Peptide hormones in the C-type allatostatin family modulate the cardiac neuromuscular system of the American lobster *Homarus americanus*

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The C-type allatostatin (C-AST) family is a group of pleiotropic neuropeptides originally described from holometabolous insects. Recent transcriptome/genome mining has shown that the AST-Cs are also broadly conserved within the Crustacea. Despite their well-characterized bioactivity in insects, the roles of these peptides in crustaceans remain largely unknown.

Here, we compared the distribution and bioactivity of the C-AST (pQIRYHQCYFNPISSF) and C-AST-like peptide (SYWKQCAFNAVSCFamide) in the American lobster *Homarus americanus*. Molecular cloning and immunohistological labeling suggested that the two peptides were encoded from different mRNAs and localized within distinct populations of neurons/release terminals in the stomatogastric nervous system and pericardial organ, but were not present in the cardiac ganglion (CG). Application of either peptide to the isolated whole heart decreased the frequency of ongoing contractions and of action potential bursts recorded on motor nerves. Frequency was similarly reduced by C-ASTs in the isolated cardiac ganglion (CG), suggesting that modulation of CG output is largely responsible for the decrease in heart rate.

C-ASTs also alter contraction amplitude: amplitude is increased in some lobsters and decreased in others. This could result from differential changes in the motor output or differential modulation of the myocardium/neuromuscular junction. To test the latter possibility, we stimulated a motor nerve with fixed burst parameters at constant frequency. The peptides did not affect 86% of preparations, suggesting that modulation is central rather than peripheral. In lobsters in which contraction amplitude increased, motor neuron burst duration and spikes/burst increased in both the intact heart and the isolated CG, suggesting that modulation of the ganglion is responsible for the increased amplitude. However, in lobsters in which contraction amplitude decreased, burst parameters did not decrease, suggesting that such modulation is not responsible for the decreased contraction amplitude. Instead, this decrease appears to result from a decrease in facilitation due to the decreased heart rate.

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