

**Determining the peripheral site at which myosuppressin modulates cardiac contractions in the  
American lobster, *Homarus americanus*  
Isabel Petropoulos, Class 2023**

Central pattern generators (CPGs) are neural circuits that, when activated, generate rhythmic behaviors and patterns. These complex circuits have the capacity to send outputs that control behaviors such as swimming, walking, and breathing (Marder & Bucher 2001). In the absence of other inputs, CPGs produce consistent patterns as the networks are relatively fixed. However, modulation of CPGs creates variations in motor patterns, which gives organisms behavioral flexibility. Neurohormones and neurotransmitters such as amines and neuropeptides are molecules that can modulate CPGs, thereby enabling organisms to respond to changes in the environment (Brezina et al., 2010). It has been shown that neuromodulators not only have effects on CPGs, but also alter muscle contractions by acting directly on the neuromuscular junction or muscle itself. The neurogenic lobster heart requires neural impulses to contract, and these impulses are controlled by a CPG called the cardiac ganglion (CG) (Cooke, 2002).

Myosuppressin is a peptide that has been shown to exert modulatory effects on the cardiac neuromuscular system. In the isolated cardiac ganglion, myosuppressin increased burst duration and decreased cycle frequency and thus, the duty cycle of the action potential bursts. In a whole heart preparation, myosuppressin increased contraction force (Stevens et al., 2009). A previous student in the Dickinson lab investigated whether myosuppressin exerted its effects at the neuromuscular junction (NMJ) and did not find any significant effects, which suggests that myosuppressin exerts its peripheral effects at the cardiac muscle. Thus, this summer I asked whether myosuppressin affects the cardiac muscle of the American lobster, *Homarus americanus*.

To answer this question experimentally, I performed physiological experiments. Because the cardiac ganglion drives the heart contractions, it was removed, so that the cardiac muscle was isolated from the cardiac ganglion; I then experimentally stimulated muscle contractions using L-glutamate, the neurotransmitter used by cardiac motor neurons, and measured the resultant muscle contractions. L-glutamate ( $5 \times 10^{-4}$  M) was administered using a syringe pump, which was connected to a micropipette that was positioned to puff glutamate onto one of the lateral muscles on the heart (the left or right muscle across a few muscle fibers). Two hooks were glued perpendicularly to either side of the muscle to stabilize the muscle and give a slight amount of stretch. A force transducer was placed between the two hooks to record the muscle contractions. Saline was continuously perfused across the heart while control puffs of glutamate were administered. Once the glutamate-evoked muscle contractions stabilized in size, myosuppressin ( $10^{-6}$  M) was perfused over the muscle for 30 minutes while glutamate was puffed at regular intervals. Subsequently, the preparation was washed with lobster saline to ensure responses returned to baseline. My data suggests that myosuppressin caused significant increases in the amplitude of glutamate-evoked contractions in the isolated cardiac muscle relative to those evoked by glutamate while lobster saline was perfused (one sample t-test,  $p=0.0111$ ;  $N=7$ ). The width of contractions and baseline did not change significantly when myosuppressin was applied (compared to control in lobster saline) (one sample t-test,  $p=0.5769$ ;  $N=7$ ) (one sample t-test,  $p=0.8381$ ;  $N=7$ ).

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