Title:	Regulation of WNT signaling at the neuromuscular junction by the immunoglobulin superfamily protein RIG-3 in caenorhabditis elegans
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Abstract:	Perturbations in synaptic function could affect the normal behavior of an animal, making it important to understand the regulatory mechanisms of synaptic signaling. Previous work has shown that in Caenorhabditis elegans an immunoglobulin superfamily protein, RIG-3, functions in presynaptic neurons to maintain normal acetylcholine receptor levels at the neuromuscular junction (NMJ). In this study, we elucidate the molecular and functional mechanism of RIG-3. We demonstrate by genetic and BiFC (Bi-molecular Fluorescence Complementation) assays that presynaptic RIG-3 functions by directly interacting with the immunoglobulin domain of the nonconventional Wnt receptor, ROR receptor tyrosine kinase (RTK), CAM-1, which functions in postsynaptic body-wall muscles. This interaction in turn inhibits Wnt/LIN-44 signaling through the ROR/CAM-1 receptor, and allows for maintenance of normal acetylcholine receptor, AChR/ACR-16, levels at the neuromuscular synapse. Further, this work reveals that RIG-3 and ROR/CAM-1 function through the β-catenin/HMP-2 at the NMJ. Taken together, our results demonstrate that RIG-3 functions as an inhibitory molecule of the Wnt/LIN-44 signaling pathway through the RTK, CAM-1.
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