Investigating Natural Stretch and SGRN-Modulated Changes in Cardiac Muscles of the American Lobster, *Homarus americanus*

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Crustacean hearts provide a comparatively simple model for understanding the mechanics of heart function. As with all hearts, the work done by the heart creates pressure that circulates blood throughout the body. The ability of the heart to do work depends on heartbeat frequency and amplitude. When tissues need more energy, the frequency and/or amplitude of the heartbeat can respond so as to increase the volume of blood being pumped through the system per time, which is the cardiac output. Cardiac output is the product of stroke volume, or the amount of blood being pumped with each beat, and the heartrate (CO = SV x HR).

Changes in heart function can alter the stretch imposed on the heart during contraction and relaxation. Stretch is imposed on the heart in two primary ways: (1) elastic pulls: anterior and posterior pulls by the arteries and lateral pulls by the alary ligaments; and (2) pressure changes: increases in pressure increase stretch of the heart wall. Changes in stretch of the heart wall alter the stretch of the intrinsic muscles of the heart, which, in effect, change the sarcomere length of those muscles, where sarcomeres are the basic contractile units of muscles. In general, up to a point, the longer a sarcomere, the greater the force that is generated by that sarcomere because of greater opportunities for actin-myosin crossbridges to form. However, contraction amplitude also depends on the rate at which muscles are lengthening and shortening. From the general muscle force-velocity curve, we know that in general muscles are able to generate more force as speed of muscle lengthening increases and less force as speed of muscle shortening increases.

The focus of our work in the Johnson lab was grounded in the inquiry of what the characteristic length-tension and force-velocity relationships of the American lobster heart are and how they are changed with inotropy (maximum tension at a given muscle length) as induced by SGRNFLRFamide (SGRN), a neuropeptide produced endogenously in *H. americanus*. We expected SGRN to increase the amplitude of the *H. americanus* heartbeats while decreasing their frequency (Dickinson *et al.*, 2014, Dickinson, Johnson Ellers, Dickinson, 2016). To determine whole heart length-tension and force-velocity relationships, a ventral dissection was performed to expose the heart of each lobster. The posterior artery was tied off and connected to a force transducer and then subjected to lengthening and shortening at four different velocities using an Aurora dual-lever system. Each set of trials was repeated at least three times with hearts perfused with (1) regular lobster saline (pre-control), (2) SGRN lobster saline (SGRN), and (3) regular lobster saline (post-control). Preliminary results show (1) that the lobster heart length-tension curve fits the general pattern observed for cardiac length-tension curves from other species and (2) that SGRN shifts the length-tension curve up (i.e., increases heart contractility; Fig. 1). Analysis of data for the force-velocity curve is still in progress. These results are particularly exciting given that such relationships have not been well-characterized, if at all, in lobsters or other crustaceans.

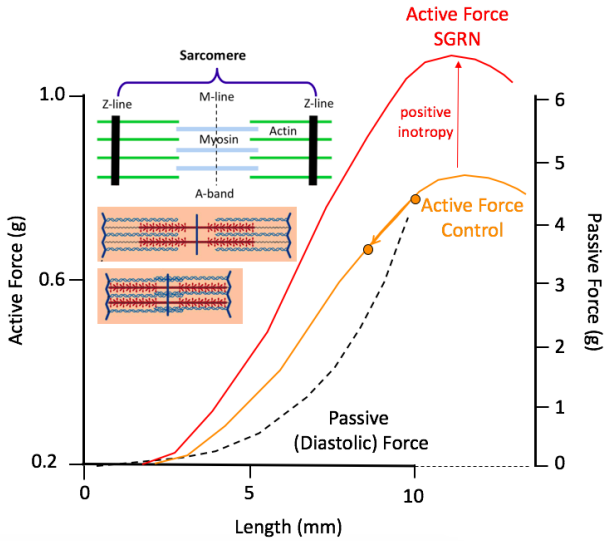

Figure 1. Cardiac-length tension curve which shows that as length of cardiac muscles increase, the forces generated by the heart increase. Bottom two sarcomeres from *Life: The Science of Biology, 9th Ed.*

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