Network responses to changes in extracellular saline concentration and temperature in the lobster *Homarus americanus* Katrina Carrier, 2024

As a result of climate change, salinity levels in the ocean are becoming more extreme and ocean temperatures are rising. Changes in the water cycle, including changes in precipitation and evaporation, mean that some regions are becoming less salty while others become saltier. The Atlantic Ocean is experiencing a basin-wide increase in salinity (Sathyanarayanan et al., 2021). As such, the individual and combinatorial effects of temperature and salinity levels on the nervous system of the American lobster are of interest as these environmental trends progress. Generating data quantifying these effects provides insight and allows us to make predictions about how these environmental perturbations will influence the physiology of the lobster.

To measure the effects of these environmental perturbations on the nervous system of the lobster, I focused my research on the stomatogastric nervous system of the lobster *Homarus Americanus*. The stomatogastric nervous system (STNS) controls the motion of the crustation foregut and is controlled by the stomatogastric ganglion (STG) (Marder & Bucher, 2007). Motor neurons contained in the STNS control two central pattern generators (CPGs) which are neural circuits that produce rhythmic output even in the absence of a rhythmic input. My focus this summer was on the pyloric rhythm, the CPG which carries out the filtration of the food that the lobster consumes. The pyloric rhythm is a triphasic motor pattern that is continually expressed *in vitro* and is a robust and well-characterized pattern, which makes it a good model for understanding the effects of environmental perturbations on its function.

To determine the effects of temperature and salinity on this system, I began by establishing the temperature range and the range of salinities that the STNS can withstand. To do so, the stomach of the lobster was dissected, then the STNS was dissected off the stomach. Extracellular recordings of the electrical activity of individual nerves were then used to measure the effects of temperature and saline on the neuronal output of the STNS. To measure the effects of temperature by 2 degrees every 2 minutes until the pyloric rhythm was identified as having ceased function. To measure the effects of salinity, the STNS was perfused with saline with the desired altered concentrations and the functionality of the pyloric rhythm was observed.

The highest temperature that the system can withstand was established in order to determine if altered saline concentration alters this maximum temperature. On average, the STNS was able to maintain function when exposed to temperatures reaching 19 C. To determine if this number will change at different saline concentrations, it was necessary to establish increased and decreased concentrations that the system can still tolerate. We had shown in previous experiments that the STNS is able to maintain function with salinity at a 0.75x concentration, but that it is not able to withstand salinity concentrations as high as 1.5x. We needed to establish an increased concentration that the STNS could withstand. The results of my experiments demonstrate that the system is robust at a saline concentration of 1.25x. Future experiments will use this information to determine whether combinatorial changes in temperature and salinity levels change the limits for each individual perturbation when they occur together.

Faculty Mentor: Patsy S. Dickinson

Funded by the Henry L. and Grace Doherty Charitable Foundation Coastal Studies Research Fellowship

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