Cardiovascular disease is the leading cause of death globally and imposes a great financial burden on the economy. Two such cardiovascular diseases are known as hypertrophic cardiomyopathy (HCM) and dilated cardiomyopathy (DCM). These diseases are the most common diseases of the heart muscle, yet very little is known regarding their development. It is known however, that mutations in proteins found in the sarcomere put one at a greater risk for cardiomyopathy. Actin is a protein found in the sarcomere which has displayed a direct link to both HCM and DCM. It is thought that these cardiomyopathies result from one of two possibilities. The first is the inability of tropomyosin to properly regulate the actin filament, while the second is that the force output of individual myofibers has been altered. To test this, several projects will be undertaken assessing these theories. The first project will aim to perform a full work up on the actin variants which lie in the tropomyosin binding site. This will include stability determination, how well the actin interacts with active myosin and the calcium sensitivity. Next, tropomyosin binding will be examined with a fluorescent binding assay. The final project will gauge the force output of reconstituted myofibers containing each actin variant using a force transducer. The combination of these projects should lead to a better understanding of how cardiomyopathy develops and what role actin plays in this mechanism.