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A role for alpha-adducin (ADD-1) in nematode and human memory

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Identifying molecular mechanisms that underlie learning and memory is one of the major challenges in neuroscience. Taken the advantages of the nematode Caenorhabditis elegans, we investigated ?-adducin (add-1) in aversive olfactory associative learning and memory. Loss of add-1 function selectively impaired short- and long-term memory without causing acquisition, sensory, or motor deficits. We showed that ?-adducin is required for consolidation of synaptic plasticity, for sustained synaptic increase of AMPA-type glutamate receptor (GLR-1) content and altered GLR-1 turnover dynamics. ADD-1, in a splice-form- and tissue-specific manner, controlled the storage of memories presumably through actin-capping activity. In support of the C. elegans results, genetic variability of the human ADD1 gene was significantly associated with episodic memory performance in healthy young subjects. Finally, human ADD1 expression in nematodes restored loss of C. elegans add-1 gene function. Taken together, our findings support a role for ?-adducin in memory from nematodes to humans. Studying the molecular and genetic underpinnings of memory across distinct species may be helpful in the development of novel strategies to treat memory-related diseases.
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