



Faculty Affairs - Women in Medicine and Science

## Call for Abstracts!

Women in Medicine and Science is seeking abstract submissions for

### Women's Health Research Day

*"Healthy Aging – Women's Health Beyond Menopause"*

Abstracts are open to any topic regarding women's health.

Tuesday, May 24, 2022

9am – 3pm

Location: Bowman Gray Center for Medical Education (BGCME)

Accepting Submissions from:

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

### Deadline for Submission is March 29, 2022.

Submit your one-page abstract (new or recycled) via REDCap ([2022 WHRD Abstract Submission](#))

Please submit 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 Abstract Example (1-inch margins, single-spaced, Arial 11, bold where indicated)
- Identify the source(s) of support for the work (in italics).

*Limited to one abstract submission per person.*

*All submissions will be reviewed and an email notification will be sent if you are selected to display a poster on the day of the event by April 26, 2022.*

*Top 10 student/trainee abstracts will be selected to provide a 1-minute preview of their poster during the event.*

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

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

**Wake Forest School of Medicine is the academic core of Atrium Health**



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

*"Healthy Aging – Women's Health beyond Menopause"*

### **Call for Abstracts Example:**

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.*