Title:	Wnt Secretion Is Regulated by the Tetraspan Protein HIC-1 through Its Interaction with Neurabin/NAB-1
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Keywords:	Wnt Secretion C. elegans claudin F-actin
Issue Date:	2018
Publisher:	Elsevier B.V.
Citation:	Cell Reports, 25(7). pp. 1856-1871.
Abstract:	The aberrant regulation of Wnt secretion is implicated in various neurological diseases. However, the mechanisms of Wnt release are still largely unknown. Here we describe the role of a C. elegans tetraspan protein, HIC-1, in maintaining normal Wnt release. We show that HIC-1 is expressed in cholinergic synapses and that mutants in hic-1 show increased levels of the acetylcholine receptor AChR/ACR-16. Our results suggest that HIC-1 maintains normal AChR/ACR-16 levels by regulating normal Wnt release from presynaptic neurons, as hic-1 mutants show an increase in secreted Wnt from cholinergic neurons. We further show that HIC-1 affects Wnt secretion by modulating the actin cytoskeleton through its interaction with the actin-binding protein NAB-1. In summary, we describe a protein, HIC-1, that functions as a neuromodulator by affecting postsynaptic AChR/ACR-16 levels by regulating presynaptic Wnt release from cholinergic motor neurons. Tikiyani et al. demonstrate that a tetraspan protein, HIC-1, maintains the actin cytoskeleton in C. elegans motor neurons. Loss of hic-1 causes enhanced Wnt secretion from these motor neurons. The findings further reveal mechanisms of Wnt secretion, which is aberrant in certain neuroinflammatory disorders.
Description:	Authors sequences are not necessary in order
URI:	https://www.sciencedirect.com/science/article/pii/S2211124718316425 (https://www.sciencedirect.com/science/article/pii/S2211124718316425) http://hdl.handle.net/123456789/1704 (http://hdl.handle.net/123456789/1704)
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