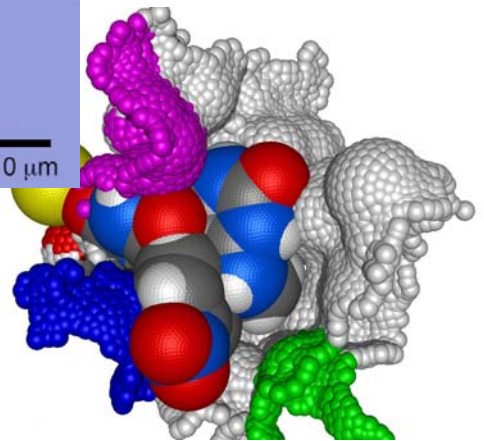
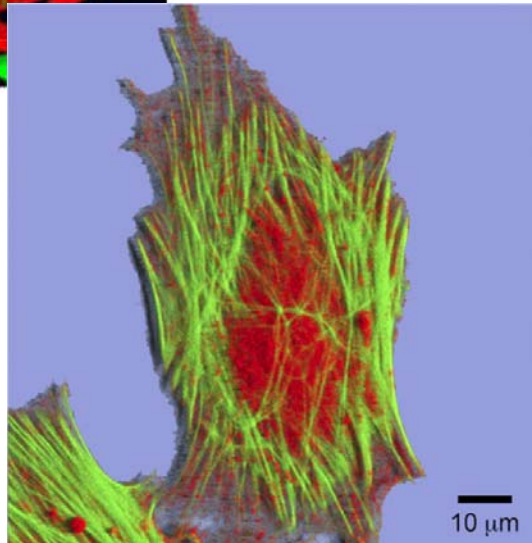
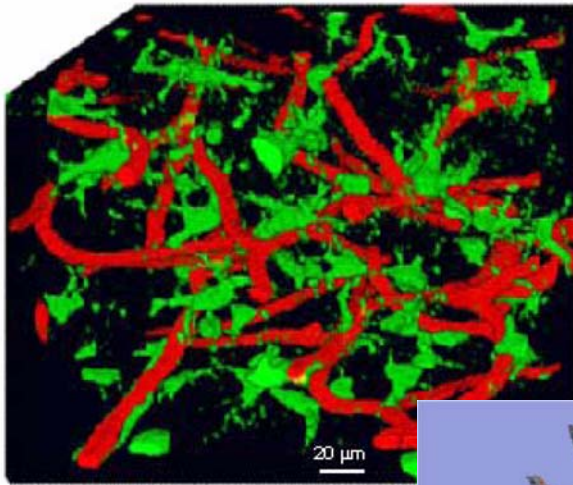




Dalton Cardiovascular Research Center



Annual Report FY 06

“Committed to Collaboration in Research and Teaching”

Explanation of Figures

Top

Dr. Shinghua Ding

Image of relationship between astrocytes (green) and the microvascular network (red) in the mouse cerebral cortex.

Middle

Dr. Zhe Sun

The distribution of $\alpha 5 \beta 1$ -integrins on VSMC. Cells were immunofluorescently labeled with antibodies against $\alpha 5 \beta 1$ -integrin (red) and with phalloidin against actin filaments (green).

From: Sun, Zhe, Luis A. Martinez-Lemus, Andreea Trache, Jerome P. Trzeciakowski, George E. Davis, Ulrich Pohl, and Gerald A. Meininger. Mechanical properties of the interaction between fibronectin and $\alpha 5 \beta 1$ -integrin on vascular smooth muscle cells studied using atomic force microscopy. *Am J Physiol Heart Circ Physiol* 289: H2526–H2535, 2005.

Bottom

Dr. Xiaoqin Zou

Computational structural biology and molecular interactions. Image illustrates rational inhibitor design against viral protein, PMM/PGM. The inhibitor is shown in a sphere representation and colored by atom types. The active site of the viral protein is shown in grainy surface representation.

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FROM THE DIRECTOR

It has been a busy year and half since my arrival and I am pleased to distribute our Annual Report for 2006. We have undergone a number of significant changes all of which point to an atmosphere of future growth and expansion of the Dalton Cardiovascular Research Center.

We have completed 5 new faculty hires; 3 with the Department of Medical Pharmacology and Physiology in the School of Medicine, one with the Department of Biomedical Sciences in the College of Veterinary Medicine and one with the Department of Bioengineering. In addition, we have partnered with the Department of Physics and Department of Biochemistry to support one of our Dalton Investigators in a new tenure track faculty position. We have also begun the process of partnering with the Department of Biomedical Sciences in the College of Veterinary Medicine to hire another new faculty member to be housed within the Dalton Cardiovascular Research Center. Collectively, these initiatives highlight the interdisciplinary nature of the Dalton and the strength obtained by cross campus collaboration.

In response to the need for modernization and creation of more usable research space in the Dalton Cardiovascular Research Center, we have updated and renovated the administrative area and added a small conference room, we have renovated faculty office space and we have modernized and renovated 11 laboratories. Our core facilities have also been substantially enhanced with the addition of a state-of-the-art imaging core that houses both a confocal microscopy system and a multiphoton microscopy system that can be used for cell and tissue level work and can accommodate the needs of investigators with wet laboratory preparations. In addition, we have two Atomic Force Microscope systems within the Center that are available on a collaborative basis. As a consequence of this emphasis on imaging, our facilities now rival anything available in the cardiovascular field. We will continue to strive to keep our facilities modern and in keeping with the needs of our investigators to stay technically competitive.

Our Investigators continue to maintain a solid record of research funding with \$3.1 million in research funding for this reporting period. A 30% increase in new funding over a similar period last year. Despite the difficult times we all face ahead with obtaining national funding, Dalton Investigators have aggressively submitted a record number of research proposals in 2006 for funding totaling \$15 million. With the addition of new research space and new investigators we are poised to take advantage of new opportunities to grow our research programs.

The Dalton Cardiovascular Research Center has a rich history of building multidisciplinary, cross campus partnerships. We will continue to honor this history and to welcome new interdisciplinary collaborative programs. I invite all of you to review our report and introduce yourselves to our investigators and the work they have accomplished.

Gerald A. Meininger, Ph.D.
Director, Dalton Cardiovascular Research Center

OVERVIEW

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 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, and veterinary medicine and surgery 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, 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 published 74 manuscripts in nationally recognized journals and books and gave 101 invited presentations.

The Dalton Cardiovascular Research Center is accredited by both the American Association for the Advancement of Laboratory Animal Care and the American Association of Laboratory Animal Sciences.

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Zach Richardson
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Stacey Mathes
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Building Coordinator

Gene Smith
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Biomedical Engineering

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

Cystic Fibrosis

Investigators: Clarke, Hwang, Milanick, Price, Soma, Zou

Exercise/Inactivity Including Atherosclerosis, Muscle Biology, Obesity, Type II Diabetes, and Vascular Biology

Investigators: Booth, Bowles, Segal, Hamilton, Hasser, Hill, Huxley, Jones, Korthuis, Laughlin, Martinez-Lemus, Meininger, Mueller, Polo-Parada, Price, Rubin, Soma, Terjung

Membrane Transport

Investigators: Clark, Gillis, Gu, Huxley, Hwang, Milanick, Polo-Parada, Price, Rovetto, Rubin, Zou

Microcirculation

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

Muscular Dystrophy

Investigators: Childers, Kornegay

Neurohumoral Control of the Circulation Including Ageing, Hypertension, Heart Failure, Respiration, and Salt and Water Homeostasis

Investigators: Blaine, Fadel, Foley, Hasser, Heesch, Kline, Meininger, Milanick, Pamidimukkala, Price, Schadt, Segal, Potts, Ding

Tumor Angiogenesis

Investigators: Hyder, G.E. Davis

DCRC Committees

The Internal Advisory Committee:

Dr. Gerald A. Meininger, Chair
Dr. Virginia H. Huxley
Dr. Alan W. Jones
Dr. Kevin Gillis
Dr. Ronald L. Terjung
Dr. Edward H. Blaine
Dr. Michael A. Hill

The Appointment and Promotions Committee:

Dr. Salman M. Hyder
Dr. Kevin Gillis
Dr. Jeffery T. Potts
Dr. Tzyh-Chang Hwang
Dr. Luis A. Martinez-Lemus

Scientific Program Committee:

Dr. Michael A. Hill, Chair
Dr. Salman M. Hyder
Dr. Eileen M. Hasser
Dr. Kevin Gillis

Core Facilities Committee:

Dr. Luis A. Martiniez-Lemus, Co-Chair
Dr. Tzyh-Chang Hwang, Co-Chair
Dr. Luis Polo-Parada
Dr. Kevin Gillis
Dr. Lane Clarke

SUMMARY OF ACCOMPLISHMENTS

Publications and Presentations

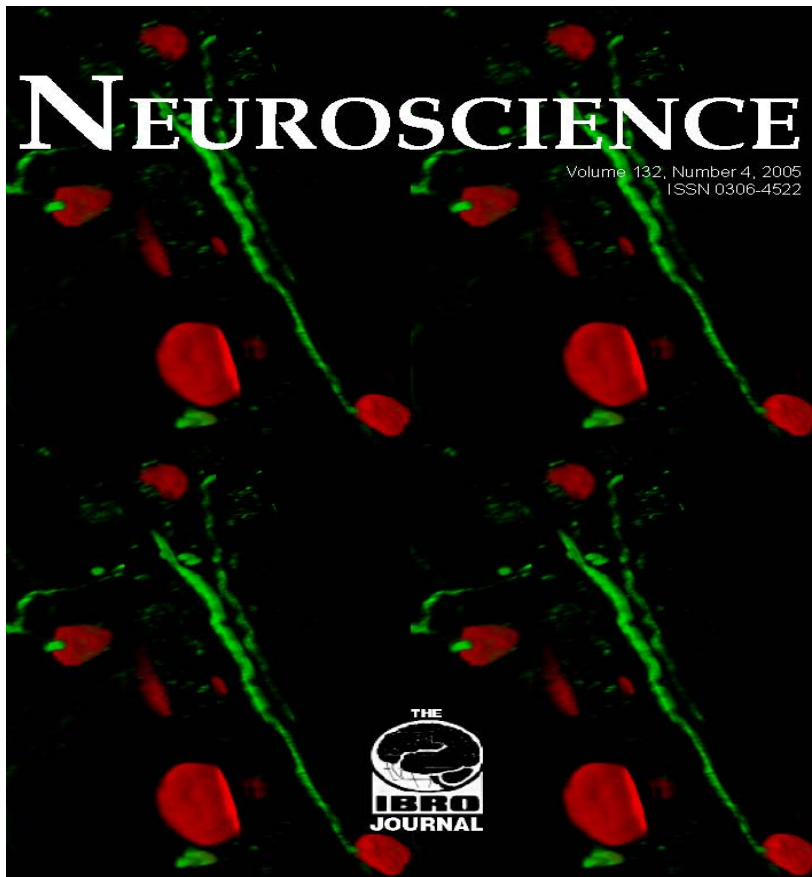
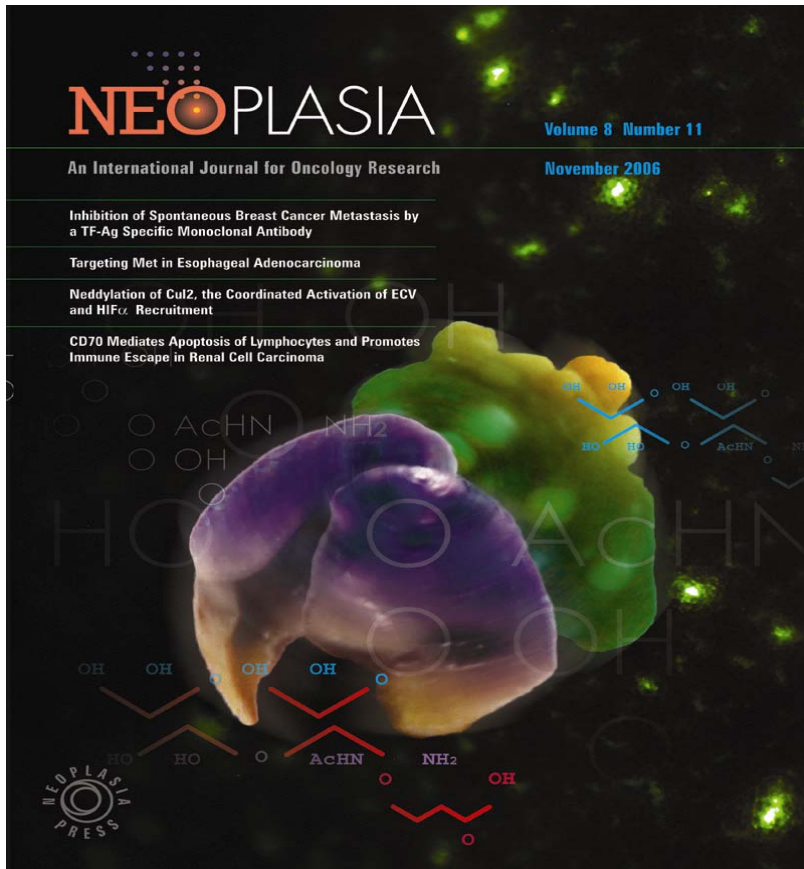
- **110** Articles published
- **105** Invited Presentations

Awards and Peer Review

- There are 36 current awards/contracts
- 13 awards/contract proposals totaling more than \$3,100,000 for research was awarded during FY06 utilizing the Dalton Center
- There are 27 grant/contract proposals currently pending totaling more than \$15M
- 7 graduate students were supported by R90/T90 training grants totaling \$320,157
- 5 investigators serve as editors or are on editorial boards of 9 scientific journals
- 10 investigators reviewed articles for 29 scientific journals
- 7 investigators reviewed grant applications for 12 granting agencies
- 6 investigators serve on 12 national study sections
- 3 patents were filed, pending or issued

Education and Training

- 25 Resident Investigators
- 15 Non-resident Investigators
- 30 Research Staff
- 9 Non Tenure Track Faculty
- 8 Post Doctoral Fellows
- 17 Graduate Students
- 17 Undergraduate Students
- 6 Administrative Staff
- 5 Building support Staff
- 1 High School Student



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2864 Interaction of L2L3 receptor-associated protein 4 (LRP4) with postsynaptic scaffolding proteins via its C-terminal FQZ domain-binding motif, and its regulation by Cdk5/p35-dependent protein kinase 1
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2930 Selenin and glycosylated peptide molecule neurite outgrowth via protein kinase C-mediated activation of extracellular signal-related kinase
J. Hawak, R. Narasimhan and M. R. Picotino

2947 Immunocytochemical evidence for the existence of substance P receptor PK11 in serotonergic neurons of rat and mouse dorsal raphe nucleus
B. Lachaux, M. Radzinski, Desarmes

Continued on inside back cover

Cover Illustration

Investment of *gfp* in Parkinson's disease: the left part of a model synapse from a Tg1 sensor targeting a synapse for the synaptic locus *ACH* (axonal) with increased axonal release and inhibition. For details see Ben-Shaul et al. (pp. 2915–2922)

Agonal-induced *Ca²⁺* transients in LC neurons may indicate acute denervation to OLF or stress. Merged optical image (left part) of a coronal section through the LC, labeled to visualize *Ca²⁺* response (red) and tyrosine hydroxylase (green). For details see Jones et al. (pp. 2991–2998)

Optical imaging of neural activity during hypoxia in the embryonic ventral respiratory group using a voltage-sensitive dye and high-speed cameras. Right panel: from a study of shifts between equine and human. For details see Puh and Puh (pp. 3055–3062)

Electrophysiological markers of vocal familiarity: good-earner 'warblers' respond to a female voice (left) and male (right) songs as well as to female voice (left) and male (right) after structural onset (top) and bottom (see, respectively). For details see Bradlow et al. (pp. 3018–3026)



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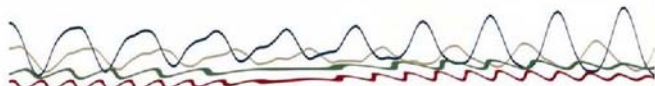


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European Journal of Neuroscience



Combined Optical and Atomic Force Microscopy: Advancing Biological Research

by Dr. Andrea L. Slade, Veeco Instruments Inc.

Atomic force microscopy (AFM) has become increasingly important in biological research. An AFM can mechanically approach a sharp probe over a sample, and by monitoring tip-sample interactions, it produces a true three-dimensional image of the sample surface. AFM has become a widely accepted technique for investigating biologically related phenomena because of its unique capability for molecular resolution imaging of biomolecules and their surfaces under near-native physiological conditions, both in real time and *in situ*. The high force sensitivity afforded by single-molecule force spectroscopy (pico-coaxors) also has facilitated in situ characterization of molecular intra- and interactions.

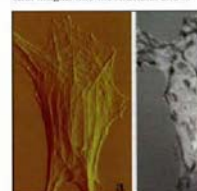


Figure 1. Vascular smooth muscle cells were imaged by AFM (a) and interference reflection microscopy (b). Overlaying of the two images (c) shows the actin filaments of the cytoskeleton, as observed by AFM, remaining in the focal adhesion, as observed by interference reflection microscopy (for arrowhead, color). Blue and red indicate the darker and lighter areas, respectively. Images acquired with permission of the Journal of Biomedical Optics.

Atomic force microscopy (AFM) has become increasingly important in biological research. An AFM can mechanically approach a sharp probe over a sample, and by monitoring tip-sample interactions, it produces a true three-dimensional image of the sample surface. AFM has become a widely accepted technique for investigating biologically related phenomena because of its unique capability for molecular resolution imaging of biomolecules and their surfaces under near-native physiological conditions, both in real time and *in situ*. The high force sensitivity afforded by single-molecule force spectroscopy (pico-coaxors) also has facilitated in situ characterization of molecular intra- and interactions.

Although the AFM has provided powerful insights into the structural and kinetic aspects of molecular self-assembly, it is inherently a topographical technique, limited to characterizing the surface of a biomolecule or biomolecular assembly. For instance, the interaction between individual protein molecules or the binding of a protein to the outer membrane of a cell is readily apparent with AFM imaging or with force measurements, but AFM cannot discern subtle changes in the secondary/tertiary structure of these biomolecules or detect the onset of intracellular signaling events that occur as a result of these interactions. As such, there is a clear need for complementary data that provides information about the molecular structure or chemical state of a sample and that can be spatially correlated with the topographical features resolved by AFM.

Optical microscopy is an ideal complementary tool to an AFM. For imaging cells, light microscopy techniques such as differential interference contrast and phase contrast allow visualization of internal cell structures. Although smaller biomolecules, such as proteins and DNA, are not directly observable by light microscopy, they can be labeled with fluorescent tags and visualized by fluorescence microscopy, including confocal laser scanning, total internal reflectance fluorescence, and Förster resonance energy transfer. This not only provides a means of spatially identifying specific biomolecules within a sample, but also can provide information as to their structural or chemical state through the presence or absence of a fluorescence signal. Optical microscopy also has faster data acquisition and, hence, the ability to detect molecular events that occur on faster timescales than an AFM can resolve.

By integrating optical microscopy and an AFM into a single imaging platform, the optical image can be directly correlated with the AFM data, providing a powerful tool for studying dynamic biological processes in real time and *in situ*. Several research groups are involved in this type of multimodal imaging and have applied it to the investigation of the mechanical properties of proteins and cells, the structure of model membrane systems, and protein-membrane interactions. For example, at Texas A&M University System Health Science Center in College Station, professors Andrea Trache and Gerald Neisinger have combined AFM with interference reflection microscopy to investigate cellular responses to changes in the external environment. The images taken of a live cell with each method can be overlaid to demonstrate the correlation of the structure of the cytoskeleton

observed by AFM with the position of the cellular focal adhesions on the underlying substrate, as revealed by interference reflection microscopy (Figure 1). The AFM probe can mechanically perturb the cell while interference reflection microscopy monitors the changes in focal adhesions in real time.

At the University of Toronto, professor Christopher Vip and PhD student James Shaw have combined AFM and confocal laser scanning microscopy to investigate domain formation in model membrane systems and, in particular, the influence of fluorescent labels on the partitioning behavior of membrane components. They found that fluorescent labeling prevented sphingomyelin, a known component of lipid "rafts," from partitioning into raft domains, as identified by confocal imaging of membrane-bound fluorescent cholera toxin and the height differences between ordered and disordered lipid domains, as observed by AFM (Figure 2). Their results caution against the interpretation of fluorescence data alone and further support the use of the combined method as a means of spatially identifying membrane domains.

Combined optical and AFM imaging platforms have addressed some of the key limitations of both individual techniques and have opened the door for researchers to apply these integrated instruments to study the fundamental and challenging issues of molecular science, namely, through the direct correlation of molecular structure and function.

Meet the author
Dr. Andrea L. Slade is lead science applications scientist at Veeco Instruments Inc. in Santa Barbara, Calif.; e-mail: aslade@veeco.com.

References
1. A. Trache and G.A. Neisinger (November/December 2005). Atomic force-microscopy integrated microscope for monitoring molecular dynamics in live cells. *J Biomed Opt*; 064023.
2. J.E. Shaw et al (March 2005). Correlated fluorescence-AFM microscopy of membrane domains: Structure of fluorescence probes determines lipid localization. *BIOPHYS J*; pp. 2170-2178.

AFM

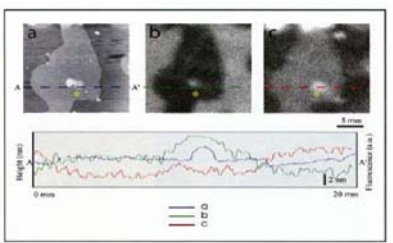


Figure 2. AFM images of supported lipid membranes (a) reveal the presence of two types of lipid domains, with the taller, more ordered domains corresponding to lipid "rafts." Confocal microscopy of fluorescently labeled sphingomyelin in the same region of the bilayer (b) reveals sphingomyelin to have been excluded from the raft domains. Confocal microscopy of fluorescently labeled cholesterol in the same region of the bilayer (b) shows them to have bound to their ganglioside receptors. (a) Rafts correspond to the taller lipid domains, as observed by AFM, confirming them to be lipid rafts. Courtesy of James Shaw and Christopher Vip, University of Toronto.

Virtual Microscopy

BIOPHOTONICS
INTERNATIONAL

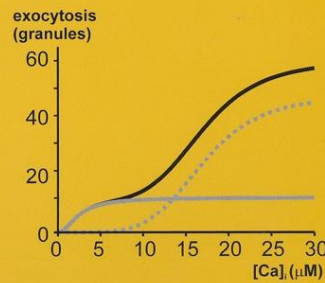
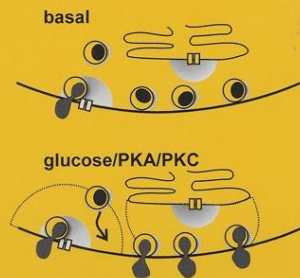
Atomic Force Microscopy

micro-architectural details of a live cell to be revealed by AFM

Surface Plasmon Resonance

THE JOURNAL OF General Physiology

VOLUME 124 · NUMBER 6 · DECEMBER 2004



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Experimental Physiology

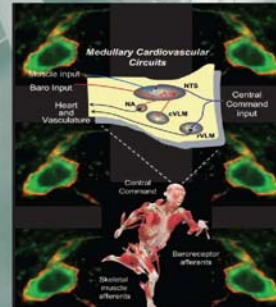
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Volume 91, Number 1, January 2006

Experimental Physiology

Translation & Integration

A Publication of the Physiological Society



Neural control of the circulation during exercise



Gerald A. Meininger

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Education:
Ph.D. from the University of Missouri, Columbia, M.S. and B.S. from
Central Michigan University

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

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 9) Mechanisms of blood flow autoregulation.

Laboratory models include study of the intact microcirculation, study 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). 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 throughput 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. In order to overcome technical limitations that have slowed progress in this field we have been developing hybridized microscopy systems in which we combine atomic force microscopy with advance fluorescence microscopy techniques. With this technology 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

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E-mail: hillmi@missouri.edu

Education:
Ph.D. and M.S. from the University of Melbourne

Appointment: Professor, Department of Medical Pharmacology and Physiology

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 Ca^{2+} . 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



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Education:
Ph.D., M.A. A.B. from University of Missouri, Columbia

Appointment: Professor, Department of Medical Pharmacology and Physiology

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 on-set 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 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.

Principal Investigators



Frank W. Booth

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Education:
Ph.D. from the University of Iowa, B.S. from Denison University

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

Research

Research interests in my laboratory currently are focusing 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 p21^{Cip1/WAF1}, p27^{Kip1}, 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, Associate Director of the National Center for Gender Physiology

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Education:

Appointments: Associate 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 2 distinct channels, the intermediate-conductance, calcium-activated K channel (IKCa1) and channels underlying store-operated calcium entry, most likely TRPC channels. We are interested on these as they are upregulated during CSM phenotype modulation that occurs during vascular injury.

Principal Investigators

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 it's 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



Martin (Casey) Childers

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E-mail:

Education:
No Information Available

Appointments: Associate Professor, Department of Physical Medicine and Rehabilitation
Associate Professor, Department of Biomedical Sciences

Research

Dr. Childers is interested in role of skeletal muscle proteins, and how they interact to protect the muscle cell from stretch-induced damage during contraction. This line of research explores the effects of protein deficiencies that cause muscle diseases (muscular dystrophies) in cultured muscle cells, single fiber and in whole muscle functional assays. Methods used in the Childers lab include targeted gene expression in cultured cells and in vivo transfection of plasmid DNA in mice. The lab also utilizes a canine model of Duchenne muscular dystrophy, termed golden retriever muscular dystrophy (GRMD). The GRMD colony at the University of Missouri is the largest and most successful colony worldwide. This canine model of dystrophin deficiency allows for function assessment of muscle strength and response to pharmacologic agents potentially useful in the treatment of humans with muscular dystrophy. The overall goal of this line of research is to better understand the functional role of muscle cell proteins and how they act to protect the cell during contraction.



Lane Clarke

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Education:
Ph.D. from North Carolina State University, D.V.M., M.S. and A.B. from the University of Missouri, Columbia

Principal Investigators

Appointments: Associate Professor, Department of Physical Medicine and Rehabilitation
Associate Professor, Department of Biomedical Sciences

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, $\text{Cl}^-/\text{HCO}_3^-$ exchangers and $\text{Na}^+/\text{K}^+/\text{2Cl}^-$ cotransporters. Molecular studies in the laboratory involve the measurements of gene expression in the mice (quantitative real-time PCR, Northern blots, 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.



George E. Davis

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Education:
Ph.D., M.D. from the University of California, B.S. from Arizona State Univ.

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

My laboratory focuses on the following questions relevant to angiogenesis, wound repair and cancer research:

1. How do endothelial cells form cell-lined tube structures with lumens in three-dimensional (3D) extracellular matrices?
2. How do endothelial cells and other cell types such as tumor cells invade 3D matrices?
3. To what extent do endothelial cells directly or indirectly play a role in tumor invasion and metastasis?
4. What molecular events control the process of vascular regression?
5. How do vascular supporting cells, such as pericytes, stabilize vascular tubes?
6. How do distinct matrix metalloproteinases and their inhibitors control the processes of vascular morphogenesis versus regression in 3D matrices?
7. How do extracellular matrix fragments (matricryptins) regulate vascular morphogenesis versus regression in normal versus diseased states (e.g. diabetes)?

Principal Investigators



Michael J. Davis, Associate Department Head, Medical Pharmacology and Physiology

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Ph.D. from the University of Nebraska, B.S. from the University of

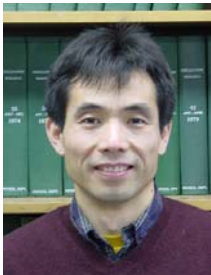
Appointments: Professor, Department of Medical Pharmacology and Physiology

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 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

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Appointment: Assistant Professor, Department of Biological Engineering

Principal Investigators

Research

My current research focuses on glial cell function and neuron-glia interactions in central nervous system using state of the art in vivo two photon fluorescent imaging and electrophysiology. My is also in also is interested in stem cell differentiation and transplantation.



Paul J. Fadel

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Education:
Ph.D. from the University of North Texas, M.S. from Northeastern University, Health Science Center, B.S. from Brooklyn College

Appointment: Assistant Professor, Department of Medical Pharmacology and Physiology

Research

Our laboratory's research focus entails the investigation of neural cardiovascular control at rest and during exercise in humans with a specific emphasis on the sympathetic branch of the autonomic nervous system. Ongoing studies involve assessing sympathetic responses during various physiological manipulations including isometric and aerobic forms of exercise, lower body negative pressure to simulate the effect of gravity when one stands up, and infusions of pharmacological agents. Studies are performed in normal healthy subjects as well as in patients with various pathophysiological conditions such as heart failure and hypertension. Our laboratory obtains direct measures of sympathetic neural firing using the technique of microneurography. This measurement allows for the assessment of moment-to-moment as well as long term changes in sympathetic nerve activity. Also, with the application of partial autospectral and time series analyses to muscle sympathetic neurograms we are beginning to investigate the central origin(s) and pattern(s) of sympathetic discharge in humans. Our current research focus is on the neural mechanisms that contribute to exercise-induced sympathoexcitation as well as the peripheral modulators of sympathetically-mediated 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.



Charles (Mike) Foley

Office:
Phone:
E-mail:

Education:
Ph.D., D.V.M. from the University of Missouri, Columbia

Principal Investigators

Research

Our research is aimed at understanding how the brain controls blood pressure and the cardiovascular system. Our overall interest is in neural pathways and neurotransmitter systems involved in autonomic control of the circulation. We are focusing primarily on the interaction of several critical transmitter systems including glutamate (both ionotropic and metabotropic receptors), aminobutyric acid (GABA), angiotensin II, and catecholamines. In addition, we are interested in how central nervous system control of blood pressure is altered by various physiologic and pathophysiologic states. We currently are involved with projects that investigate changes in autonomic and humoral function following specifically either cardiovascular deconditioning or pregnancy. Our laboratory strives to integrate multiple levels of physiology extending from whole animal studies to expression changes of specific proteins in particular regions of the brain. Changes in the GABA neurotransmitter/receptor system within the brain seem to be involved in the altered autonomic control of blood pressure observed during pregnancy. Our laboratory is investigating if changes in expression of specific subunits of the GABAA receptor occur during pregnancy. We hypothesize that changes in expression during pregnancy increase the sensitivity of GABA receptors to positive modulation by metabolites of progesterone. If so, this could contribute to altered control of sympathetic nerve activity observed during pregnancy. The current studies will evaluate changes during pregnancy in GABA sensitivity contributing to control of sympathetic activity and correlate this altered autonomic control with observed pattern of expression of GABAA receptor subunits within specific brain nuclei. Another project we are involved with is investigating the effects of cardiovascular deconditioning which occurs after a period of bedrest 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 bedrest 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.



Kevin D. Gillis

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Education:
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Appointments: Professor, Department of Biological Engineering
Adjunct Professor, Department of Medical Pharmacology and Physiology

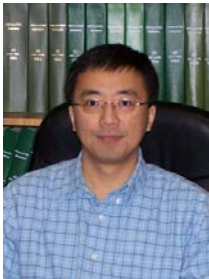
Research

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

Principal Investigators

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.



Liqun (Andrew) Gu

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Education:
Ph.D. from Nankai University

Appointment: Assistant 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.

Principal Investigators

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.



Marc Hamilton

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Education:
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Appointment: Assistant Professor, Department of Biomedical Sciences

Research

Dr. Hamilton studies the molecular and physiological mechanisms by which physical inactivity causes chronic metabolic diseases related to poor plasma lipid metabolism (coronary artery disease, Type II diabetes, obesity). A focus of the research in our laboratory is on translational research. In so doing, studies of humans, animal, and cell cultures are performed with the goal of integrating fundamental new insights regarding molecular processes while seeking solutions to practical clinical outcomes for metabolic diseases caused by physical inactivity. Multidisciplinary work exposes lab members to a diversity of modern research techniques. Studies have sought to discover the genes and signals linking physical inactivity to disease, especially those processes related to lipoprotein metabolism and skeletal muscle metabolism. A major question we are addressing is the underlying role of lipid metabolism in signaling for adaptations within vascular cells as one explanation for why exercise prevents atherosclerosis. This work largely involves pigs, isolated blood vessels in culture, and pri-

Principal Investigators

mary endothelial cell cultures. Rat and human work is also performed to understand regulation of processes controlling muscle metabolism and plasma lipids. Using microarray methodologies, our laboratory has been characterizing the response of a large percentage of the genome to exercise training, inactivity, and identifying both the transcriptional and post-transcriptional events influenced by lipids. In both human and animal studies, we have been testing the new paradigm of “inactivity physiology”. Studies are partly focused on comparing and contrasting the underlying metabolic responses to normal non-exercise physical activity to more intense and structured exercise. These studies are leading to the emerging school of thought that sitting too much (non-exercise activity deficiency) is a unique stimulus from exercising too little (exercise deficiency), while both types of physical activity can produce potent cellular signals important for combating the metabolic problems associated with metabolic syndrome, coronary artery disease, Type II diabetes, and obesity.



Eileen M. Hasser

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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

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Education:
Ph.D. from University of Texas Health Science Center, B.S. from New Mexico State University

Principal Investigators

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, Director of the National Center for Gender Physiology

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Education:
Ph.D. from the University of Virginia, B.A. from Hollins University

Appointments: 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.

Principal Investigators

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

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Education:
Ph.D. from The Johns Hopkins University, M.D. from the National Yang-Ming Medical School, M.S. from the National Tawain University School of Medicine

Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

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

Principal Investigators

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

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Education:
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Appointment: Associate 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.

Principal Investigators



Allan Jones

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Education:
Ph.D. from the University of Pennsylvania

Appointment: Professor Emeritus, Department of Medical Pharmacology and Physiology

Research

My research program currently focuses on mechanisms of membrane regulation and vascular smooth muscle function leading to abnormalities associated with hyper-lipidemia as well as adaptive mechanisms during exercise training. We are 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. We have 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. These studies involve close collaboration with members of a program project team on exercise physiology, the Center for Gender Physiology and Environmental Adaptation, and the Center for Diabetes and Cardiovascular Health. Methods utilize microfluorometry of calcium probes, digital image analyses, patch clamp techniques, radio isotopes fluxes, contractile responses, phosphor-image analyses, and biochemical and immuno-measures of AMP kinase.



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Education:
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Appointment: Assistant Professor, Department of Biomedical Sciences

Research

Our laboratory focuses on the autonomic nervous system, in particular the cardiovascular and respiratory system. These vital systems operate to keep our bodies within “normal” physiological limits to preserve homeostasis. When challenged acutely or chronically with low environmental oxygen levels (hypoxia) respiration, blood pressure and heart rate compensate to maintain arterial blood gas levels. This can happen during high altitude ascent 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

Principal Investigators

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.



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Appointment: Professor, Department of Biomedical Sciences

Research

Duchenne muscular dystrophy (DMD) is an X-linked recessive disorder affecting approximately 1 of 3,500 newborn human males in which absence of the protein dystrophin causes progressive degeneration of skeletal and cardiac muscle. Currently, no treatment halts or reverses progression of DMD. Although cellular and gene therapies are promising, key questions must first be addressed in relevant animal models. Spontaneous forms of X-linked muscular dystrophy due to dystrophin deficiency have been identified in mice, multiple dog breeds, and cats. Unlike the dystrophin-deficient mdx mouse, which remains relatively normal clinically, affected dogs develop progressive, fatal disease strikingly similar to the human condition. Accordingly, studies in the canine dystrophin-deficient models, such as golden retriever muscular dystrophy (GRMD), are more likely than those in mdx mice to predict pathogenesis and outcome of treatment in DMD.

Both conditions (XMD and DMD) are X-linked recessive traits in which absence of the protein dystrophin causes progressive degeneration, fibrosis and mineralization of skeletal and cardiac muscle. We have previously shown that GRMD results from a defect in RNA processing due to a single base change in the 3' consensus splice site of intron 6. Our studies have focused on mechanisms to explain and potentially reverse phenotypic features in affected dogs. In vivo contraction force studies have documented preferential involvement of extensor muscles and associated debilitating flexor contractures. Preferential extensor muscle involvement may occur due to particularly deleterious effects of dystrophin deficiency on extensors during eccentric muscle contractions. Muscle ischemia, perhaps due, in part, to a compartment-like syndrome, could also be involved. Muscle blood flow studies are underway to address this hypothesis. Our force measurement studies have also shown that contraction and relaxation times of muscles of some dogs with GRMD are dramatically prolonged, due to a shift in the skeletal muscle resting membrane potential towards threshold. These changes in contraction kinetics may occur due to direct or indirect effects of dystrophin deficiency on membranal ion channels. We have also conducted studies to document whether transplantation of normal myogenic cells will restore function in dystrophic muscles. However, as has been shown in DMD patients, most injected cells are destroyed, presumably due to an immunologic reaction. More recently, in collaboration with investigators at other universities, we have initiated studies of dystrophin gene therapy in normal and dystrophic dogs, using adeno- and adeno-associated viruses. Contraction force generated by treated muscles can be monitored before and after treatment. Muscles are also studied pathologically and immunohistochemically.

Principal Investigators



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Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

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

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

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

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

Principal Investigators



M. Harold Laughlin, Chair of the Department of Biomedical Sciences

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

Research

I focus my research on cardiorespiratory effects of exercise. The primary goal is understanding of the effects of exercise training on the coronary circulation and skeletal muscle vascular beds. Exercise training produces increases in the capacity of myocardial and skeletal muscle vascular beds to transport oxygen and other nutrients. The training induced changes in vascular transport capacity are associated with growth of new capillaries, enlargement of 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.



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Appointment: Assistant 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

Principal Investigators

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.



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Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

There are two major research efforts in our laboratory:

One effort is devoted to determining how proteins located in cell membranes move molecules across the membrane. This includes elucidating some of the mechanisms of transport, determining how changes of the cytoplasmic milieu modulate transport, determining how changes in the rate of transport regulate cell function, and understanding the relationship between protein structure and transporter function. Our studies are currently focused on two transport systems: the plasma membrane calcium pump and the Na/K pump. Our recent work is devoted to examining the movement of the Na pump protein from plasma membrane to endosomes and back. We are determining the molecular basis for the integration of hormonal responses, e.g., what happens when one hormone signals increased endocytosis and another hormone signals exocytosis-who wins? We have also worked on zinc transporters, sodium/calcium exchanges, and anion exchangers. We use biophysical, bioengineering, biochemical, and optical approaches to studying the relevant physiology and pharmacology of these transport systems, including the development of a molecular mechanistic view of their function and an integrated view of how regulation of cellular transport related to cell, tissue, organ and organism function and pathophysiology.

Another effort is the development of sensors for molecules of biological interest in collaboration with Sheila Grant in Bioengineering and Josh Millspaugh in Fisheries and Wildlife. We are designing sensors that can be used in vivo for continuous monitoring and also sensors that can be used in the field for monitoring wildlife or at home using saliva, urine, or sweat for measurement of pet or human samples. We are currently developing approaches for the measurement of glucose, cortisol, aldosterone and sex steroids. Our techniques include absorbance, fluorescence, FRET and lanthanide luminescence.

Principal Investigators



Patrick Mueller

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Appointment: Adjunct Professor, Department of Biomedical Sciences

Research

The goal of my research is to learn more about how the brain controls the heart and blood vessels and therefore, its role in determining arterial blood pressure and organ blood flow. In particular, I am interested in how the brain adapts its control of the cardiovascular system to various physiological and pathophysiological states. Currently, research in the lab is focused on examining how levels of physical activity contribute to alterations in neurohumoral control of the circulation. A sedentary lifestyle is a major risk factor for cardiovascular disease, the leading cause of death in the United States. Nonetheless, rates of physical inactivity in the general population continue to increase and inactivity related diseases such as obesity, diabetes, and hypertension are burdening our health care system at an epidemic rate. Despite this important health care problem, the mechanisms by which a sedentary lifestyle contributes to cardiovascular disease are unknown. However, recent evidence suggests that overactivity the sympathetic nervous contributes to the development and maintenance of cardiovascular disease. Our hypothesis is that a sedentary lifestyle may result in overactivity of the sympathetic nervous system and contribute to the increased incidence of cardiovascular disease in inactive individuals. The goal of my current research is test the hypothesis that a sedentary lifestyle alters central nervous system mechanisms that result in overactivity of the sympathetic nervous system. Specifically, we will examine the structure and function of a specific population of neurons in the brain (i.e. spinally projecting rostral ventrolateral medulla neurons) that are critical for blood pressure regulation via generation of sympathetic nervous system activity. These neurons play important roles in physiological and pathophysiological disease processes and are likely to contribute to conditions involving overactivity of the sympathetic nervous system. The results of these experiments will provide novel mechanisms by which a sedentary lifestyle impacts cardiovascular health and well being as well as define potential therapeutic targets for the treatment and prevention of cardiovascular diseases.



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Education:
Ph.D. from the University of Houston, B.S. from Birla Institute of

Research

Research interests include neurohumoral control of reflex regulation of cardiovascular function.

Principal Investigators

Specifically, changes in cellular processes underlying synaptic communication between baroreceptor afferents and the nucleus tractus solitarius with the help of optical imaging techniques and electrophysiology. Other interests include modulation of reflex regulation of autonomic regulation of cardiovascular function by circulating estrogen and identifying the role of circumventricular organs such as area postrema and the subfornical organs and the estrogen receptors subtypes involved in mediating the effect of estrogen.



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Appointment: Assistant Professor, Department of Medical Pharmacology and Physiology

Research

- 1) The development of the electrical activity of the different regions of the heart.

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.

- 2) The role of cell adhesion molecules in the development of diabetes type II.

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.



Jeffrey T. Potts

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Appointment: Associate Professor, Department of Biomedical Sciences

Principal Investigators

Research

Research in our laboratory examines how individual neurons and interconnected populations of neurons encode information in the central nervous system, particularly as it relates to reflex control of the cardiovascular and respiratory systems. The goal of this research is to identify causal relationships between cellular/molecular processes and systems level function. To bridge these levels of analyses, we use a variety of techniques including electrophysiological (extracellular, intracellular and patch-clamp recording), neuroanatomical tracing and neurochemical studies in conjunction with *in vivo*, *in situ* and *in vitro* preparations to examine the connectivity of neurons in the pontomedullary axis.

Currently, we are investigating the cellular mechanisms underlying short-term changes in synaptic strength (ie. synaptic plasticity) and the consequence of these changes on function of the arterial baroreflex system. We have found that peripheral neurogenic feedback alters the excitability of barosensitive medullary neurons via local GABAergic circuits. These findings suggest that activation of intrinsic GABA neurons modulate neurotransmission in central baroreceptor circuits. This mechanism, in addition to other cellular and molecular events, may contribute to the induction of short-term changes synaptic plasticity in central baroreflex circuits. These findings have direct relevance to the regulation of cardiovascular function during both physiological (wake, sleep, exercise) and pathological (hypertension, congestive heart failure, diabetes) states, which will be the focus of our future endeavors.

In addition, we are interested in examining the neural pathways and synaptic mechanisms responsible for establishing basal breathing rhythms. Breathing is an unconscious behavior that we all perform and it is tightly coordinated with other motor behaviors such as speaking, eating and exercising. Our research has identified the synaptic connections between skeletal muscle and respiratory neurons in the brainstem that are responsible for changing breathing patterns. In particular, we have found that respiratory neurons in the pontine region of the brain play an important role in shaping breathing rhythms during exercise. By identifying these pathways, we are striving to learn more about sleep disordered breathing, such as sleep apnea and Sudden Infant Death Syndrome (SIDS), which are characterized by alterations in the normal processing of sensory signals. In the future, we will also incorporate transgenic mice to investigate the cellular and molecular basis underlying the generation of breathing rhythms.

The overall goal of research in our laboratory is to better understand the role of sensory feedback in the induction of neuroplasticity in pontomedullary neural circuits in a physiologically-relevant context and to determine whether short-term and long-term changes in synaptic strength participate in cardiorespiratory homeostasis.



Elmer M. Price

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Education:
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Appointment: Associate Professor, Department of Biomedical Sciences

Principal Investigators

Research

Research in the Price Lab involves study of the molecular, biochemical and cellular mechanisms underlying health and disease. We are specifically interested in two aspects of biomedicine, namely, cardiovascular disease and neurological disorders. Our research is based on the premise that an understanding of the mechanisms that lead to disease will help us discover new therapies based on modern molecular medicine.

Our cardiovascular program involves identification of the genes that play a role in preventing or reversing cardiovascular disease, with an emphasis on endothelial cell biology and endothelial nitric oxide synthase. It has been known for years that exercise is one of the key factors in preventing heart disease and our lab is interested in uncovering the genes that are responsible for the beneficial effect of exercise on the heart and circulatory system. Exercise is a model that can be used to identify factors (the products of exercise-regulated genes) that can be identified and harnessed to develop new therapeutic approaches. We are also exploring the role of adult stem cells in the maintenance of cardiovascular health and will use such cells to reverse disease using cell transplantation to grow new cardiac muscle or blood vessels.

Our neurobiology focus is centered around adult stem cells and their application in cell-based therapies. We have developed multipotent adult stem cell lines that appear to differentiate into several cell types, depending upon the chosen conditions. One novel aspect of these studies is that under select conditions these cells differentiate into apparent neural cells and this has led to studies involving stem cell transplantation into models of neurological disorders.

The Price Lab also collaborates extensively in the diverse areas of aging, neurohumoral control, and membrane transport. Our lab has enjoyed funding from the NIH, the Cystic Fibrosis Foundation, the American Heart Association, the University of Missouri Research Board, and the Missouri Spinal Cord Injury Research Program.



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Education:
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Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

Myocardial ischemia caused by inadequate blood flow to the heart muscle leads to purine loss from cells. The extent to which they are lost is directly related to recovery of mechanical function upon restoration of blood flow to the heart. Thus, decreased purine levels and decreased ability of the heart to perform useful work are coupled. I am interested in determining what controls rates of transport of purines into and out of cells and how this process can be altered to enhance the energy state of the heart.

Related to these studies are investigations of how the cardioprotective substance, fructose phosphate crosses heart cell membranes. These studies are done in collaboration with Dr. Christopher Hardin.

Principal Investigators

Membrane transport of glucose also is of interest and of naturally occurring and genetically-induced animal models that lead to diabetes and/or hypertension are used to understand the role of the renin-angiotensin system in the pathologic consequences of diabetes & hypertension. These studies are done in association with Drs. James Sowers and Craig Stump, department of medicine and the Truman V.A. hospital.

A model of hypothyroidism is used to alter the contractile activity of myocardial muscle in order to determine how specific changes in the contractile protein myosin affect the rate of force development by isolated muscle cells and intact heart. This is a collaborative research project with Dr. Kerry McDonald.

A similar investigation to that in hypothyroidism, but in hearts from genetically altered mice that exhibit traits of an inherited form of muscular dystrophy, are being carried out in collaboration with Drs. Joe Kornegy and Casey Childers college veterinary medicine and department of physical and rehabilitation medicine.



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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.

Principal Investigators



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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 2, 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.



Steven S. Segal

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Appointment: Professor, Department of Medical Pharmacology and Physiology

Research

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

Principal Investigators

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 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.



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Ph.D. and M.D. from Osaka Medical College

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, Ca^{2+} -activated, voltage-dependent potassium (BK_{Ca}) channel. BK_{Ca} 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 O_2 associated with hemeoxygenase-2 and, moreover, some splice-variants respond to membrane stretch. Based on these findings, BK_{Ca} 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 GABA_A receptor channels and GABA_B -mediated modulation of K^+ and Ca^{2+} 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.

Principal Investigators



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Appointment: Professor, Department of Medicine
Professor, Department of Medical Pharmacology and Physiology

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Appointment: 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 many-fold. 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

Principal Investigators



Xiaoqin Zou

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Education:
Ph.D from the University of California, San Diego, B.S. from Wuhan, University

Appointment: Assistant 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

**ACTIVE GRANTS and
CONTRACTS**

**PROFESSIONAL SERVICE
ACTIVITIES**

PUBLICATIONS

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Hancock, C.R.; Janssen, E. and Terjung, R.L.; "Contraction-Mediated Phosphorylation of AMPK is Lower in Skeletal Muscle of Adenylate Deficient Mice"; *J. Appl. Physiol.*, 100:406-413; 2006.

Carr, A.N.; Howard, B.W.; Davis, M.G.; Eby-Wilkens, E.; Loos, P.; Varbanov, A.; Qu, A.; Demuth, J.; A. Proia, A.; Yang, H.T.; Terjung, R.L. and Peters, K.G.; "Proangiogenic Effects of SDF1CXCR4 Signaling Pathway: Endothelial Expression of CXCR4 during Angiogenesis, Direct Angiogenic Effects of SDF-1, and Efficacy of Systemic Administration of SDF-1 in a Model of Vascular Insufficiency Support an Endothelium-Dependent Mechanism"; *Cardiovasc. Res.*, 69:925-935; 2006.

Allen, L.A.; Terjung, R.L. and Yang, H.T.; "Exogenous Fibroblast Growth Factor Increases Collateral Blood Flow In Female Rats with Femoral Artery Occlusion"; *J. Cardiovasc Pharmacol* 47:146-154; 2006.

Hancock, C.R.; Brault, J.J.; Meyer, R.A. and Terjung, R.L.; "Protecting the Energy State In Muscle During Contractions: Role of AMP Deaminase"; *J. Physiol. Pharmacol.*, 57:(Suppl 10) 17-29; 2006.

Cruze, C.A.; Su, F.; Limberg, B.J.; Deutsch, A.J.; Stoffolano, P.J.; Daia, J.; Buchanan, D.D.; Yang, H.T.; Terjung, R.L.; Spruella, R.D.; Mittelstadt, S.W. and Rosenbaum, J.S.; "The Y2 Receptor Mediates Increases in Collateral-Dependent Blood Flow in a Model of Peripheral Arterial Insufficiency"; *Peptides*, 28:000-000; 2007

Terjung, R.L., Prior, B.M. and Yang, H.T.; "Exercise and Vascular Remodeling in Health and Disease. In: Promotion and Health and Welfare in the Aging Society. International Sports Science Network Forum"; Volume 3. H. Nose (Ed), Cooper Pub., Traverse City, MI; 2006

Walker, N.M.

Walker, N.M.; Boyle, K.T.; Simpson, J.E. and Clarke, L.L., "Talinflumate (LoMucin) is an Inhibitor of Intestinal Cl/HCO₃ Exchange and Increases the Survival of Cystic Fibrosis Mice"; *J. Pharmacol. Exp. Therap.*, DOI: 10.1124/jpet. 105.094847; 2005.

PUBLICATIONS

Wang, X.

Zhou, Z.; Wang, X.; Li, M.; Soma, Y.; Zou, X. and Hwang, T.C., "High Affinity ATP/ADP Analogs as New Tools to Study CFTR Gating"; *Journal of Physiology*, 569, 447-57; December 2005.

Zhou, Z.

Zhou, Z.; Wang, X.; Li, M.; Soma, Y.; Zou, X. and Hwang, T.C.; "High Affinity ATP/ADP Analogs as New Tools to Study CFTR Gating"; *Journal of Physiology*, 569, 447-57; December 2005.

Zou, X.

Zhou, Z.; Wang, X.; Li, M.; Soma, Y.; Zou, X. and Hwang, T.C.; "High Affinity ATP/ADP Analogs as New Tools to Study CFTR Gating"; *Journal of Physiology*, 569, 447-57; December 2005.

Liu, H.-Y. and Zou, X.; "Electrostatics of Ligand Binding: Parameterization of the Generalized Born Model and Comparison With Poisson-Boltzmann Approaches"; *Journal of Physical Chemistry B*, 110, 9304-13; 2006.

Huang, S.-Y., and Zou, X.; "An Iterative Knowledge-based Scoring Function to Predict Protein-ligand Interactions: I. Derivation of the Interaction Potentials"; *Journal of Computational Chemistry*, 27, 1866-75; 2006.

Huang, S.-Y. and Zou, X.; "An Iterative Knowledge-based Scoring Function to Predict Protein-ligand Interactions: II. Validation of the scoring function"; *Journal of Computational Chemistry*, 27, 1876-82; 2006.

PRESENTATIONS

Bogan, J.

Lecturer, “Clinical and sanitary handling of GRMD dogs”, University of Sao Paulo-College of Veterinary Medicine, Sao Paulo, Brazil SP. (February 2006)

Bogan, J.

Lecturer, “Animal Models of DMD”, University of Sao Paulo-College of Veterinary Medicine, Sao Paulo, Brazil, SP. (February 2006)

Booth, F.W.

“Fundamental Question of Biology: How and Why does the Body Adapt to Physical Inactivity”; Adolph Lecture for American Physiological Society Meeting at Experimental Biology, San Francisco, CA.

Booth, F.W.

“Impaired Regrowth in Old Skeletal Muscle”; American Physiological Society meeting at Experimental Biology, San Francisco, CA.

Booth, F.W.

“Evidence for abnormalities of gene expression attributable to mismatching of biological heritage and lifestyle”; ACSM Annual Meeting, Denver, CO.

Booth, F.W.

“Impaired Hypertrophy of Slowly Atrophying Skeletal Muscle in Old Animals”; ACSM Annual Meeting, Denver, CO.

Booth, F.W.

Keynote speaker, “How and Why does the Body Adapt to Physical Inactivity?”; European Conference of Sports Sciences, Lausanne, Switzerland.

Booth, F.W.

“The Process of Change: *Paradigm Shifts* in Physical Activity Studies”; International Conference on the Biochemistry of Exercise. Seoul, South Korea.

Booth, F.W.

“How and Why does the Body Adapt to Physical Inactivity?”; USC Dept Preventive Medicine Seminar, Los Angeles, CA.

Booth, F.W.

“Metabolic Responses to Reductions in Physical Activity from Ambulatory Levels”; Center for Immunology and Muscle, Copenhagen, Denmark.

Benakanakere, I.N.

“Increased VEGF and Blood Vessel Density are Associated with Progestin-accelerated Mammary Tumors in DMBA Treated Sprague-dawley Rats”; 88th Annual Endocrine Society Meeting, Boston, MA. (June 2006)

Benakanakere, I.N.

“Natural and Synthetic Progestins Accelerate Tumor Development and Increased VEGF production in a Chemically Induced Mammary Tumor Model: A Potentially Novel Target for Anti-angiogenic Therapeutics”; AACR Special Conference in Advances in Breast Cancer, San Diego, CA, Abstract A15. (2005)

PRESENTATIONS

Childers, M.K.

“Translational Research in Duchenne Muscular Dystrophy”; Grand Rounds, Department of Family & Community Medicine, University of Missouri, Columbia, MO. (February 1, 2006)

Childers, M.K.

“Translational Research in Duchenne Muscular Dystrophy”; Grand Round, Department of Neurology, Wake Forest University, Winston-Salem, NC. (February 13, 2006)

Childers, M.K.

“Translational Research in Duchenne Muscular Dystrophy”; Grand Rounds, Department of PM&R, The Ohio State University, Columbus, OH. (February 6, 2006)

Clarke, L.

Poster, “CFTR Facilitates PAT-1 (Slc26a6) Cl/HCO₃ Exchanger Activity in Intact Epithelium from Murine Duodenum”; 19th Annual North American Cystic Fibrosis Conference, Baltimore, MD. (October 21, 2005)

Clarke, L.

Poster, “Inhibition of the Putative Anion Transporter (PAT-1, Slc26a6) Impairs the Ability of Vilous Epithelial Cells to Regulate intracellular pH (pH_i) During Peptide Transport”; Experimental Biology Meeting, San Francisco, CA. (April 2006)

Clarke, L.

Oral Workshop Presentation, “A Major Contribution of Cl/HCO₃⁻ Exchange Activity to cAMP-stimulated HCO₃⁻ secretion Across the Duodenum is Revealed by the DRA (Slc26a3) Knockout Mouse”; 19th Annual North American Cystic Fibrosis Conference, Baltimore, MD. (October 21, 2005)

Clarke, L.

Oral Symposium Presentation, “CFTR and Anion Exchangers of the Murine Duodenum”; 19th Annual North American Cystic Fibrosis Conference, Baltimore, MD. (October 21, 2005)

Clarke, L.

Oral Workshop, “Perspectives on the Cystic Fibrosis Mouse Model”; Strategic Planning Meeting of Cystic Fibrosis Foundation, Bethesda, MD. (January 11, 2006)

Clarke, L.

Oral Workshop, “Intestinal Disease and Electrolyte Transport in the Cystic Fibrosis Mouse”; 5th Annual McGill Workshop on Bioinformatics: Endoplasmic Reticulum Trafficking Diseases, Hometown, Barbados. (January 25, 2006)

Clarke, L.

“Interactions of CFTR and Anion Exchangers in the Murine Duodenum”; Frontiers in Epithelial Transport, Focused Meeting of The Physiological Society, Manchester, U.K. (April 7, 2006)

Clarke, L.

“Down-regulated in adenoma (DRA, Slc26a3) Cl-/HCO₃⁻ exchanger is a Major Contributor to Basal and cAMP-stimulated HCO₃⁻ secretion Across Murine Duodenum”; 107th Annual Meeting

PRESENTATIONS

of the American Gastroenterological Association Institute, Los Angeles, CA. (May 21, 2006)

Clarke, L.

Poster of Distinction, “Down-regulated in Adenoma (DRA, Slc26a3) is the predominant $\text{Cl}^-/\text{HCO}_3^-$ exchanger in the Lower Villous Epithelium of Murine Duodenum”; 107th Annual Meeting of the American Gastroenterological Association Institute, Los Angeles, CA. (May 21, 2006)

Clarke, L.

“Inhibition of the Putative Anion Transporter (PAT-1, Slc26a6) Impairs the Ability of Upper Villous Epithelial Cells to Regulate intracellular pH during H^+ -dipeptide transport”; 107th Annual Meeting of the American Gastroenterological Association Institute, Los Angeles, CA. (May 22, 2006)

Clarke, L.

“Intestinal Barrier Repair is Impaired Following Ischemic Injury in NHE2-deficient Mice”; 107th Annual Meeting of the American Gastroenterological Association Institute, Los Angeles, CA. (May 24, 2006)

Davis, M.J.

“Regulation of Calcium Channels by Integrins and Extracellular Matrix”; Department of Cell & Molecular Physiology, University of North Carolina, Chapel Hill, NC. (February 2006)

Davis, M.J.

“Novel Mechanisms in Myogenic Control of Lymphatic Diameter”; University of Calgary. (May 2006)

Foley, C.M.

“Alterations in Reflex Control of Sympathetic Nerve Activity Following Cardiovascular Deconditioning”; Medtronic. (February 2006)

Foley, C.M. and Price, E.

“Neurogenic Peripheral Blood-Derived Multipotent Adult Progenitor Cells”; Stem Cells: Research and Therapeutics, San Francisco, CA. (March 2-3, 2006)

Foley, C.M.

“Altered Arterial Baroreflex Control of Sympathetic Nerve Activity in an Animal Model of Microgravity and Bedrest”; Colorado State University. (May 25, 2006)

Foley, C.M.

“Alterations in Control of the Cardiovascular System Following Deconditioning”; Abbott. (May 12, 2006)

Foley, C.M.

“Altered Arterial Baroreflex Control of Sympathetic Nerve Activity in an Animal Model of Microgravity and Bedrest”; CVRx. (May 15, 2006)

Gillis, K.D.

“Studies of Exocytosis from Chromaffin Cells using Microchip Electrochemistry and Scanning Ion Conductance Microscopy”; 13th International Conference of Chromaffin Cell Biology, Pucon, Chile. (January, 2006)

PRESENTATIONS

Gillis, K.D.

“Phosphomimetic Mutations of Ser187 of SNAP-25 Increase a Highly Ca²⁺ Sensitive Pool of Vesicles and Promote Asynchronous Exocytosis”; 13th International Conference of Chromaffin Cell Biology, Pucon, Chile. (January, 2006)

Gu, L.

“Single Molecule Detection in a Nanaopore-from RNAi to Anthrax”; Bond Life Science Center, Columbia, MO. (June 30, 2006)

Hamilton, M. T.

“Inactivity Physiology and Non-Exercise Activity Thermogenesis (NEAT): Clinical and Molecular Insights” (& Overview for the session); National Meeting for American College of Sports Medicine, Denver, CO. (June, 2006)

Hamilton, M. T.

“Unique Molecular and Physiological Responses to Inactivity Leading to Metabolic Disorders”; National Meeting for American College of Sports Medicine, Denver, CO. (June, 2006)

Hamilton, M. T.

“Human and Rat Skeletal Muscle Expresses a Potentially Anti-Inflammatory-and Anti-Thrombotic Gene (LPP1/PAP2a) Rapidly Suppressed by Physical Inactivity”; Theodore Zderic and Marc T. Hamilton. Phi Zeta Research Day Poster. (March 2006)

Hamilton, M. T.

“Relationship Between Skeletal Muscle LPL Activity and Ability of Endothelial Cells to Bind LPL” Perminder Gulani and Marc T. Hamilton. Phi Zeta Research Day Poster. (March 2006)

Hamilton, M. T.

“Human and Rat Skeletal Muscle Expresses a Potentially Anti-Inflammatory-and Anti-Thrombotic Gene (LPP1/PAP2a) Rapidly Suppressed by Physical Inactivity”; Theodore Zderic and Marc T. Hamilton. Missouri Life Sciences Week. (April 2006)

Hamilton, M. T.

“Skeletal Muscle LPL Activity and Endothelial Cells Bind LPL”; Perminder Gulani and Marc T. Hamilton. Missouri Life Sciences Week. (April 2006)

Hamilton, M. T.

“Human and Rat Skeletal Muscle Expresses a Potentially Anti-Inflammatory-and Anti-Thrombotic Gene (LPP1/PAP2a) Rapidly Suppressed by Physical Inactivity”; Theodore Zderic and Marc T. Hamilton. Cardiovascular Day. (February 2006)

Hill, M.A.

“Mechanisms of Myogenic Tone”; 11th International Vascular Neuroeffector Mechanisms Symposium, Suzhou, China. (June 26-29, 2006)

Hill, M.A.

“The Microvasculature as a Therapeutic Target in Diabetes; Possible Influence of Vascular Heterogeneity”; World Congress of Pharmacology, Beijing, China. (July 2-7, 2006)

Huang, S.

PRESENTATIONS

“A Fast Ligand Algorithm to Incorporate Receptor Flexibility Using Multiple Protein Structures”; Biophysical Society Meeting, Salt Lake City, UT. (February 2006)

Huxley, V.H.

Workshop, “Sex and Gender Differences in Obesity & Cardiovascular Disease”; Society for Women's Health, Washington, DC. (November 2-4, 2005)

Huxley, V.H.

"Microvascular Exchange: How Up to Date is What We Learn from the Text Books?"; Chulalongkorn University Department of Physiology Seminar, Bangkok, Thailand. (December 9, 2005)

Huxley, V.H.

“Just as $(7-3)$ squared and $(5+3)*2$, both = 16: Males and Females Attain Volume Homeostasis by Different Means”; Chulalongkorn University Medical School, Bangkok, Thailand. (December 14, 2005)

Huxley, V.H.

“Microcirculation: a Perfect Field for Bioengineering”; Asian Union for Microcirculation, Thai Society of Microcirculation and Thai Atherosclerosis Society Plenary Lecture, Bangkok Thailand. (December 16, 2005)

Huxley, V.H.

Workshop, “The Application of Confocal Laser Microscopy”; Asian Union for Microcirculation, Thai Society of Microcirculation and Thai Atherosclerosis Society, Bangkok Thailand, (December 16, 2005)

Huxley, V.H.

Co-chair & speaker, “Cardiovascular System: Gender Differences in Normal Function and Disease” American Physiological Society Refresher Course on Gender Differences in Physiology: Session. EB, San Francisco, CA. (April 1, 2006)

Hyder, S.M.

“Medroxyprogesterone Acetate Accelerates Tumor Development and Increases Tumor Multiplicity in DMBA Treated Sprague-Dawley Rats”; 87th Annual Endocrine Society Meeting, San Diego, CA, P2-648. (2005)

Hyder, S.M.

“Progesterin-dependent induction of VEGF is dependent on functional PR, SP-1 transcription factor and SP-1 recognition sites on the VEGF promoter in human breast cancer cells”; 87th Annual Endocrine Society Meeting, San Diego, CA P3-281. (2005)

Hyder, S.M.

“Progesterin-induced VEGF from human breast cancer cells increases proliferation of endothelial and tumor epithelial cells via paracrine and autocrine mechanisms”; 87th Annual Endocrine Society Meeting, San Diego, CA P2-646. (2005)

Hyder, S.M.

“Identification and Transcriptional Activity Characterization of Two Novel Splicing Isoforms of

PRESENTATIONS

Human ERR-beta”; 87th Annual Endocrine Society Meeting, San Diego, CA Abst. P3-262. (2005)

Hyder, S.M.

“Natural and Synthetic Progestins Accelerate Tumor Development and Increased VEGF Production in a Chemically Induced Mammary Tumor Model: A Potentially Novel Target for Anti-angiogenic Therapeutics”; AACR Special Conference in Advances in Breast Cancer, San Diego, CA Abstract A15. (2005)

Hyder, S.M.

“Increased VEGF and Blood Vessel Density are Associated with Progestin-Accelerated Mammary Tumors in DMBA Treated Sprague-Dawley Rats”; 88th Annual Endocrine Society Meeting, Boston, MA. OR 7-1 (2006)

Hyder, S.M.

“Suppression of Breast Tumor Growth by Reactivation of p53 Pathway in Cancer Cells”; 88th Annual Endocrine Society Meeting, Boston, MA. P1-148 (2006)

Hyder, S.M.

“Re-activation of Mutant p53 by PRIMA-1 suppresses Breast Tumor Growth”; 97th Annual American Association of Cancer Research Meeting, Washington, DC. Abst 2215 (2006)

Hyder, S.M.

“VEGF is a Proliferative and a Survival Factor for Breast Cancer Cells Expressing VEGFR2 (flk/kdr): Implications for Anti-hormone Resistance”; 88th Annual Endocrine Society Meeting, Boston, MA. P3-23 (2006)

Hyder, S.M.

“VEGF Induces Proliferation of Breast Cancer Cells via VEGFR2 (flk/kdr) and Inhibits the Growth Suppressive Effects of Anti-hormones”; 97th Annual American Association of Cancer Research Meeting, Washington DC. Abst 3650 (2006)

Hwang, C.

“Cooperative Gating of CFTR Channels Revealed by High Affinity ATP/ADP Analogs”; Biophysical Meeting, Salt Lake City, UT. (February 2006)

Kornegay, J.N.

Lecturer, “A Practical Approach to Lesion Localization and the Neurologic Examination”, Inland Empire VMA 13th Annual Ski Meeting, Schweitzer Mountain Resort, ID. (February 2006)

Kornegay, J.N.

Lecturer, “A Canine Model of Duchenne Muscular Dystrophy”; University of North Carolina Gene Therapy Center, Chapel Hill, N. C. (March 2006)

Li, M.

“Cooperative Gating of CFTR Channels Revealed by High Affinity ATP/ADP Analogs”; Biophysical Meeting, Salt Lake City, UT. (February 2006)

Liang, Y.

“Suppression of Breast Tumor Growth by Reactivation of p53 Pathway in Cancer Cells”; 88th Annual Endocrine Society Meeting, Boston, MA. (June 2006)

PRESENTATIONS

Liang, Y..

“Re-activation of Mutant p53 by PRIMA-1 Suppresses Breast Tumor Growth”; 97th Annual American Association of Cancer Research Meeting, Washington, DC. (April 2006)

Liang, Y.

“VEGF Induces Proliferation of Breast Cancer Cells via VEGFR2 (flk/kdr) and Inhibits the Growth Suppressive Effects of Anti-hormones”; 97th Annual American Association of Cancer Research Meeting, Washington DC. (April 2006)

Liang, Y.

“VEGF is a Proliferative and a Survival Factor for Breast Cancer Cells Expressing VEGFR2 (flk/kdr): Implications for Anti-hormone Resistance”; 88th Annual Endocrine Society Meeting, Boston, MA. (June 2006)

Liang, Y.

“Progesterin-induced VEGF from Human Breast Cancer Cells Increases Proliferation of Endothelial and Tumor Epithelial Cells via Paracrine and Autocrine Mechanisms”, 87th Annual Endocrine Society Meeting, San Diego, CA P2-646. (2005)

Liu, H.

“Development of a Generalized Born model: Parameterization for Electrostatics of Ligand Binding”; Biophysical Society Meeting, Salt Lake City, UT. (February 2006)

Martinez-Lemus, L..

“Mechanisms of Myogenic Tone”; 11th International Vascular Neuroeffector Mechanisms Symposium, Suzhou, China. (June 26-29, 2006)

Meininger, G.A.

“Studying How Cells Respond to Mechanical Forces: A Full Contact Sport” or “Knock, Knock-Who’s there?”; Dalton Science Teachers Symposium, Dalton Cardiovascular Research Symposium, Columbia, MO. (November 2005)

Meininger, G.A.

“Vascular Mechanobiology is a Contact Sport: Studies using Atomic Force Microscopy”; Department of Physiology, Medical College of Georgia. (December 2005)

Meininger, G.A.

Lecturer, “Mechanical Characteristics of Integrin-extracellular Matrix Interaction Studied with Atomic Force Microscopy”; VIII Annual Linz Winter Workshop Symposium, Advances in Single Molecule Research for Biology and Nanoscience, Linz, Austria. (February 2006)

Meininger, G.A.

Lecturer, “Using Atomic Force Microscopy as a Tool in Biological Research: From Cell to Molecule”; Nebraska Research Exposition and Innovation Forum, Lincoln, NE. (March 2006)

Meininger, G.A.

Lecturer, “Mechanical Characteristics of Extracellular Matrix Integrin Interactions Studied Using Atomic Force Microscopy”; Seminar to the Physiology Institute, Ludwig-Maximilians University, Munich, Germany. (February 2006)

PRESENTATIONS

Meininger, G.A.

Lecturer, "Integrins and Mechotransduction in Vascular Smooth Muscle: A Sticky Topic"; Seminar to the Department of Integrative and Cellular Physiology, Indiana University School of Medicine. (February 2006)

Milanick, M.

"Clinical Biodetectives"; MU Family Medicine Research Seminar Series. (November 2005)

Milanick, M.

"NaK Pump and Nanopump"; Seminar, Emory University. (June 2006)

Mueller, J.

Lecturer, "Neural Control of the Circulation"; Graduate Course, Biomedical Sciences-VCSCI 9467; two lectures and three paper discussions. (January-March 2006)

Mueller, J.

"Blunted Centrally Mediated Sympathoexcitation in Exercise Trained Rats"; Featured Topic: "Activity-Dependent Plasticity in Central Homeostatic Systems" Experimental Biology Meeting, San Francisco, CA. (April 2006)

Mueller, J.

"Physical Activity Dependent Plasticity in Neural Control of the Circulation: The Anti-Hypertensive Brain?" Department of Physiology, Medical College of Georgia, Augusta, GA. (March, 2006).

Polo-Parada, L.

"The Role of the Cardiac Cushions in the Development of the Cardiac Action Potential"; Weinstein Cardiovascular Development Conference, St. Petersburg, Florida. (May 11-13, 2006)

Polo-Parada, L.

"Insulin Induces a Desensitization of Insulin and IGF-1 Signaling in INS1-E beta-cells: Mechanisms and Consequences on Function and Survival"; EB2006, San Francisco, CA. (April 1-5, 2006)

Potts, J.T.

"Cardiovascular Integration and Exercise: Role of GABAergic and SPergic Circuits in NTS"; Department of Physiology and Biophysics, Case Western Reserve University, Cleveland, OH. (July, 2005)

Potts, J.T.

"Synaptic Plasticity in Autonomic Neural Circuits – Role in Cardiorespiratory Homeostasis"; Department of Medical Pharmacology and Physiology, University of Missouri, Columbia, MO.

Potts, J.T.

"Neural Plasticity in Autonomic Circuits: Lessons Learned from the NTS"; Iowa Cardiovascular Center, University of Iowa, Iowa City, IA. (March 2006)

Potts, J.T.

"Nucleus Tractus Solitarii – So Much More than a Simple Sensory Relay"; Department of Cellular and Integrative Physiology, University of Nebraska Medical Center, Omaha, NE. (May 2006)

PRESENTATIONS

Price, E.

“Development of Molecular or Cellular Therapeutics”; Pfizer Inc., (September 14, 2005)

Price, E.

“Neurogenic Peripheral Blood-Derived Multipotent Adult Progenitor Cells”; Stem Cells: Research and Therapeutics, San Francisco, CA. (March 2-3, 2006)

Price, E.

Poster: “Neurogenic Peripheral Blood Derived Adult Stem Cells”; EB2006, San Francisco, CA. (April 1-5, 2006)

Price, E.

Poster: “The role of NOS in Exercise-Induced Changes in EPC Number”; EB2006, San Francisco, CA. (April 1-5, 2006)

Price, E.

Poster: “Effects of Chronic NOS Inhibition on Responses to Acute Exercise”; EB2006, San Francisco, CA. (April 1-5, 2006)

Price, E.

Lecturer, “Angiogenic Peripheral Blood Derived Adult Stem Cells”; EB2006, San Francisco, CA. (April 1-5, 2006)

Schadt, J.C.

“The Neurohumoral and Hemodynamic Response to Blood Loss: What’s Important and What’s Not?”; Departments of Pediatrics and Physiology, New York Medical College, Valhalla, New York. (April 10, 2006)

Schadt, J.C.

“The Neurohumoral and Hemodynamic Response to Blood Loss: What’s Important and What’s Not?”; War Related Injury and Illness Study Center, VA Medical Center, East Orange, New Jersey. (April 11, 2006)

Simpson, J.E.

Poster, “CFTR Facilitates PAT-1 (Slc26a6) Cl/HCO³ Exchanger Activity in Intact Epithelium from Murine Duodenum”; 19th Annual North American Cystic Fibrosis Conference, Baltimore, MD. (October 21, 2005)

Soma, Y.

“Cooperative Gating of CFTR Channels Revealed by High Affinity ATP/ADP Analogs”; Biophysical Meeting, Salt Lake City, UT. (February 2006)

Soma, Y.

“Two-dimensional Dwell-time Analysis for Mg²⁺-induced Enhancement Effect in a Native BK Channel in Cultured Human Renal Proximal Tubule Cells.”; Biophysical Meeting. Salt Lake City, UT. (February 2006)

PRESENTATIONS

Soma, Y.

“Implication of Plasma Membrane Raft in Modulation of CFTR Channel Function”; Physiological Society Meeting of Japan. Maebashi, Gunma, Japan. (March, 2006)

Sun, Z.

“Mechanisms of Myogenic Tone”; 11th International Vascular Neuroeffector Mechanisms Symposium, Suzhou, China. (June 26-29, 2006)

Terjung, R.L.

“Vascular Remodeling Induced by Exercise: Functional Significance & Health Implications”; Texas A & M. (February 1, 2006)

Terjung, R.L.

“Collateral Vessel Remodeling: Exercise, Growth Factors, and Nitric Oxide. Congress of the Polish Physiological Society, Warsaw, Poland”; (September 15, 2006)

Terjung, R.L.

“Factors Influencing Collateral Vessel Development in Experimental Peripheral Arterial Insufficiency. Boehringer-Ingelheim Lecture”; Center for Cardiovascular Research. University of Medicine, Berlin, Germany. (October 25, 2006)

Walker, N.

Oral Workshop Presentation, “A Major Contribution of Cl/HCO₃ Exchange Activity to Camp”; 19th Annual North American Cystic Fibrosis Conference, Baltimore, MD. (October 21, 2005)

Zhou, Z.

“Cooperative Gating of CFTR Channels Revealed by High Affinity ATP/ADP Analogs”; Biophysical Meeting, Salt Lake City, UT. (February 2006)

Zou, X.

“ITScore: An Iterative Knowledge-based Scoring Function for Binding Predictions on Ligand-protein Interactions”; Gordon Conference, Tilton, NH. (August 2005)

Zou, X.

“Development of a Generalized Born Model: Parameterization for Electrostatics of Ligand Binding”; Biophysical Society Meeting, Salt Lake City, UT. (February 2006)

Zou, X.

“A Fast Ligand Algorithm to Incorporate Receptor Flexibility Using Multiple Protein Structures”; Biophysical Society Meeting, Salt Lake City, UT. (February 2006)

Zou, X.

“Cooperative Gating of CFTR Channels Revealed by High Affinity ATP/ADP Analogs”; Biophysical Meeting, Salt Lake City, UT. (February 2006)

ACTIVE GRANTS & CONTRACTS

PRINCIPAL INVESTIGATOR <i>TITLE</i>	AGENCY	AMOUNT PERIOD
Bompadre, Silvia G. <i>Physiology and Pharmacology of G5551 Mutant CFTR</i>	Cystic Fibrosis Foundation	\$175,375 4/06-3/08
Childers, Martin K. <i>Leupeptin in a Canine Model of DMD</i>	NIH National Institute of Health	\$335,944 6/05-5/07
Childers, Martin K. <i>Use of a Novel Calpain Inhibitor, C101, in Canine MD</i>	Ceptor Corporation	\$200,000 10/04-6/06
Clarke, Lane L. <i>CFTR and Acid-Base Transporters of the Intestinal Epithelia</i>	Cystic Fibrosis Foundation	\$100,000 10/05-9/06
Clarke, Lane L. <i>Slc26a Exchangers in the Cystic Fibrosis Intestine</i>	Cystic Fibrosis Foundation	\$194,400 4/05-3/07
Clarke, Lane L. <i>CFTR and Duodenal Acid-Base Transport</i>	NIH National Institute of Health	\$870,000 12/00-11/05
Foley, Charles M. <i>GABA Transmission, Gender and Hindlimb Unloading</i>	National Aeronautics and Space Administration	\$692,668 3/04-9/06
Gillis, Kevin D. <i>Microchip Devices to Assay Quantal Exocytosis</i>	NIH National Institute of Health	\$1,618,659 9/04-7/07
Hamilton, Marc T. <i>Transcriptional and Metabolic Profiling During Chronic Paralysis (SCI) and Exercise</i>	Spinal Cord Injury Research Program	\$100,000 9/04-10/06

ACTIVE GRANTS & CONTRACTS

PRINCIPAL INVESTIGATOR <i>TITLE</i>	AGENCY	AMOUNT PERIOD
Hamilton, Marc T. <i>Transcriptional Regulation in Skeletal Muscle</i>	College of Veterinary Medicine	\$18,000 3/05-2/06
Hamilton, Marc T. <i>Lipoprotein Lipase Regulation of Arterial Endothelium During Exercise and Atherosclerosis</i>	NIH National Institute of Health	\$1,248,342 1/06-12/11
Hasser, Eileen M. <i>Cardiovascular Regulation in Hindlimb Unweighted Rats</i>	NIH National Heart Lung and Blood Institute	\$1,303,201 4/02-3/07
Heesch, Cheryl M. <i>Gender Related Differences in Central Autonomic Control Following Simulated Microgravity in Rats</i>	National Aeronautics and Space Administration	\$953,039 12/02-11/6
Heesch, Cheryl M. <i>Neural Circulatory Control: Pregnancy & Ovarian Hormones</i>	NIH National Heart Lung and Blood Institute	\$880,807 3/04-2/08
Hwang, Tzyh C. <i>Confocal Equipment for Biomedical and Nanomedicine Research</i>	NIH National Institute of Health	\$402,030 4/06-4/08
Hwang, Tzyh C. <i>Gating of CFTR CL Channels by ATP Hydrolysis</i>	Cystic Fibrosis Foundation	\$75,000 7/05-6/06
Hwang, Tzyh C. <i>Molecular Pathophysiology of Cystic Fibrosis</i>	NIH National Institute of Health	\$1,015,447 9/03-8/07
Hyder, Salman M. <i>Activation of Nonfunctional p53 Protein in Breast Cancer</i>	US Army Medical Research and Materiel CMD	\$110,250 7/05-7/06

ACTIVE GRANTS & CONTRACTS

PRINCIPAL INVESTIGATOR <i>TITLE</i>	AGENCY	AMOUNT PERIOD
Hyder, Salman M. <i>Estrogen and Progesterone Regulation of Thrombospondin-1</i>	Susan G. Komen Breast Cancer Foundation	\$250,000 5/06-4/08
Hyder, Salman M. <i>Progesterone Regulation of VEGF in Human Breast Cancer Cell</i>	NIH National Cancer Institute	\$1,043,099 8/02-3/07
Hyder, Salman N. <i>P53-activating Compounds as Therapeutic Tools for Progression</i>	Susan G. Komen Breast Cancer Foundation	\$135,000 5/06-4/09
Kline II, David D. <i>Serotonergic Modulation of Synaptic Transmission</i>	American Heart Association	\$54,391 12/05-6/07
Kornegay, Joe N. <i>Investigative Therapeutics in a Canine Model of DMD</i>	Duchenne Muscular Dystrophy Center	\$264,946 1/04-7/06
Kornegay, Joe N. <i>MDCRC Supplement Canine Colonies</i>	University of Pittsburgh	\$86,216 6/05-5/07
Kornegay, Joe N. <i>Preclinical Gene Therapy in a Large Animal Model of DMD</i>	University of Pittsburgh	\$567,093 9/03-5/07
Kornegay, Joe N. <i>Preclinical Gene Therapy in a Large Animal Model of DMD</i>	University of Pittsburgh	\$133,914 1/04-12/08
Martinez-Lemus, Luis A. <i>Remodeling of the Resistance Microvasculature</i>	American Heart Association	\$227,643 2/06-12/08

ACTIVE GRANTS & CONTRACTS

PRINCIPAL INVESTIGATOR TITLE	AGENCY	AMOUNT PERIOD
Meininger, Gerald A. <i>Microvascular Control: A Role for Intergrins</i>	NIH National Institute of Health	\$698,922 2/06-11/07
Meininger, Gerald A. <i>Microvascular Control: A Role for Intergrins</i>	NIH National Institute of Health	\$698,922 2/06-11/07
Milanick, Mark <i>Cytosolic Modulation of Plasma Membrane Ion Transport</i>	NIH National Institute of Health	\$867,135 8/03-5/08
Milanick, Mark <i>From Clinic to Bench & Back: Clinical Biodetective Training</i>	NIH National Institute of Health	\$419,035 9/04-7/07
Mueller, Patrick J. <i>Central Control of Sympathetic Outflow Following Exercise</i>	American Heart Association	\$143,000 1/06-12/07
Pamidimukkala, Jayabala <i>Frequency Dependent Depression of Exocytosis</i>	American Heart Association	\$143,000 1/04-12/06
Polo Parada, L. <i>Role of the Cardiac Cushions</i>	American Heart Association	\$220,245 2/06-1/09
Potts, Jeffrey T. <i>Central Interaction of Baroreceptor and Somatic Afferent</i>	NIH National Institute of Health	\$560,924 8/04-5/07
Zhou, Zhen <i>Molecular Physiology of CFTR Gating</i>	Cystic Fibrosis Foundation	\$86,400 4/06-3/08

ACTIVE GRANTS & CONTRACTS

Zou, Xiaoqin

Quantitative Structure & Function of ABC Transporters
NIH National Institute of Diabetes and Digestive

\$572,877

6/02-5/08

PROFESSIONAL SERVICE ACTIVITIES

Blaine, E. H.

University of Missouri Research Board; grant review and awards
University of Missouri Long-Term Planning Committee
University of Missouri Faculty Grievance Committee
University of Missouri, Dalton Cardiovascular Research Center, Advisory Committee
University of Missouri, Department of Medical Pharmacology and Physiology, Graduate Education Committee
University of Houston, External examiner for faculty tenure appointment
Manuscript reviews for American Journal of Physiology and Cardiovascular

Booth, F.W.

Editorial Board, *Journal of Applied Physiology*, 1980; 1993; 1996-1999; 2005-present
Editorial Board, *American Journal of Physiology: Cell Physiology*, 1994- present
Editorial Board, *Physiological Genomics*, 2005-
Editorial Board, *Health and Physical Fitness*, 2004-present
Senior Editor, *CardioMetabolic Syndrome*, 2006
ACSM Inactivity Policy Meeting, Washington DC
ACSM Steering on Public Policy for Physical Activity/Inactivity
University of Missouri Wellness Committee

Bowles, D.

Biomedical Sciences Promotion and Tenure Advisory Committee
Biomedical Sciences Faculty Mentoring Committee (Dr. Kovarik)
CVM Administrators Evaluation Committee
CVM Graduate/Resident Training Committee
Research Mentor, Comparative Medicine Training Program
Chair, CVM Visiting Lecturers Committee
Trustee and Chair, Health Activity Center M. Harold Laughlin Scholarship Fund
Member, NIH Special Emphasis Panel/Scientific Review ZRG1 MDCN-G
Member, NIH Special Emphasis Panel ZRG1 CVS-A
Member, NIH Special Emphasis Panel HM-D

Childers, M. K.

Course Instructor, "Mice are not men and dogs are not mice", Association of Academic Physiologists, Daytona Beach, FL, March 1-4, 2006

Clarke, L.

Director of Graduate Studies, Biomedical Sciences Area
Research Mentor, Comparative Medicine Training Program
Member, University of Missouri Research Advisory Committee
Chair, CVM Search Committee for Associate Dean of Research and Graduate Studies
Roundtable Moderator: CFTR and Anion Exchange in the Intestine; 19th Annual North American Cystic Fibrosis Conference, Baltimore, MD; October 19-23, 2005
Moderator, Topic Forum: Bicarbonate transport, 107th Annual Meeting of the American Gastroenterological Association Institute, Los Angeles, CA (May 23, 2006).
Moderator, Translational Symposium: CFTR Pancreatic Disease and Secretory Diarrhea - Lessons From Animal Models, 107th Annual Meeting of the American Gastroenterological Association Institute, Los Angeles, CA (May 21, 2006)
Ad Hoc Reviewer, NIH – Gastrointestinal Cellular and Molecular Biology Study Section
Abstract reviewer, Epithelial Transport section, Am. Gastroenterological Assoc. Institute
Abstract reviewer, Genetics and Model Systems, Cystic Fibrosis Foundation

PROFESSIONAL SERVICE ACTIVITIES

Councilor, Intestinal Disorders Section, Am. Gastroenterological Assoc. Institute
Member, Cystic Fibrosis Foundation Research and Research Training Review Committee

Davis, M.J.

NIHICI study section, June, 2006

Gillis, K.D.

NIH study sections, BSCT, February, 2006, BPNS, June, 2006

Manuscript reviews: Biophysical Journal, Journal of General Physiology

Gu, Li-Qun

Manuscript reviewer: Journal of American Chemical Society, Analytical Chemistry

Hamilton, M.

Biomedical Sciences Department Graduate Program Advisory Committee

College of Veterinary Medicine Research Committee

Admissions and Scholarships Committee 2006-2009

Animal Resources Committee 2006-2008

Library Committee 2006-2008

Cardiovascular Day Judge

Research Mentor, Physiology Training Program and Exercise Training Program

Member - Molecular Biology Program, Health Activity Center and Gender Physiology Center

Community Science Education - Columbia Public School District Volunteer

DCRC Science Teacher Symposium - Assisted with Lecture Coordination

Co-Chair of Cellular and Molecular Regulatory Advisory Group of American College of Sports Medicine

Session Organizer and Chair - National ACSM Meeting

Manuscript Reviewer - Editorial Board of Journal of Applied Physiology

Ad hoc reviewer: American Journal of Physiology

Ad hoc reviewer: Journal of Lipid Research

Ad hoc reviewer: Physiological Genomics

Ad hoc reviewer: Journal of Comparative Physiology-B

Ad hoc reviewer: Journal of Applied Physiology

Hasser, E.

APS Publications Committee

Board of Scientific Counselors; National Space Biomedical Research Institute

University of Texas Medical Branch BBSC 6102-Research using ground-based flight analog models

Lecture: Hindlimb unloaded rats as a model of microgravity May 30, 2006

Manuscript review: *American Journal of Physiology, Hypertension, J. Physiol*

Heesch, C. M.

National Service: Chair, Research Committee American Heart Association Heartland

Affiliate: Meeting; November 2005

Manuscript reviews; American Journal of Physiology, Journal of Physiology (London), Brain Research, Journal of Applied Physiology, and European Journal of Pharmacology; October-December 2005

Chair, Research committee; American Heart Association, Heartland Affiliate Manuscript reviews January- March 2006: European Journal of Neuroscience

PROFESSIONAL SERVICE ACTIVITIES

Hill, M.A.

Research Investigator/Professor, Department of Medical Pharmacology and Physiology,
University of Missouri-Columbia; January 1, 2006

Huxley, Virginia

NIH Modeling and Analysis of Biological Systems (MAB) Study Section, ad hoc
Rippel Foundation Review, 2006
Chair, United States National Committee (USNC) for the International Union of
Physiological Sciences (IUPS)
International Union of Physiological Sciences (IUPS) commission on Committee on
Physiome and Bioengineering
Editorial Board, Journal of Vascular Research
Associate Editor, American Journal of Physiology (Heart & Circulation)
American Heart Association Abstract reviewer

Hwang, C.

BSCT NIH Study Section February 16-17, 2006
AHA Study Sections
Moderator, Williamsburg Conference sponsored by the Cystic Fibrosis Foundation
Instructor, Minicourse, 19th Annual North American Cystic Fibrosis Conference, Balti
more, MD;
October 19-23, 2005
Editorial board: Biophysical Journal, J. Gen. Physiol.
Manuscript review: J. Gen. Physiol., Biophys. J., Nature, Biochem. J.
Grant review: AHA, NIH, Dutch Digestive Foundation
Search committee member (Biological engineering)
Graduate education committee (Medical Pharmacology and Physiology)

Hyder, S.M.

Elected to Editorial Academy: International Journal of Oncology.
Ad hoc Reviewer: J Cellular Physiology,
Ad hoc Reviewer: J. Clinical Endocrinology and Metabolism
Ad hoc Reviewer: Cancer Research
Ad hoc Reviewer: Endocrinology
Ad hoc Reviewer: American Journal of Physiology
Ad hoc Reviewer: Phillip Morris External Grant Pgm
Ad hoc Reviewer: Breman Family Breast Cancer Institute, University of Miami, FL

Kline, D.

Assistant Professor, Department of Biomedical Sciences, University of Missouri-
Columbia; December 1, 2005
Ad hoc reviewer: Acta Physiologica Scandinavica, Physiological Genomics
Ad hoc reviewer: *Journal of Physiology* (London)
Reviewer for UM Research Board

Kornegay, J.N.

Board of Directors (Secretary), Association of American Veterinary Medical Colleges,
2006

Martinez-Lemus, L.

Assistant Professor, Department of Medical Pharmacology and Physiology, University of

PROFESSIONAL SERVICE ACTIVITIES

Missouri-Columbia; November 15, 2005
Member, Vascular Biology and Blood
Pressure Regulation Review Committee, American Heart Association-National, October
2006
Manuscript reviewer for: Journal of Vascular Research; Arteriosclerosis, Thrombosis, and
Vascular Biology; American Journal of Physiology
Associate Editor for the Physiology and Reproduction section of Poultry Science

Meininger, G.A.

External Evaluator of Candidates for Associate Professor for Physiology of Microcircula-
tion for Search Committee, Faculty of Medicine, Ludwig Maximilians Universitat,
Muenchen, Germany; October 2005
Member of Shared Instrumentation Microscopy (S 10) Study Section panel; Center for
Scientific Review, National Institutes of Health; October 2005
Program Grant review team for National Institutes of Health, Heart, Lung and Blood Insti-
tute; Cardiovascular and Renal Study Section; October 2005
Member, Vascular Wall Biology 2 Review Committee, American Heart Association-
National; October 2005
American Physiological Society: Strategic Planning Meeting; December 2005
Re-appointed Co-Editor (US) for Journal of Vascular Research, 2006-2009
Elected APS: Cardiovascular Section Secretary/Treasurer, 2006-2009
Appointed Co-Chair, Vascular Wall Biology 2 Grant Review committee, American Heart
Association-National, 2006-2007
Outside Committee Member, Thesis defense, Sungsoo Na, Effects of mechanical Force4s
on Cytoskeletal Remodeling and Stiffness of cultured Smooth Muscle Cells, March 2006

Mueller, P.

Faculty Grant Writing Institute; Office of Research, University of Missouri, May-
December 2006
Manuscript Reviewer, Hypertension, June 2006

Polo-Parada, L.

Assistant Professor, Department of Medical Pharmacology and Physiology, University of
Missouri-Columbia: February 1, 2006

Potts, J.T.

Member, AHA Study Section, Cardiac Biology/regulation; 2005-present
Member; American Physiological Society Communications Committee; 2005-present
Member; Joint programming Committee Rep, Neural Control and autonomic Regulation
section, American Physiological Society; 2005-present
Member; Faculty Responsibility Committee, College of Veterinary Medicine, University
of Missouri; 2005-present
Member; Graduate Program Advisory Committee, Department of Biomedical Science,
University of Missouri-Columbia; 2005-present
Fellow, American Heart Association and the Council for High Blood Pressure Research
AHA study section, Dallas, April 6-7
APS joint programming committee meeting, Washington, DC, June 13-14
Ad Hoc reviewer: Neuroscience, Journal of Neuroscience, Journal of Physiology, Ameri-
can Journal of Physiology – Heart / Reg, Journal of Applied Physiology

PROFESSIONAL SERVICE ACTIVITIES

Price, E.M.

Teaching: Course director, "Veterinary Cell Biology" (BmS 8506/7333), Fall 2005, 25 formal contact hours
Life Sciences Core Facilities Review Committee
Campus F&A Recovery Committee
Campus Revision of Records Committee
Teaching: Vet. Cell. & Mol. Biol. Course #VBmS 506 (Course Director)
MU Faculty Grievance Committee

Schadt, J.C.

Organizer/Moderator, Dalton Cardiovascular Research Center Science Teacher's Symposium; September and November, 2005 and February and April, 2006
Organizing committee and moderator, Phi Zeta Research Day in the College of Veterinary Medicine, University of Missouri-Columbia; March 17, 2006
Editorial Board, *Journal of Applied Physiology*.
Editorial Board, *American Journal of Physiology Heart and Circulatory Physiology*
Ad hoc, *American Journal of Physiology Regulatory, Integrative and Comparative Physiology*

Soma, Y.

Kyoto Prefectural University of Medicine, Graduate School of Medical Science, Kyoto, Japan, Adjunct Assistant Professorship; January 1, 2006
Editorial Board, Journal of the Physiological Society of Japan. ; July 1, 2006

Terjung, R.L.

Chair, elected by Environmental and Exercise Physiology Section, American Physiological Society. April, 2004-2007.
Member, NASA Artificial Gravity Research Program Oversight Committee, May, 2003 present.
Chair, Organizing Committee, Am. Physiol. Soc. Conference: Integrative Biology of Exercise V, 2006-2008.
Member, Section Advisory Committee, American Physiological Society, November 2004 - April 2007.
Member, Publications Committee, American Physiological Society, January 2004 - December 2006.
Member, Hypertension/Microvasc Study Section, NIH, Bethesda, MD, June/Oct 2006.
Member, Executive Committee, College of Veterinary Medicine, 2004-Present.
Member, Council of Research Advisors, Asst Provost for Research, 2004-Present
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, 2004-Present.
Member, Search Committee, Life Sciences Director
Member, Advanced Degree in Clinical & Translational Science Committee, 2006-Present.

Zou, X.

Reviewed manuscripts for the following journals: Proteins: Structure, Function and Genetics; Journal of Computational Chemistry; Journal of Chemical Information and Modeling; Current Computer-Aided Drug Design

Explanation of Figures

Top

Dr. Luis A. Martinez-Lemus

Three-dimensional reconstruction (frontal view) of a rat cremaster arteriole after both 5 minutes and 4 hours of exposure to 10^{-5.5} mol/L norepinephrine (NE). Two cell taper ends were digitally colored to show the increase in cell overlap occurring during the 4 hour-exposure to NE while the vessel maintained its reduced diameter.

From: Martinez-Lemus, L.A., S.S. Bolz, M.A. Hill, U. Pohl and G.A. Meininger. Acute mechanoadaptation of vascular smooth muscle cells in response to continuous arteriolar vasoconstriction: Implications to functional remodeling.

FASEB Journal, 18(6): 708-710, 2004.

Middle

Dr. Zhe Sun

Deflection image of a cultured vascular smooth muscle cell. The image was acquired using atomic force microscopy.

From: Sun, Zhe, Luis A. Martinez-Lemus, Andreea Trache, Jerome P. Trzeciakowski, George E. Davis, Ulrich Pohl, and Gerald A. Meininger. Mechanical properties of the interaction between fibronectin and $\alpha 5 \beta 1$ -integrin on vascular smooth muscle cells studied using atomic force microscopy. *Am J Physiol Heart Circ Physiol* 289: H2526–H2535, 2005.

Bottom

Dr. Jeffery T. Potts

Image showing Nuerokinin-1 receptor expressing neurons in the nucleus tractus solitarii.

From: Potts JT, Fong AY, Anguelov PI, Lee S, McGovern D, Grias I. *Neuroscience*. 2007 Mar 30;145(3):1168-81.

