

Abstract

C. elegans CASY-1 is a cadherin-like type-I transmembrane protein which has been shown to be associated with defects in multiple forms of associative learning. In this study, I characterize the defects in locomotion behavior of *casY-1* mutants both in the absence and presence of food. I show that *C. elegans casy-1* exhibits behavioral deficits similar to mutants defective for neuropeptide signaling and release. Based on my findings and previous work, I propose a model based on which CASY-1 functions to regulate neuropeptide and neurotransmitter release from the sensory neurons. In *casY-1* mutants, disrupted neuronal signalling from the sensory neurons leads to altered activity in the locomotor circuit resulting in the behavioral defects that I observe.