Tension production and sarcomere length in lobster (*Homarus americanus*) cardiac muscles: the mechanisms underlying mechanical anisotropy

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Honors Project Overview

My honors project in the Department of Biology focused on the biomechanical properties of cardiac muscles in the American lobster (*Homarus americanus*). Specifically, I was able to characterize length-tension curves of lobster cardiac muscles, which are defined by the ability of lobster cardiac muscle to generate force when stretched to different lengths by the filling of the heart. With the help of the Grua-O'Connell funding, the Department of Biology was able to purchase a highly sensitive translation stage (Fig. 1), which without, this honor's project would

not have been possible. Thanks to the Grua-O'Connell fellowship, this study presented the first known set of crustacean cardiac length-tension curves. By comparing the lobster cardiac lengthtension curves generated in this study, to mammalian cardiac length-tension curves,

we determined both lobster and mammalian hearts operate at similar locations along the length-tension curve, suggesting that these different hearts respond to similar functional demands.

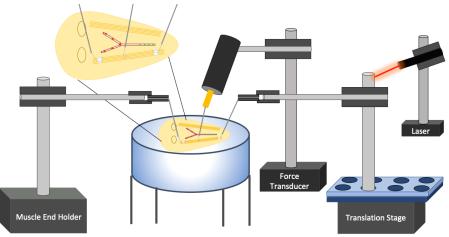


Figure 1: Experimental set-up of length-tension experiments. The heart (yellow) was dissected ventrally and stretched out to expose intracardiac muscles. GluTure glue (white glue patches) was used to fix two ends of a muscle. A third hook was placed in the center of the muscle and was hooked underneath, or placed on top, of the muscle bundle to record both active and passive forces via an optical force transducer. A translation stage lengthened and shortened the muscle bundle at an extension rate of 10 μ m/s. A He-Ne laser was used to measure displacement of the translation stage from rest.

Abstract

With increased activity, the heart's blood volume during diastole increases, which consequently increases the stretch on the walls of the heart. As is typical with striated muscles, the ability of the heart to generate tension increases with increasing stretch up to some maximum. This increase in tension-generating ability may be due to sarcomeres generating greater force at longer lengths on the ascending limb of the length-tension curve. However, lobster hearts are suspended by elastic ligaments and arteries within a pericardial space; thus, the tension on the heart is influenced both by changes in sarcomere length during diastole and by artery stretch during systole. Furthermore, the heart is anisotropic, with characteristically different active and passive forces along the transverse and longitudinal axes. We examined the effects of sarcomere length changes on tension generation by the heart at different points in the cardiac cycle as well as the potential contribution of differences in sarcomere length to the observed anisotropy. Additionally, we determined the length-tension curves for longitudinal and transverse cardiac muscles. Sarcomere length (Fig. 2) experiments showed that no significant differences in sarcomere length were found between longitudinal and transverse fibers. Length-tension curve experiments, revealed that tension increased with increasing muscle length, indicative of a classic Frank-Starling mechanism (Fig. 3).

We also found transverse muscles produced greater contractile forces at a given strain compared with longitudinal muscles. In contrast, longitudinal muscles produced greater passive forces at a given strain compared with transverse muscles. These data corroborate past findings that the lobster heart is mechanically anisotropic. Furthermore, we found

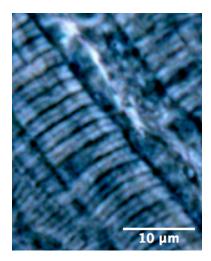


Figure 2: Lobster sarcomeres from left longitudinal muscle group. Sarcomeres were measured as distance between Zdiscs (black lines). Light micrograph at 100X using Olympus BX51.

maximum strain ranges along the transverse and longitudinal axes to be greater in the lobster than in mammals, but smaller than those characterized in fish hearts. Additionally, frequency was found to increase with increasing length in longitudinal muscles; both of which are indicative of Frank-Starling mechanisms. We hypothesize that the lobster heart modulates cardiac output by altering stroke volume and heart rate, but the extent to which is unknown.

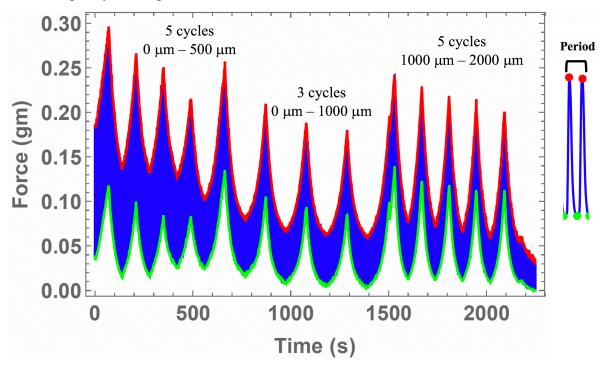


Figure 3: Active force (blue), passive force (green), and total force (red) measured over time in a single lobster, transverse cardiac muscle bundle stretched at varying lengths. Each cycle represents a ramp of increasing and decreasing length. In this particular lobster we see five cycles of lengths from $0 \,\mu\text{m} - 500 \,\mu\text{m}$, three cycles from $0 \,\mu\text{m} - 1000 \,\mu\text{m}$, and five cycles 1000 $\mu\text{m} - 2000 \,\mu\text{m}$. Extension rate was 10 $\mu\text{m/s}$. Period is the time between peaks of the heartbeat; frequency is 1/period.