Many organisms have central pattern generators; these sets of neurons regulate motor functions such as walking, respiration and heartbeat. In the Dickinson lab we specifically study the cardiac ganglion (CG) of *Homarus americanus*, the American lobster, which is a central pattern generator that regulates heartbeat. It consists of two sets of neurons: four small pacemaker neurons and five large motor neurons. These nine neurons are electrically and chemically coupled. Together, they regulate the rhythm of the lobster’s heartbeats.

The Dickinson lab has been exploring what happens when the CG is given a tonic (continuous) stretch, as well as phasic stretches (repeated short stretches). Curiously, they have seen two very different responses under continuous stretch. If the CG had short burst durations then there was little to no change, but if the CG had longer burst durations then burst duration shortened and frequency of bursts increased. When given phasic stretches, some CG’s would be successfully entrained (the bursts would sync up with the stretches) whereas others would not be.

My project this summer was to use mathematical modeling to begin to figure out what the mechanism behind this stretch response is. It is known that there are stretch gated ion channels in lobsters, and so I incorporated them into a mathematical model of the CG. The model I started with is a system of differential equations developed by Alex Williams, a former student in the Dickinson lab. It is unknown which ion or ions pass through the stretch gated channels, thus I ran simulations for a number of different reversal potentials (reversal potential is dependent on the particular ion). I then compared the data from these simulations to the data found experimentally.

Unfortunately, no particular value for reversal potential created a model that functioned just like the experimental data. Some values did accurately resemble aspects of what was seen in the lab though, which could indicate that something more complicated than just one type of stretch gated ion channel is involved. Another possibility is that the model I started with was too simple to effectively study burst duration with. Williams built his model from a pair of Morris-Lecar oscillators, which are the simplest canonical model of the neuron. One future direction, which I plan to work on as an independent study project this fall semester, is to attempt to optimize the current model by adjusting the kinetics of the opening and closing of the stretch gated channels to better reflect what is seen experimentally. Another direction, which I also hope to work on, is to create a new model for the CG using a more complicated model of the neuron than the Morris-Lecar oscillator as a foundation. Now that I have spent a summer developing and learning the tools necessary, hopefully I will be able to make much more progress this semester.

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