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Major Professor: Dr. Derek Boeldt

Degree Objective: Ph.D. Endocrinology and Reproductive Physiology

Background: B.Tech Biotechnology, National Institute of Technology - Warangal, India 2016

Current Research Project: Study of calcium signaling due to immune-endothelial interactions in preeclampsia.

I intend to study the effect of the cross talk between immune cells and endothelial cells (EC) on Ca2+ bursting in ECs in the context of pregnancy. A demand for increased blood flow to the uterus in pregnancy is met by vasodilation of the uterine arteries. Enhanced Ca2+ signaling in ECs is responsible for increasing the production of nitric oxide that acts as a vasodilator. Our laboratory has already shown that Ca2+ bursting is reduced in ECs in preeclampsia compared to normal pregnancies. Further, preeclampsia is associated with aberrant infiltration of immune cells and changes in numbers/proportions of immune cells, implying that immune cell mediated signaling could be aberrant. This aberrant signaling could potentially inhibit Ca2+ signaling in ECs due to inhibition of gap junction function or inhibition of adhesion molecule interactions. It is also known that treatment with cytokines like TNFα can inhibit Ca2+ bursting in ECs. The effect of cytokines in modulating Ca2+ bursting suggests that immune cells themselves can modulate Ca2+ bursting.

Studies using Human umbilical vein endothelial cells (HUVEC) and Peripheral Blood Mononuclear cells (PBMC) co-cultures have demonstrated that trans-endothelial migration of immune cells is associated with endoplasmic reticulum Ca2+ release in endothelial cells. Apart from the effect of immune cell secretions on Ca2+ bursts, the presence of immune cells in close proximity to ECs in the utero-placental microenvironment renders immune cells to the possibility of influencing EC Ca2+ bursts through heterogeneous cell-cell contact. This may be indicative of an element of immune cell influenced Ca2+ bursting in ECs, which is potentially dysfunctional in the case of preeclampsia. My aim is to study the Ca2+ response that occurs as a result of a co-culture of HUVECs with PBMCs to gain insights into the pathophysiology of preeclampsia.

Honors:

Grants Received:

Publications:

National Presentations:

Other Presentations:

Teaching and Mentorship:

ERP Service: