Discovery Science Emerging Scholars Lecture

"OPA-1 Deficiency in Skeletal Muscle Increases Mitochondria ER Contact Formation Through an ATF-4 Dependent Mechanism"



Antentor "A.J." Hinton, Ph.D. Postdoctoral Fellow University of Iowa In the laboratory of E. Dale Abel we have found that reduction in OPA1 (Optic Atrophy 1, a mitochondrial dynamin-like GTPase) expression in skeletal muscle results in endoplasmic reticulum (ER) stress response and FGF21 secretion. We predicted that there was an association between the loss of OPA1, an increase in ER-Mitochondrial Contact sites, and prevalence of diabetes. Mitochondrial endoplasmic reticulum contact sites (MERCs) are specialized membranes that are enriched with specific proteins believed to be important for calcium flux, lipid transfer, and mitochondria morphology. This observation provides a plausible mechanism linking altered mitochondrial dynamics with the activation of the ER stress response pathway and may give novel insight into how some patients develop insulin resistance. We propose to use this information to provide the foundation for future research that will lead to the discovery of more effective biomarkers that identify insulin resistance in skeletal muscle.

Thursday March 5, 2020 9:30 a.m.

206 Preston Research Building

Refreshments will be served!

This lecture series features the most promising young scientists who are making notable discoveries as postdoctoral fellows or early career faculty.

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