Interaction of crustacean myosuppressin (pQDLDHVFLRFamide) and stretch in the *Homarus americanus* cardiac muscle

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Rhythm-producing networks, called central pattern generators (CPGs), are circuits of neurons that generate rhythmic output without requiring sensory input. Through their interactions, the neurons in these networks produce a periodic output that controls rhythmic biological functions. The cardiac system of the American lobster *Homarus americanus* is a much-studied model for understanding CPGs. The CPG of a lobster’s heart, the cardiac ganglion, drives the lobster’s rhythmic heartbeat and can be modulated in several ways, including stretch feedback and neuromodulation.

To understand the mechanism behind stretch feedback in the lobster heart, it is pertinent to understand how the effects of stretch are modulated by other influences, particularly by neuropeptides (Cooke, 2002). Many neuropeptides are known to alter the lobster’s heartbeat. Among these, the neuropeptide crustacean myosuppressin (pQDLDHVFLRFamide) has been extensively studied. For example, it is known that at a constant, slightly stretched length, myosuppressin increases force and decreases frequency of heart contractions (Steven, et al., 2009). The present study investigated the effect of stretch feedback on neuropeptide action in the lobster heart by administering myosuppressin while mechanically stretching the muscle.

Stretch has been shown to alter the frequency of heartbeats under some conditions (Fig. 1). This study showed there was a significant change in frequency at the rate and extent of stretch for both control saline and myosuppressin (Fig. 2). Data from these experiments suggest myosuppressin alters the relationship between stretch and contraction force.

Since myosuppressin alters not only the cardiac muscle, but also the motor output from the cardiac ganglion, additional experiments used a preparation in which the endogenous neural output of the ganglion was replaced with stimulation controlled by the experimenter; this enable us to determine that myosuppressin alters stretch feedback at the level of the neuromuscular junction.

We found that myosuppressin modulates stretch feedback pathway to enhance contraction amplitude and diminish contraction frequency, and modulates the neuromuscular junction to decrease passive tension. This study revealed that myosuppressin modulates contraction frequency through a unique feedback pathway in the cardiac ganglion to decrease contraction frequency, and suggests that myosuppressin acts as an inhibitor in the stretch feedback pathway.
Figure 1. Studies have shown there are two possible sites of modulation in the central pattern generator of the *H. americanus* lobster heart. These include changes at the level of the cardiac ganglion, and the level of the cardiac muscle, particularly the neuromuscular junction.
Figure 2. Application of $10^{-7}$ M myosuppressin during a 400 second ramp period in *H. americanus* increased active force of contractions. (A) Contraction active force and stretch extension during stretch pyramids for hearts perfused with saline. (B) Contraction active force and stretch extension during stretch pyramids for heart perfused with 10-7 M myosuppressin.
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References


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