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Snapin is critical for presynaptic homeostatic plasticity

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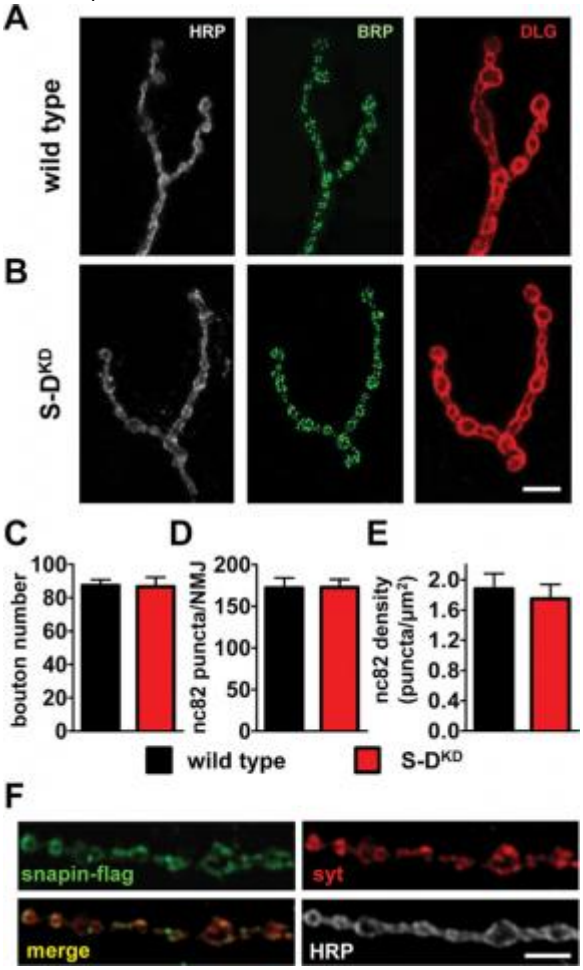

Snapin is critical for presynaptic homeostatic plasticity [1]

Dickman DK [2], Tong A [3], Davis GW [4]

Journal of Neuroscience
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Abstract

The molecular mechanisms underlying the homeostatic modulation of presynaptic neurotransmitter release are largely unknown. We have previously used an electrophysiology-based forward genetic screen to assess the function of >400 neuronally expressed genes for a role in the homeostatic control of synaptic transmission at the neuromuscular junction of Drosophila melanogaster. This screen identified a critical function for dysbindin, a gene linked to schizophrenia in humans (Dickman and Davis, 2009). Biochemical studies in other systems

have shown that Snapin interacts with Dysbindin, prompting us to test whether Snapin might be involved in the mechanisms of synaptic homeostasis. Here, we demonstrate that loss of snapin blocks the homeostatic modulation of presynaptic vesicle release following inhibition of postsynaptic glutamate receptors. This is true for both the rapid induction of synaptic homeostasis induced by pharmacological inhibition of postsynaptic glutamate receptors, and the long-term expression of synaptic homeostasis induced by the genetic deletion of the muscle-specific GluRIIA glutamate receptor subunit. Loss of snapin does not alter baseline synaptic transmission, synapse morphology, synapse growth, or the number or density of active zones, indicating that the block of synaptic homeostasis is not a secondary consequence of impaired synapse development. Additional genetic evidence suggests that snapin functions in concert with dysbindin to modulate vesicle release and possibly homeostatic plasticity. Finally, we provide genetic evidence that the interaction of Snapin with SNAP25, a component of the SNARE complex, is also involved in synaptic homeostasis.

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