

SPECIAL RESEARCH SEMINAR

Presented by the Center for Vascular and Heart Research at the Fralin Biomedical Research Institute at VTC



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In Person Seminar: Fibrosis and Aberrant Ca²⁺ Handling in the Failing Heart

Fibrosis and aberrant Ca²⁺ handling are hallmarks of heart failure but are considered separate features. However, the fibrotic disposition of nanometer-sized collagen fibrils, particularly collagen VI (ColVI), may directly diminish Ca²⁺ signalling in human heart failure. Using super-resolution microscopy Dr. Crossman and his team found increased ColVI within the transverse(t)-tubules. Moreover, t-tubule remodeling disrupts Ca²⁺ signaling in heart failure. Recently, circulating levels of ColVI were found to independently associate with poor outcomes in heart failure. While the importance of ColVI to skeletal muscle function is underscored by its mutation causing muscular dystrophy, its role in cardiac contraction remains poorly understood. Significantly, muscular dystrophy patients often develop heart failure. To understand the role of ColVI in the heart the lab created a knockout of this gene in the rat (Col6a1^{-/-}). Echocardiography showed both impaired systolic and diastolic function. Cardiac myocytes from Col6a1^{-/-} have increased systolic Ca²⁺ transient and greater response to b-adrenergic stimulation that is pro-arrhythmogenic. Similar disturbance in Ca²⁺ handling is found in the cardiac myocytes of the mdx mouse which has non-functional dystrophin supporting our hypothesis that ColVI is part of the dystrophin-glycoprotein-complex and may have a role in regulating Ca²⁺ dynamics.

THURSDAY, FEB. 15, 2024 at 11 a.m.

Room R3012, 2 Riverside Circle.

Or watch via Zoom: <https://virginiatech.zoom.us/j/85661403607>



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