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Neural Circuit Resilience in the Lobster Cardiac Ganglion

Over the summer, I conducted research in Professor Powell's neuroscience lab focusing on the cardiac ganglion (CG) of lobsters. The CG is a small but well-defined neural circuit responsible for generating rhythmic motor output that drives the heartbeat. Its structural clarity and reliability make it an excellent model for studying how neural circuits respond to environmental stressors. In my case, my work studied how the physiology of the CG adapts to one perturbation, changes in salinity, while simultaneously being exposed to another: increasing temperature.

To investigate this interaction, I used temperature ramps to determine the "crash temperature" of the CG, or the point at which rhythmic firing ceases. Preparations were then refrigerated for approximately 24 hours, which allowed me to assess salinity as a stressor across different timescales. In the short term, altered salinity produces immediate ionic shifts that can affect neuronal firing and rhythm stability. Over longer exposures, salinity drives physiological changes at the cellular level, particularly in ion channel function and expression, which may make the circuit either more or less resistant to rising temperatures. By manipulating salinity and then applying temperature ramps, I essentially examined how these short- and long-term responses interact to shape the CG's tolerance to heat stress.

The experiments highlighted some suggestive patterns, but the outcomes were limited by the small number of preparations completed in each salinity condition. While not entirely conclusive, the preliminary data indicated that crash temperature may vary depending on salinity levels. That being said because the dataset was small, no firm conclusions could be drawn, but the work established a foundation for understanding how salinity and temperature together affect CG function.