Dr. Daniel Fitzsimons' Lab: Molecular mechanisms underlying cardiac muscle function in normal and diseased states

The overall objective of my research program is to identify the molecular processes that underlie contraction and relaxation of mammalian cardiac muscle under normal conditions and in disease states, such as cardiomyopathy and heart failure. From the viewpoint of systems design, the expression of proteins within the myocardial proteome (i.e., contractile, calcium handling, sarcolemmal etc…) ensures a close, synchronous relationship between acute, beat-to-beat changes in myocardial work capacity and the kinetics of contraction and relaxation of force. This tight structure-function relationship is readily apparent across the biological spectrum of mammalian myocardium. However, with the onset and progression of disease states, such as cardiomyopathy, the heart typically exhibits a diminished ability to acutely regulate myocardial work capacity due to a mismatch between contractile kinetics and the requirements for myocardial work. Therefore, the characterization of the regulation of cardiac contraction and relaxation under normal conditions will allow us to gain a more complete understanding of the beat-to-beat regulation of cardiac function. Furthermore, this approach will also identify specific molecular defects in heart disease, thereby providing insight for potential therapeutic interventions and treatments.

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