THE UNIVERSITY OF ARIZONA
COLLEGE OF AGRICULTURE & LIFE SCIENCES
Nutritional Sciences & Wellness

# SEMINAR ANNOUNCEMENT

The School of Nutritional Sciences and Wellness presents:

## "Myristate, Sphingolipids, and Endoplasmic Reticulum Stress in Intestinal Epithelial Cells"


By

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*Moderated by: Yuze Wu*

*Wednesday, April 27th 2022*
*12pm*
*Shantz Building, Room 247*
https://arizona.zoom.us/j/82678706371

**Myristate, Sphingolipids, and Endoplasmic Reticulum Stress in Intestinal Epithelial Cells**

Rates of inflammatory bowel disease (IBD) have been steadily increasing in the United States over the last 20 years. A variety of factors such as endoplasmic reticulum (ER) stress, sphingolipid metabolism, and diet have all been implicated in IBD. We previously found that the C14:0 saturated fatty acid myristate (found predominately in high fat dairy) induced ER stress and inflammation in intestinal epithelial cells. Specifically, C14-Ceramide generated by ceramide synthase (CerS) 5 and/or 6 regulated IRE1α-mediated XBP1 splicing and downstream target genes. To further understand the mechanism involved and extend our previous findings, we are transitioning from rat intestinal epithelial cells (IEC6) to a human intestinal epithelial cell line (HCEC 1CT) as a more relevant model for human disease. To that end, we treated HCEC 1CT cells with myristate and analyzed ER stress signaling and downstream target genes. As demonstrated previously in IEC6 cells, ER stress was significantly increased in HCEC 1CT, including activation of IRE1α. Knockdown of IRE1α suppressed myristate induced XBP1 splicing and IL6 expression. Together these data suggest a role for IRE1α in myristate-induced ER stress. Future work will define the role of specific sphingolipid enzymes and their bioactive products in fatty acid induced ER stress and inflammation.