

University of South Florida

The Department of Cell Biology,
Microbiology and Molecular Biology

Announces a seminar by

Sasanka Ramanadham, Ph.D.

Professor, Department of Cell,
Developmental, and Integrative Biology
Comprehensive Diabetes Center
University of Alabama at Birmingham

on January 4th, 2018
at 3:15pm in ISA 2023

“Altering Lipid Signaling in Immune Cells and Islet Beta-cells to Mitigate Type 1 Diabetes Development”

Type 1 diabetes (T1D) is a consequence of autoimmune destruction of β -cells, involving activation of cellular immunity leading to leukocyte infiltration of islets. An understudied area is the role lipid signals play in this process. We find that the Ca^{2+} -independent phospholipase $\text{A}_2\beta$ (iPLA $_2\beta$) is induced under a diabetic milieu and the mitigation of iPLA $_2\beta$ attenuates β -cell death. The iPLA $_2\beta$, a member of the PLA $_2$ family, hydrolyzes the *sn*-2 substituent from glycerophospholipid substrates to yield a free fatty acid, which can be metabolized to bioactive lipids. The focus of our lab is to identify the role of lipid signaling originating from immune cells and beta-cells in promoting beta-cell death, leading to T1D. We utilize beta-cell lines to examine specific mechanisms (i.e., ER stress, mitochondrial processes, alternate splicing) and mouse models (spontaneous diabetes-resistant and spontaneous diabetes-prone) to identify the impact of iPLA $_2\beta$ -derived lipids on beta-cell death. Recent studies have identified a link between iPLA $_2\beta$ activation and T1D incidence, in association with alterations in select lipids. Our long-term goal is to identify specific signaling pathways that involve iPLA $_2\beta$ -derived lipids and assess their feasibility for intervention to counter T1D development.



THE PUBLIC IS INVITED.
FOR DISABILITY
ACCOMODATIONS, PLEASE
CALL (813) 974-4071

ResearchOne
ONE UNIVERSITY : ONE COMMUNITY : ONE VISION