Greetings WHRC members!

With the return of Fall, I’m sure everyone is back to working hard, doing experiments, writing papers, teaching, and chairing and serving on committees.

I just returned from a conference at National Heart, Lung, and Blood Institute at NIH on how to roll out the new initiative that takes effect with grants submitted beginning with the October deadlines that basic science investigators must identify the sex of the material they are using, whether cells, tissue, or animals, and where appropriate, propose to use both males and females in their studies. Many of us do not agree with the way NIH plans to roll out these new guidelines, despite the fact that we support the concept. If you want your opinion heard, respond to the Request for Information put out by the NIH Director’s Office on the topic: NOT-OD-14-128: “Consideration of sex as a biological variable in biomedical research”.

Interestingly, while these new guidelines are for basic research, the NIH still fails to require that clinical studies recruit enough men and women to be able to separate gender differences if they exist. Don’t get me started!

Kudos to Kedra Wallace from OB-GYN who just received a grant to study the etiology of uterine fibroids, recent findings highlighted on page 7! Congrats, Kedra!

You all have a great Fall.

Jane F. Reckelhoff

From the Director
Spotlight on Research

Joey Granger Ph.D, is the Billy S. Guyton Distinguished Professor, Professor of Physiology and Medicine, Director of the Cardiovascular-Renal Research Center, and Dean of the School of Graduate Studies in the Health Sciences at the University of Mississippi Medical Center in Jackson, MS. His laboratory has been continuously funded by mechanisms from the National Heart, Lung and Blood Institute (NHLBI) since 1985. Granger also currently serves as the principal investigator of a NHLBI Institutional Training Grant entitled “Hypertension and Cardiorenal Diseases Research Training Program”.

Granger has been a member of Council on Hypertension for over 30 years and is the incoming Chair of the Council. He has been a long time member of the American Physiological Society (APS) and recently completed his term as President of the APS.

He has authored or co-authored over 250 peer-reviewed publications, many of them in the Hypertension journal. Granger is currently an Associate Editor for Hypertension and serves as Co-Editor with his brother, Neil Granger, on the eBook series entitled Integrative Systems Physiology. He served as the Editor of the Council for High Blood Pressure Newsletter and an Associate Editor for News in Physiological Sciences and American Journal of Physiology: Regulatory and Integrative Physiology.

Granger has received several awards for his research, including the 2010 AHA Distinguished Scientist Award, APS Ernest H. Starling Distinguished Lecture Award, APS Bodil M. Schmidt-Nielsen Distinguished Mentor and Scientist Award, Dahl Memorial Lecture of the AHA, American Society of Hypertension Young Scholar Award, the International Society of Hypertension Demuth Research Award, Inter-American Society of Hypertension Young Investigator Award, the Regulatory and Integrative Physiology Young Investigator Award of the APS Water and Electrolyte Section, the Harold Lamport Award of the Cardiovascular Section of the APS the Henry Pickering Bowditch Lecture of the APS, and the Established Investigator Award of the AHA. Dr. Granger’s research focuses on the mechanisms responsible for preeclampsia and on the role of endothelin in hypertension.

The WHRC is proud to have Dr. Joey Granger as an active member!
At the recent Council on Hypertension in San Francisco, Dr Reckelhoff received the Distinguished Achievement Award for her research and her contributions to the American Heart Association and the Council.

Dr. Reckelhoff is a Billy S. Guyton Distinguished Professor, and Professor of Physiology & Biophysics at the University of Mississippi Medical Center. She is also the Director of the Women’s Health Research Center and Director of Research Development in the UMMC Office of Research. Dr. Reckelhoff has been a leader in research related to understanding how sex steroids and gender differences impact the development of hypertension and renal disease.
Denise Cornelius, Ph.D., postdoctoral fellow in Pharmacology & Toxicology in the LaMarca Laboratory received the Annual HBPR Conference New Investigator Award. Her abstract was entitled “T cell Dependent B cell activation plays a role in mediating hypertension and pathophysiology in response to CD4+ T cells from RUPP rats.” She received her award in San Francisco in September 2014 at HBPR Scientific Sessions.

Xuexiang Wang, Ph.D., received an Onsite Trainee Poster Award at HBPR Scientific Sessions 2014 in San Francisco for his poster entitled, “Nephron Deficient Rats are Highly Susceptible to Hypertension Induced Kidney Injury”. Xuexiang Wang is a senior graduate student in Dr. Michael Garrett’s lab in the Department of Pharmacology & Toxicology.

Lorena Amaral, Ph.D., postdoctoral fellow in the Lamarca Laboratory was received a Young Investigator Travel Award to attend The XIX World Congress for the Study of Hypertension in Pregnancy; Hilton New Orleans Riverside Hotel, New Orleans, LA, USA, October 25 to 29, 2014. Her abstract is entitled “17-hydroxyprogesterone caproate attenuates hypertension and uterine artery resistance in response to Reduced Uterine Perfusion Pressure (RUPP) in pregnant rats”.
University of Mississippi Medical Center
Division of Nephrology and
Women’s Health Research Center

Present:
Phenotypes in Hypertension: One size does not fit all!
Marcelo Orias, M.D., Ph.D. Chief of Nephrology,
Chief of Renal Transplant
National University of Cordoba, Argentina

Monday, November 17, 2014
12:00PM — 1:00 PM, 6A

SAVE THE DATE!
The Fifth International Conference on Sex and Gender
Sponsored by the American Physiological Society and
the Women’s Health Research Center
Metabolic, Cardiovascular, and Renal Disease: Physiology & Gender
Annapolis Sheraton, November 17-20, 2015

ISSHP XIX World Congress
October 24 – 29, 2014
New Orleans, LA
USA
http://isshp2014.com/
Cerebrovascular (CB) events contribute to ~40% of preeclampsia/eclampsia-related deaths, and neurological symptoms are common among preeclamptic patients. These authors tested the hypothesis that placental ischemia leads to impaired CBF autoregulation and disruption of blood brain barrier (BBB). CB flow autoregulation was significantly impaired in placental ischemic rats, while brain water content and BBB were increased. These results are consistent with the hypothesis that placental ischemia mediates anterior cerebral edema through impaired CBF autoregulation and associated increased transmission of pressure to small vessels that increases BBB permeability leading to cerebral edema.

HELLP syndrome is a severe form of preeclampsia with placental ischemia hypothesized as being the culprit in causing this disease. To date, the occurrence of neurological complications in these women has been reported, but few studies have examined whether impairment in blood-brain barrier (BBB) permeability or cerebrovascular reactivity is present in patients with HELLP syndrome. These authors hypothesized that plasma from women with HELLP syndrome causes increased BBB permeability and cerebrovascular dysfunction. Posterior cerebral arteries from female nonpregnant rats were perfused with 20% serum from women with normal pregnancies (n = 5) or women with HELLP syndrome (n = 5), and BBB permeability and vascular reactivity were compared. Plasma from women with HELLP syndrome increased BBB permeability while not changing myogenic tone and reactivity to pressure. Thus, plasma from patients with HELLP syndrome increased BBB permeability and was associated with selective endothelial dysfunction.
Efforts have been made to catalog the cardiac extracellular matrix and analyze the topology of identified proteins for the design of therapeutic targets. The aim of this study was to use a glycoproteomics and MS approach to identify glycoproteins in the extracellular matrix of the infarcted left ventricle (LV) and provide experimental evidence for topological determination. Glycoproteomics analysis was performed on eight biological replicates of LV samples from wild-type mice 7 days following myocardial infarction using SPE of glycopeptides, followed by mass spectrometric identification of N-linked glycosylation sites for topology assessment. These authors identified hundreds of glycoproteins, and the identified N-glycosylation sites provide novel information on the correct topology for membrane proteins present in the infarct setting. Our data provide the foundation for future studies of the LV infarct extracellular matrix, which may facilitate the discovery of drug targets and biomarkers.

Wallace K, Chatman K, Porter J, Scott J, Johnson V, Moseley J, LaMarca B. **Endothelin 1 is elevated in plasma and explants from patients having uterine leiomyomas.** *Reprod Sci.* 2014 Sep;21(9):1196-205.

The objective of this study was to determine a role for endothelin (ET) in progression of uterine fibroids utilizing an in vitro model of fibroid and myometrium cultivation. A total of 32 women undergoing hysterectomies for uterine fibroids and 11 women undergoing hysterectomies for abnormal uterine bleeding (control population). Women with uterine fibroids were hypertensive and displayed significantly greater circulating ET-1 compared to control patients. Secretion of ET-1 was greater from the fibroids compared to myometrium explants, and was attenuated with blockade of the angiotensin II type 1 or endothelinA receptors. Hypoxia stimulated ET-1 secretion from both myometrium and fibroid explants while endothelin mRNA increased with hypoxia from only fibroid explants compared to normoxic controls. These studies support the hypothesis that vasoactive pathways may be stimulated and playing a role in hypertension during the development of uterine fibroids.

Women’s health issues are underfunded and understudied. Help support women’s health research by making a tax-deductible contribution. 

Contact the Development Office at UMMC at 601-815-7473 for more information.