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Does the neuropeptide GYS modulate stretch feedback pathways in the lobster cardiac neuromuscular system?

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Tricia Hartley- Summer 2014 Summary

In many animals, there are groups of neurons, known as central pattern generators (CPGs), which are capable of controlling major everyday life functions. CPGs are responsible for functions that require patterned rhythmic activity, such as the heartbeat, digestion and locomotion. A CPG called the cardiac ganglion, consisting of only nine neurons, controls the rhythmic beating of the heart of the American lobster, Homarus americanus, by stimulating the muscle cells of the heart.

My summer consisted of two separate projects in Patsy Dickinson’s neurophysiology lab, both studying the interaction of the cardiac ganglion with neuropeptides. These neuropeptides, GYSDRNYLRFamide (GYS) and SGRNFLRFamide (SGRN) are released hormonally into the cardiac neuromuscular system. The overarching goal of both projects was to determine the role of these neuropeptides in the lobster’s cardiac neuromuscular system.

For my first project, I studied the interaction of the neuropeptide GYS with the stretch receptors of the lobster heart. Previous research has found these stretch receptors to be a form of excitatory feedback from the lobster heart to the cardiac ganglion, as heartbeat amplitude and frequency increase as heart is stretched. Further, the dendrites along the cardiac ganglion have been found to be stretch-sensitive, meaning when these dendrites were cut, this excitatory response is no longer observed. By stretching the heart with the dendrites intact and with GYS and next when the dendrites were cut and with GYS, the goal of this project was to determine if GYS would alter the feedback of the stretch receptors back to the cardiac ganglion to change heartbeat frequency and amplitude. Unfortunately, the intricacy involved in being able to cut the dendrites while allowing the heart to continue to beat proved very difficult and I moved on to my next project.

The goal of my next project was to examine the interactions of the neuropeptides GYS and SGRN with the decreased and increased presence of nitric oxide, the second form of feedback from the heart muscle to the cardiac ganglion. Previous research shows nitric oxide as having an inhibitory effect, decreasing heartbeat amplitude and frequency. By applying both GYS and SGRN to both the isolated cardiac ganglion and the whole heart in the presence of both a nitric oxide inhibitor and donor, the hope is to be able to determine the interaction of these peptides with and without the presence of the feedback of nitric oxide. Because I started this project later in the summer, with the assistance of Sophie Janes’ data, I have been able to look at the effects of GYS on the whole heart, in addition to the combination of GYS with L-NA, a nitric oxide inhibitor. So far, the data has shown that the combination of GYS with L-NA causes less of a decrease in heartbeat frequency than GYS alone, which shows a significant decrease. We predict this is because GYS enhances the nitric oxide pathway, while L-NA is blocking the nitric oxide pathway, thus giving insight into the role of GYS within the lobster’s cardiac neuromuscular system. For my senior independent study I hope to continue this research and be able to continue to compile data for both SGRN and GYS on the isolated cardiac ganglion as well as on the whole heart, with a nitric oxide inhibitor and donor.