “Neuromodulation of Cell-to-Cell Signaling In Resistance Networks”

Active Grants

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

R01 HL086483; National Institutes of Health/NHLBI (Segal, PI)
Title: “Microcirculation in Aging Skeletal Muscle”
Current Project Period: 09/01/2007-02/28/2014 (with 1-year no-cost extension)

Professional Service

Invited Reviews


Westcott EB and Segal SS. Perivascular innervation: A multiplicity of roles in vasomotor control and myoendothelial signaling. Microcirculation; accepted 10/25/2012) doi:10.1111/micc.12035 PMID: 23289720
 Yoshiro Sohma

Publications


Presentations


Active Grants


Sohma continued


2011.4 – 2013.3 Research support: PI, “Approach to a novel membrane transport physiology based on a direct observation of water and lipids by a non-linear optical microscopy” funded by Keio Gijuku Fukuzawa Memorial Fund for the Advancement of Education and Research


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


Co-chairman in Symposium “Motion pictures of the functional membrane molecules in action - What we want to see in the movies and what they will teach us -”, 89th Annual Meeting, The Physiological Society of Japan, Matsumoto, Japan, 2012

Publications


Sowers continued


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. Sowers (PI), $1,250,000.

2009-2014 NHLBI, Ang II and Aldosterone Effects on Insulin Resistance in Cardiovascular Tissue, James R. Sowers (PI), $1,250,000.

2008-2012 VA Merit Review, Mechanisms of Angiotensin II and Aldosterone Inhibition of Insulin Metabolic Signaling, James R. Sowers (PI), $650,000.
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
Cardiometabolic Syndrome 2006-2010
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
- 1996-2004    Endocrinology, Nutrition, Metabolism and Cardiovascular Diseases, Journal of Clinical Endocrinology & Metabolism
- 1994-present  Journal of Human Hypertension
- 1991-present  Metabolism
- 1991-present  Hypertension

Editorships - Reviewer

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 Clinical Science Research and Development Scientific Merit Review Board
- VCMB (Vascular Cell Molecular Biology) Study Section - NIH
- 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
Sowers continued

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
• Chair Brooklyn VA R and D 2001-2004

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

HONORS & AWARDS
2012 Irvine Page-Alva Bardley Lifetime Achievement Award in Hypertension
Publications


Active Grants


Professional Service

2004-Present Member, Executive Committee, College of Veterinary Medicine

2004-Present Member, Council of Research Advisors, Asst Chancellor for Research

2004-Present Director, Grant Review Program, College of Veterinary Medicine

2004-Present Co-Chair, Search Committee, Clinician Scientists-Mission Enhancement, Division of Cardiology, Dept. of Internal Medicine, College of Medicine

1998/2000/02/04/06/08/10/12 Course Director with Dr. C. Hardin, Control of Energy Metabolism (VBMS/MPP 9431), Joint listed: Biomed Sci and Med Pharm & Physiol: Selected lectures (10 3-hr sessions)
Xiaoqin Zou

Publications


Medical Research Council, School of Medicine, University of Missouri-Columbia, Missouri, 2012.

Active Grants

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: NIH/ National Institute of General Medical Sciences Grant # 1R21GM088517 Project Title: A new scoring framework for selecting structural models Funding Period: September 30, 2009 – November 30, 2012 Total Amount: $411,125 Principal Investigator: Xiaoqin Zou

Professional Services


We have overexpressed cyclophilin-D (CypD) in the hearts of mice (CypD TG). CypD is a critical component of a protein complex in the mitochondria call the mitochondrial pore, that play a role in the death of heart cells during a heart attack and other forms of heart disease.

Overexpression of CypD resulted in an enlarged heart that was less efficient at pumping blood than hearts from normal mice. When we looked at the structure of the heart cells we saw that increased CypD caused a massive enlargement and swelling of mitochondria compared to normal mice. This in turn eventually lead to the death of the heart cells.