Temperature dependencies of myosuppressin modulation of the cardiac neuromuscular system of the American lobster, Homarus americanus

Stephanie Lemus, Class of 2025

American Lobsters inhabit a large oceanic range where temperature can vary by as much as 25°C, and daily temperature can fluctuate up to 12°C at a single location. The lobster cardiac neuromuscular system is sensitive to these temperature changes, as lobsters cannot regulate their own body temperature. Modulation of neuron and muscle function by amines and peptides is critical to the circuit functioning of the lobster cardiac system to help the organism adapt to and survive in any environment. Recent data has shown that neuromodulation is itself modulated as a function of temperature, yet the extent to which this is true is still relatively unknown (Marder and Rue, 2021). Studying this phenomenon in the lobster may reveal insights into the mechanisms that allow these circuits to function when ambient temperature is constantly changing.

To better understand how temperature affects the modulation of the cardiac system by neuromodulation in the American lobster, I focused my research on the lobster heart. The lobster heart is neurogenic and controlled by the cardiac ganglion central pattern generator (CPG). CPGs are neural circuits that generate rhythmic motor patterns like heartbeats, so the beating rhythm is set through nerve impulses. Previous studies have shown that with increasing temperature, the heartbeat frequency shows a biphasic pattern in which it initially increases before decreasing rapidly while contraction force mostly just decreases. Similar to temperature, myosuppressin exerts its effects in the frequency and amplitude of the lobster heart. The peptide myosuppressin is endogenous to the American lobster and acts at the cardiac muscle as well as in the nervous system itself to elicit whole heart responses. With the application of myosuppressin, crash temperature (the temperature at which the heart stops functioning) is lower when perfused with the modulator than not when exposed to elevated temperatures. Because the peptide reduces heart resiliency to higher temperatures, the peptide was expected to negatively affect the frequency and amplitude of heart contractions, yet that is not the case according to preliminary studies. Myosuppressin does not only decrease the frequency at which neurons fire, but it also increases the amplitude of the contractions after a short decrease in force contraction. This may suggest that there must be an excitatory effect on the muscles of the neuromuscular system versus an inhibitory effect on the neuron to decrease frequency.

My project focused on the contraction force and amplitude of the heartbeat under modulation in response to different temperatures. To do this, I first dissected the lobster heart. I covered and perfused it with physiological saline to allow the heart to continue to beat outside of its body. To record the muscular contraction of the heartbeat, I tied the anterior arteries to a force-displacement transducer. Then, I fit a temperature probe inside the ostium located along the lateral sides of the heart to record the internal temperature. In my project, temperature was increased and held for at least 45 minutes to explore how temperature alters the effects of myosuppressin in the lobster heart. The temperatures used for the experiments were 7, 10, 13, and 16°C. For each experiment, baseline function at each of these temperatures was recorded as the control for 5 minutes. Then, myosuppressin was perfused into and over the heart at that same temperature for 10 minutes. Lastly, perfusion was switched back over to the saline to let it wash out the neuromodulator for 30 minutes.

All temperatures followed a similar pattern: there was an initial dip in amplitude right after the application of myosuppressin followed by a rapid increase in contraction force. The period seemed to increase rapidly before decreasing and regulating soon after for all temperatures. After using separate graphs to plot the mean and standard deviation of average amplitude and period at the 1st minute of the control, 2 minutes into the myosuppressin application, 5 minutes in, and 10 minutes in as a function of temperature instead of time, I was able to see the rate of change of the modulation process in response to a 3°C increase to temperature for amplitude and period. The rate at which these attributes changed from one temperature to the next was not consistent. Additionally, it was clear that the rate at which temperature changed amplitude and period of the heartbeat was inconsistent between both graphs, which tells us that the temperature dependencies of the amplitude and the period under the modulation of myosuppressin are different. In other words, temperature affects amplitude and period at different rates. This is interesting given that these changes are seen in 3°C increments while they live in environments that can fluctuate up to 12°C at a given time. Future experiments will use this data to further determine how the CPGs of the cardiac system of the lobster heart deal with the natural modulation of their neuromuscular system while also navigating the differing effects temperatures seems to have on the system.

Faculty Mentor: Daniel Powell
Funded by the Henry L. and Grace Doherty Charitable Foundation Coastal Studies Research Fellowship

References:

Marder, E., Rue, M.C.P. (2021). From the Neuroscience if Individual Variability to Climate Change. The Journal of Neuroscience. 41(50):10213-10221. 10.1523/JNEUROSCI.126-21.2021.