FIU Engineering & Computing
Biomedical Engineering

Miami Heart ♥ Day
Friday, February 21, 2020

Wallace H. Coulter Foundation
Biomedical Engineering Seminar Series

KATHERINE E. YUTZEY, PHD, FAHA, FAAA, Professor of Pediatrics, Cincinnati Children's Hospital Medical Center. She has a BA in Biology from Oberlin College (1986), a PhD from Purdue (1992), and post-doctoral training in heart development. She joined CCHMC as Assistant Professor (1995) and was appointed Professor in 2007. Dr. Yutzey is the first recipient of the Schmidlapp Women Scholars Award and currently holds an endowed chair from the Cincinnati Children's Research Foundation. Her work is supported by NIH and the American Heart Association. Current areas of research include heart valve development and disease, including inflammation, and mechanisms of cardiomyocyte proliferation.

DR. KATHERINE YUTZEY

Professor, UC Department of Pediatrics

Cincinnati Children's Hospital Medical Center

Heart Valve Development and Disease Mechanisms

ABSTRACT: Congenital heart valve malformations or developmental anomalies can lead to progressive valvular degeneration and dysfunction, necessitating replacement later in life. Myxomatous valve disease (MVD) is characterized by thickening of valve leaflets and increased proteoglycan accumulation, leading to prolapse and valvular insufficiency. Mouse models of MVD were used to identify critical contributions of valve and immune cell lineages, extracellular matrix (ECM) organization, and cell signaling to the initiation and progression of heart valve disease. Mice with the Marfan Syndrome Fibrillin1(FBN1) C1039G mutation exhibit mitral valve thickening, increased numbers of CD45+ leukocytes, and abnormal ECM remodeling within 2 months after birth. Interestingly, increased Wnt/beta-catenin signaling is detected in valve interstitial cells of the FBN1C1039G mice, along with increased numbers of CD45+ cells and cytokine signaling, at early stages of the disease. Moreover, increased numbers of CD45+ cells and macrophages are present in a pig Marfan syndrome model with myxomatous mitral valves, as well as in human MVD. In normal heart valves, CD45+ myeloid lineage cells are present at birth, and their numbers increase during post-natal heart valve remodeling. The majority of the valvular CD45+ cells are macrophages, as confirmed by flow and lineage tracing of Cx3Cr1 expressing cells. In MVD, CD45+ proinflammatory monocytes are increased prior to major ECM abnormalities. In Marfan syndrome mice, deficiency of circulating macrophages improves valve morphology and prevents ECM abnormalities. Thus targeting macrophages may be an effective strategy for development of new therapeutic approaches to preventing myxomatous disease progression.

Wallace H. Coulter Foundation

Through the generous support of the Wallace H. Coulter Foundation the Department of Biomedical Engineering facilitates weekly lectures each year during academic terms. Experts in all areas of Biomedical Engineering are invited to campus to provide a research seminar and to meet with faculty and students and to tour our academic and research facilities.

Friday, February 21, 2020
9:30AM-10:30AM MMC Everglades 141