Mechanisms underlying variable responses to isoforms of the neuropeptide C-type allatostatin (AST-C) in the American lobster,

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My honors research centers around understanding the mechanism underlying the variable physiological responses to the perfusion of AST-C isoforms through the lobster heart. My hypothesis is that amidation of AST-C isoforms may influence binding to four putative AST-C receptors, thus causing the varying physiological responses we see. I came to Arizona with the hopes of performing AST-C receptor binding studies at the USDA-ARS lab in Maricopa, AZ to see if amidation was indeed influencing receptor binding. These studies are an important addition to my honors research and cannot be conducted at Bowdoin due to the lack of resources in that particular area. However, due to the government shut down, the USDA lab was closed for the entirety of my visit to Arizona. Therefore, I was not able to conduct receptor binding experiments. Nevertheless, I was able to make the best of my time in Arizona and make progress on my honors work as well as learn vital computer based lab skills from our collaborators, Andy Christy from the Békésy Laboratory of Neurobiology at the University of Hawaii at Manoa, and Joe Hull from the USDA-ARS lab in Maricopa, Arizona.

My first task was to analyze my physiological data from lobster whole heart perfusions of AST-C amidated and non-amidated isoforms and to re-analyze previous Bowdoin students data so as to maintain a consistent data analysis method. Once all 47 data sets were compiled, I ran statist (i) o2(t) -4.6(e) 9.4(d) hypothesis, and

suggest that amidation is not responsible for the differences in cardiac responses across peptides.