

Public Abstract

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Effects of ischemic metabolites and chronic exercise on cardiac myocyte power output

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Heart disease is the leading cause of morbidity and mortality in the civilized world. However, the molecular mechanisms leading to decreased heart function are unknown. My dissertation focused on molecular mechanisms that regulate the heart's ability to pump blood to the periphery during times of low blood flow. The power generating capacity of single heart muscle cells was measured with conditions mimicking those occurring with low blood flow. In addition, the effects of these conditions were measured in cells containing the fast molecular motor present in young, healthy hearts compared to cells containing the motor associated with aged, diseased hearts. It was found that single myocytes expressing the fast motor were more affected by metabolites associated with low flow conditions, implying a switch in motor expression may be a protective mechanism.

In addition, exercise has been shown to be an effective means to prevent heart disease. I studied how individual muscle cell contraction is affected by endurance exercise training. It was found individual cells had increased contractile abilities after chronic exercise training, possibly contributing to increased heart function following exercise. Overall, this research provides insight into molecular regulators of heart contractility with acute and chronic stress and may provide a molecular basis for alterations with heart disease.