Abstract: Heart failure with preserved ejection fraction (HFpEF) is an increasingly prevalent, complex clinical syndrome with no proven treatment strategy. HFpEF is characterized by impairment in ventricular filling and increased diastolic stiffness. The two major sources of left ventricular passive stiffness are the extracellular matrix and the cardiomyocytes, in which passive stiffness is mainly determined by the giant sarcomeric protein titin. Titin stiffness can be modulated through differential splicing (changes in titin isoform expression) and through posttranslational modification, including phosphorylation. Due to its inherent tuneability, titin lends itself to being a potential therapeutic target for ameliorating increased diastolic stiffness. Recent work by Muller, et al. has suggested that the commonly used diabetes drug, metformin, induces titin isoform switching in cells in culture. In this present study, we investigated the effect of metformin on diastolic stiffness in pathological hearts specifically focused on its effects on titin.

Please join us on

Monday, October 17th, 2016
2:00-2:50 pm, Keating Bldg, Room 103

Host: Henk Granzier, Ph.D.
granzier@email.arizona.edu

Persons with a disability may request a reasonable accommodation by contacting the Disability Resource Center at 621-3268 (V/TTY).