

## **Call for Abstracts!**

*Women in Medicine and Science* is seeking abstract submissions for

### **Women's Health Research Day**

*What's on HER mind? Gender Differences in Cognitive Function*

Wednesday, April 17, 2019

8:30am – 4:00pm

Commons Conference Rooms - Wake Forest Baptist Medical Center

Accepting submissions from:

- Faculty/Staff
- Students (medical, graduate, PA, and other)
- Medical Interns, Residents & Fellows
- Post-Doctoral Fellows & Research Fellows

**Deadline for submissions is February 18, 2019!**

Submit your one-page abstract (new or recycled) to [wims@wakehealth.edu](mailto:wims@wakehealth.edu).

Please include the following with your submission:

- Full name
- Degree
- Department
- If student or trainee provide your school, program and year (e.g., Graduate School, MS in Biomedical Science, Year-1)
- Project title
- Project co-authors
- Use the format on the Example Abstract (margins 1 inch, single-spaced, Arial11, bold where indicated)
- Identify the source(s) of support for the work (in italics).

*Limited to one abstract submission per person.*

*Top 20 abstracts will be invited to display their posters.*

*Top 10 student/trainee abstracts will be selected to provide a 1-minute preview of their poster at the podium to encourage the audience to visit their poster.*

*While all women's health & sex differences abstracts are encouraged, special consideration will be given to those with focus on cognitive function in women.*

*Best Poster Awards will be presented at the end of WHRD.*

*(Faculty submissions are not eligible for Awards or 1-minute previews)*

## *Sample Abstract*

Hannah M. Atkins  
DVM  
Department of Pathology,  
WFSM Research Fellow

### **Endometrium mitochondrial function is decreased in a nonhuman primate model of endometriosis**

Hannah M. Atkins, DVM; Susan E. Appt, DVM; Manish Bharadwaj, PhD; David L. Caudell, DVM, PhD

Biomarkers of oxidative stress, such as DNA damage and increased reactive oxygen species, are associated with endometriosis pathogenesis. However, little is known about the effects of oxidative stress on endometrium metabolism, inflammation, and infertility. We used a nonhuman primate (NHP) model of endometriosis to determine if endometriosis tissue and endometrium had abnormal mitochondrial function and energy metabolism. Mitochondrial function was analyzed using mitochondrial respirometric analysis to determine if changes in oxidative phosphorylation exist in endometrium and endometriosis tissue compared to clinically healthy control endometrium. Targeted metabolomics was applied to tissues in order to identify pathways relevant to energy production. Endometrium from NHPs with endometriosis had reduced complex II-mediated oxygen consumption rates across all energy states (basal,  $p = 0.01$ ; state 3,  $p = 0.02$ ; state 3u,  $p = 0.04$ ; state 4o,  $p = 0.008$ ). Endometriosis tissue had reduced state 3, complex I-mediated oxygen consumption ( $p = 0.02$ ) and respiratory control rates ( $p = 0.01$ ) compared to control endometrium. Pyruvate ( $p = 0.01$ ), FAD ( $p = 0.004$ ), and malic acid ( $p = 0.03$ ) were significantly decreased in endometrium from macaques with endometriosis compared to control endometrium. Citric acid was also decreased and trended towards significance ( $p = 0.06$ ). Carnitine ( $p = 0.001$ ), pyruvate ( $p = 0.03$ ), NADH ( $p = 0.001$ ), glycerophosphocholine ( $p = 0.006$ ), FAD ( $p = 0.001$ ), and malic acid ( $p = 0.005$ ) were decreased in endometriosis tissue compared to control endometrium. Again, citric acid was decreased in endometriosis tissue but not significant ( $p = 0.06$ ). Significant metabolites identified in both endometriosis and endometrium samples from animals with endometriosis were matched to citric acid cycle, glyoxylate and dicarboxylate, and pyruvate metabolism pathways. Altogether, these data suggest that reactive oxygen species may play a role in feedback mechanisms onto endometrial mitochondria to decrease energy production. Decreased endometrial metabolism may contribute to endometriosis-associated infertility and reducing systemic oxidative stress could be a tool for improving fertility in women with endometriosis.

*Supported by XXXXX.*