The Role of Nitric Oxide Feedback in the Thermally Robust Lobster Cardiac System

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The electrical and chemical complexity of the neuron makes it very sensitive to small changes in temperature. American lobsters are poikilothermic animals, meaning that they cannot internally regulate their body temperature. Therefore, the body temperature is controlled by the local water temperature, which often shows rapid changes of over 10°C due to tidal currents (Manning 2009). As a result, the neuronal system that controls the heart has adapted to operate at a wide range of temperatures despite the thermal sensitivity of individual ion channels.

The heartbeat of a lobster is locally generated by a simple network of 9 neurons called the cardiac ganglion (CG). Although the CG is able to produce a rhythm in the absence of external input, the activity of the CG has been shown to be modulated by nitric oxide. The CG is inhibited by the nitric oxide released by an active heart in a negative feedback loop. This negative feedback loop mirrors the countless examples of negative feedback loops in biology that moderate processes and maintain stability.

It has been shown that the heating of a lobster from 2°C to 20°C results in an increase in heart rate. Within this range, a lobster counteracts the heart rate increase by decreasing the strength of the ganglion signal to the cardiac muscle. This causes the heart to pump less blood with every stroke. However, as temperatures exceed 20°C, the cardiac system fails, and the heart rate rapidly decreases before stopping all together (Camacho et al. 2006). Although you may expect the negative feedback from heart derived nitric oxide to stabilize the ganglion, it has been shown that the isolated CG is able to sustain activity at higher temperatures than the heart/ganglion system (Owens 2014). The mechanisms that underlie the discrepancy between the thermal stability of the whole heart and the isolated ganglion are currently unknown. One hypothesis, which we propose to test, is that the nitric oxide generated by the heart increases temperature sensitivity of the ganglion.

To begin studying the effects of temperature ramps on the lobster cardiac system we subjected hearts to four successive temperature ramps noting the changing frequency of the heart rate and force of contraction. We found that there was a rise in heart rate during the first half of the ramp followed by a plateau then a precipitous drop in frequency during the crash. Additionally, we found that the force of contraction was relatively stable for the beginning of the temperature ramp, then the amplitude of the force began decreasing at an increasing rate into the crash. When comparing the crash temperature at the successive ramps, we found that there was, on average, a slight decrease in crash temperature across the successive ramps on the same heart.

We also began studying the effects of nitric oxide using L-NA, a molecule that blocks the enzyme that produces nitric oxide. We ran two temperature ramps with saline as a control, then ran two temperature ramps with L-NA to see how removing the nitric oxide feedback affects the cardiac system. Due to the natural variation between the physiologies of the individual lobsters, there was a wide range of crash temperatures. Therefore, the control saline ramps are necessary to show the baseline crash temperatures of each heart without interfering with nitric oxide feedback. The initial results of these tests show that the crash temperature appears to not change significantly due to the removal of the nitric oxide feedback. However, when accounting for the slight decrease in crash temperature observed when running successive temperature ramps, there is a significant increase in crash temperature associated with removing nitric oxide feedback. Although this finding is inconsistent with the hypothesis that nitric oxide feedback has a stabilizing role in the cardiac system, this finding provides some explanation of the results from Owens' thesis. It suggests that nitric oxide feedback may be destabilizing the lobster cardiac system: the lack of nitric oxide feedback in the isolated CG contributes to its increased thermal stability found in Owen's thesis.

In the fall, I will continue this work by replicating the previous experiments while directly recording the electrical activity of the ganglion to explore the mechanism of the hearts response to nitric oxide and thermal stress.

Faculty Mentor: Patsy Dickinson

Funded by the Bowdoin Life Sciences Fellowship

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