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Title:	Control of Locomotory Behavior of <i>Caenorhabditis elegans</i> by the Immunoglobulin Superfamily Protein RIG-3
Authors:	Bhardwaj, Ashwani (/jspui/browse?type=author&value=Bhardwaj%2C+Ashwani) Pandey, P. (/jspui/browse?type=author&value=Pandey%2C+P.) Babu, Kavita (/jspui/browse?type=author&value=Babu%2C+Kavita)
Keywords:	Immunoglobulin Proteins <i>Caenorhabditis elegans</i>
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Abstract:	Cell surface immunoglobulin superfamily (IgSF) proteins play important roles in the development and function of the nervous system . Here we define the role of a <i>Caenorhabditis elegans</i> IgSF protein, RIG-3, in the function of the AVA command interneuron. This study reveals that RIG-3 regulates the abundance of the glutamate receptor subunit, GLR-1, in the AVA command interneuron and also regulates reversal behavior in <i>C. elegans</i> . The mutant strain lacking <i>rig-3</i> ( <i>ok2156</i> ) shows increased reversal frequency during local search behaviors. Genetic and behavioral experiments suggest that RIG-3 functions through GLR-1 to regulate reversal behavior. We also show that the increased reversal frequency seen in <i>rig-3</i> mutants is dependent on the increase in GLR-1 abundance at synaptic inputs to AVA, suggesting that RIG-3 alters the synaptic strength of incoming synapses through GLR-1. Consistent with the imaging experiments, altered synaptic strength was also reflected in increased calcium transients in <i>rig-3</i> mutants when compared to wild-type control animals. Our results further suggest that animals lacking <i>rig-3</i> show increased AVA activity, allowing the release of FLP-18 neuropeptide from AVA, which is an activity-dependent signaling molecule. Finally, we show that FLP-18 functions through the neuropeptide receptor, NPR-5, to modulate reversal behavior in <i>C. elegans</i> .
URI:	<a href="https://www.genetics.org/content/214/1/135">https://www.genetics.org/content/214/1/135</a> ( <a href="https://www.genetics.org/content/214/1/135">https://www.genetics.org/content/214/1/135</a> ) <a href="http://hdl.handle.net/123456789/3456">http://hdl.handle.net/123456789/3456</a> ( <a href="http://hdl.handle.net/123456789/3456">http://hdl.handle.net/123456789/3456</a> )
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