Greetings WHRC members!

As we move into summer, I know everyone is busy with summer students in the lab and new patients. A few things that are going this summer:

Dr. Ida Llewellyn-Smith from Australia will be visiting us Sunday through Wednesday the week of July 20, 2014. She is a neurophysiologist and is very interested in pregnancy. She is giving our WHRC seminar on July 21 at 4PM in CW106. If you would like to meet with her, let me know. In addition, she’s a kick and lots of fun!

If you haven’t heard the buzz and seen the paper in Nature by Francis Collins and Janine Clayton (Director of the Office of Women’s Health Research in HHS), take a look (Nature, May 2014: Policy: NIH to balance sex in cell and animal studies.) Beginning this Fall, NIH is going to require the basic science grants include males AND females where appropriate and that all cell studies identify the sex of the cells. Then see the response by Kathryn Sandberg et al. (Am J Physiol. 2014, Point/Counterpoint: Sex and Basic Science. A Title IX Position). Interesting reading on both!

We are beginning to plan for our Fifth APS conference for the Fall of 2015, October 14-16. Please put it on your calendars. I’ll be sending out “Save the date” announcements soon. Hope you will suggest potential speakers, yourself or others who are interested in women’s health, sex and gender differences.    Happy summer, Janie

From the Director

Jane F. Reckelhoff
Licy Yanes Cardoza, MD., Endocrine Fellow in the Department of Medicine, was awarded with the 2014 Research Fellow of the Year by the departmental medical staff. In addition, Dr Yanes Cardoza was also awarded a Clinical Fellows Abstract Award/Travel Grant in Women’s Health supported by Pfizer, Inc, and has been selected as a finalist in the poster presentation at the Minority Affairs Committee at the Endocrine Society Meeting held in Chicago.

Dr. Yanes completed her medical training at the National University of Asuncion, Paraguay, and moved to the Department of Physiology at UMMC in Jackson to train as a postdoctoral research fellow under the guidance of Dr Jane Reckelhoff. While in the Reckelhoff laboratory, Licy studied the roles of several vasoconstrictor pathways and their roles in mediating postmenopausal hypertension in a rat model. In addition, she developed a rat model of polycystic ovary syndrome (PCOS) that exhibited many of the characteristics of cardiovascular disease in women with PCOS, including hyperandrogenemia, metabolic syndrome, insulin resistance, reductions in glomerular filtration rate and elevated blood pressure.

Dr Yanes Cardoza was promoted to instructor after she received a Postdoctoral Fellowship Award from the American Heart Association, and then rose to an Assistant Professor in Physiology after she received a Scientist Development Grant from the American Heart Association. She then began her residency in 2010 followed by a fellowship in Endocrinology in 2013. In her recent grant from the Endocrine Fellows Foundation in 2014, entitled, "Mechanisms of testosterone replacement-mediated hypertension in obesity-related hypogonadism", Licy expanded previous studies in which she found that androgen treatment of obese male rats reduced body mass, improved insulin sensitivity, and reduced inflammatory cytokine markers, but caused an increase in blood pressure. In this grant, she will focus on the molecular mechanisms responsible for testosterone supplementation-mediated renal injury and hypertension in an animal model of obesity and hypogonadism. These timely studies will allow us to understand more about the complex effects of testosterone supplementation in hypogonadal men.

The WHRC is proud to have Dr. Yanes Cardoza as a member and we wish you the very best as you grow in your career!
Barbara Alexander, Ph.D., Professor of Physiology, was awarded an Grant-in-Aid entitled “Age-Dependent Hypertension in Female Growth Restricted Rats” from the American Heart Association which will begin July 1, 2014.

Sydney Murphy, Ph.D., Instructor in Pharmacology and Toxicology, was awarded a Scientist Development Grant from the American Heart Association entitled “Vascular eicosanoids and preeclampsia” which will began July 1 2014. Dr Murphy performed her postdoctoral training under the tutelage of Dr. Richard Roman.

Kedra Wallace, Ph.D., Instructor in Obstetrics & Gynecology, was recently awarded an NIH R03 grant funded through the Center for Psychiatric Neuroscience, beginning July1, for her work entitled “Does Dexamethasone improve postpartum psychological health in rats with HELLP Syndrome?”
Peter Mittwede, mentored by Robert Hester in the Department of Physiology and Biophysics, was selected Predoctoral Research Recognition Awardee for 2014 by the Water and Electrolyte Homeostasis Section of the American Physiological Society (APS). In addition Peter was recently awarded the Young Investigator Award from the Society for Experimental Biology and Medicine, 2014.

Kasi McPherson, mentored by Jan Williams in the Department of Pharmacology and Toxicology, was selected as a recipient of the Kent Scientific Physiological Genomics Trainee Research Excellence Award for her oral presentation at the APS Physiological Genomics Trainee Highlights Session.

Maryam Syed, mentored by Dr. Damian Romero in the Department of Biochemistry, was selected as a recipient of Physiological Genomics Trainee Research Excellence Award for her oral presentation at the APS sponsored 2014 Experimental Biology Meeting in San Diego, CA.
**WHRC Seminar**

Dr. Ida Llewellyn-Smith, Professor in Medicine, Department of Medicine School of Medicine, Flinders University, Bedford Park, South Australia, Australia, Adjunct Professor, Department of Physiology, School of Medicine, Wayne State University, Detroit, Michigan, will discuss “Changes in uterine innervation during pregnancy and post-partum in the rat”. Her seminar will be July 21, 2014 in 6A at 4pm. All students, faculty and staff are invited. Coffee and cold drinks will be served. *Please come and join us!*

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**ISSHP XIX World Congress**

October 24 – 29, 2014
New Orleans, LA
USA
http://isshp2014.com/

*Abstract deadline is July 1, 2014!*

This review highlights mechanisms proposed by the Developmental Origins of Health and Disease (DOHaD) suggesting that adverse events during early life program an increased risk for cardiovascular disease. Experimental models provide proof of concept but also indicate that insults during early life program sex differences in adult blood pressure and cardiovascular risk. This review further highlights the potential mechanisms that contribute to the etiology of sex differences in the developmental programming of cardiovascular disease.


Acute renal injury (AKI) is more prevalent in males than in females suggesting a role for adverse effects of testosterone. The oxidant sensor p66shc is regulated by testosterone, and therefore may be responsible for the gender disparity. In this study the authors found that testosterone in the form of DHT increased hydrogen peroxide-dependent oxidative stress and renal injury via p66shc and expression of p66shc via promoter activation. Renal expression of p66shc was higher in male compared to female kidneys. The authors conclude that higher sensitivity of the male kidney to AKI may be due to the testosterone-dependent increase in p66shc expression.


The mechanisms responsible for the gender difference in blood pressure (BP) in humans are not clear. In this study the authors tested the hypothesis that renal vascular and microsomal epoxieicosatrienoic acid (EETs) I were higher in female than male spontaneously hypertensive rats (SHR) and that blocking epoxide hydrolase would reduce BP more in males than females. Renal vascular and microsomal EETs and BP were higher in female SHR than males, however, the hydrolase inhibitor did not affect BP. These data suggest that EETs do not contribute to the sex differences in hypertension in young SHR.

Utilizing a relevant preclinical rodent model of placental ischemia-induced hypertension, the reduced uterine perfusion pressure (RUPP) model, to determine the effect of chronic placental ischemia on the underlying chorionic tissue and placental villi, these authors found via microarray analysis a strong effect on inflammatory pathways, including those involving NF-κB and inflammatory cytokines. Of the most differentially expressed genes, the predominant gene classes were extracellular remodeling proteins, pro-inflammatory proteins, and a coordinated upregulation of the prolactin genes. This paper discusses the potential functional implications of these novel factors in mediating this disease.


The mechanisms causing excessive aldosterone production and hypertension in primary aldosteronism (PA) are complex and often incompletely recognized. Autoantibodies to the angiotensin AT1 receptor (AT1R) (AT1-AA) have been reported in some PA patients with an aldosterone-producing adenoma but not with idiopathic adrenal hyperplasia. These authors investigated whether AT1-AA contribute to the pathophysiology of PA. Sera from each of the 13 PA patients significantly increased AT1R activation in cultured cells compared with 20 control subjects, and this activity was inhibited by losartan. Sera and purified AT1-AA caused vasoconstrictive effects in isolated rat cremaster arterioles which was blocked by losartan. Moreover, the AT1-AA directly stimulated aldosterone production in the cultured adrenal cells and enhanced angiotensin-induced aldosterone production in these cells, which was blocked by candesartan. These data support a probable pathophysiological role for AT1-AA in PA and thereby raise important etiological and therapeutic implications.

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.