A Game of Operation: Investigating the Modulatory Effects of Myosuppressin Isoforms on the *Homarus* Cardiac Ganglia

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The nervous system is responsible for manipulating our behaviors, like from walking to running. These changes are the result of generated rhythmic movements controlled by central pattern generators (CPGs). For example, the *Homarus americanus* cardiac ganglion (CG), a central pattern generator, is responsible for the American lobster heartbeat. The *Homarus* CG is a simple model system for investigating the underlying mechanisms in generating rhythmic movements such as the heartbeat, as it contains only nine neurons and does not require outside sensory input to function. This is incredibly important as its study may elucidate the roles of CPGs in more complex biological systems. Moreover, because the manipulation of behavior itself require regulation, the study of the *Homarus* CG also allows the investigation of the modulations of neuropeptides responsible for the CG's behavior. In other words, study of these modulations may offer insight into how the modifications of chemical structures ultimately alters biological behavior.

Considering this, this study investigated the modulatory effects of three chemical isoforms endogenous to the *Homarus americanus* on the *Homarus* CG. The goal of this study was firstly to explore how chemical modifications altered on the CG's behavior, secondly to how these behavioral changes occur. To do this, myosuppressin isoforms were perfused across the *Homarus* CG, and their effects on the CG's behavior, specifically burst duration and cycle frequency, were analyzed. Note, a burst refers to the phenomenon of action potentials firing rapidly, whereas burst duration is the timing in which it takes for one burst to fire, and cycle frequency describes how many bursts occur in one second. Currently, results suggest both the 'myosuppressin full' and 'myosuppressin no pGlu' isoforms to significantly increase burst duration, and decrease in burst cycle frequency in the *Homarus* CG. Conversely, the nonamidated myosuppressin isoform does not significantly affect burst cycle frequency, but has revealed a slight decrease in burst duration instead (n=13). With this, experimental data has also confirmed that the number of action potentials spikes is positively correlated with the burst duration as shown below. In order to deduce how these isoforms cause these behavioral changes, this project is now beginning to analyze how these isoforms interact with the CG on a cellular level, and will be continued this Fall. Moreover, while also increasing both sample size and varying the concentrations of the myosuppressin isoforms, I hope to discern how these chemical modifications alter biological behavior, and how simple networks such as the *Homarus* CG, generate rhythmic movements such as the lobster heartbeat.

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