Effects of temperature on the modulation of a small motor network
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This summer, my project worked to examine the interface between the effects of temperature to a nervous system globally against its effects on individual neuron processes. Previous studies have shown that individual neuronal processes change dissimilarly from one another. Because of this, my project aimed to explore if there are mechanisms that enable temperature stability within environments in which temperatures fluctuate, building on the overarching question – are there features of biology that enable system stability and stable output across temperature fluctuations?

In order to examine this further, I utilized the heart of the American lobster, chosen due to its well defined neuron circuit and the unique ability of the heart to maintain output in vitro as long as it is perfused with saline. Utilizing the whole heart, I worked to examine the effects of temperature on the modulation of the heart by specific neuromodulators across two parameters: contraction force and cycle period. Neuromodulators are substances that can change synaptic dynamics or the excitability of neurons. For my experiment, I utilized three neuropeptides (MYO, SGRN, and GYS) endogenous to the lobster that when applied, cause increases in contraction force of the lobster heartbeat. Previous studies examined neural responses to various modulators on the lobster heart without examining the role of temperature. Here, I aimed to understand whether the modulatory processes themselves are temperature-dependent. Specifically, I worked to investigate whether neuromodulators known to increase contraction force of the lobster heart aid in providing system stability to increasing temperatures through increased activation of the cardiac muscles of the whole heart. It was hypothesized that neuromodulation enables neurons and muscles to maintain activity despite temperature changes.

In order to test this, the whole heart was dissected out and pinned in a Sylgard 170-coated dish ventral side up. The anterior arteries of the heart were tied off to a force transducer that converted each contraction force into a heartbeat using Spike2. The posterior artery of the heart was cannulated with a short piece of metal tubing to allow perfusion of saline over the heart. A temperature ramp was performed across 7°C to 16°C with temperature steps of 3°C. Once the prep was on the rig, a 60 minute saline wash was conducted at 10°C. After the control wash established the lobster's baseline, the temperature was moved down to 7°C. At a stable 7°C, a 10 minute acclimation period was begun to allow the heart to stabilize after the temperature change. This was then followed by a 20 minute neuropeptide application. After each application period, a 60 minute saline wash followed. These steps were repeated for each 3°C temperature increment.

Each peptide's respective modulation was found to provide temperature compensation in regards to contraction force as temperature increased. However, MYO provided temperature compensation differently as the modulator brought about increases in contraction force that were proportional to increases in temperature unlike the other two peptides. In regards to cycle period, SGRN modulation causes an increase in cycle period at cooler temperatures while GYS modulation of cycle period was found to be temperature dependent. MYO modulation was found to decrease cycle period as temperature increases. Overall, the data highlights that each of the tested modulators enable the nervous system to regulate its output as temperature changes. In the end, it is possible that the nervous system is able to regulate its output through varying degrees of neuromodulator release as different levels of neuromodulators would produce unique responses of the nervous system.

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