RIM Controls Homeostatic Plasticity through Modulation of the Readily-Releasable Vesicle Pool

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Müller M [2], Liu KS [3], Sigrist SJ [4], Davis GW [5].

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Abstract

Rab3 interacting molecules (RIMs) are evolutionarily conserved scaffolding proteins that are located at presynaptic active zones. In the mammalian nervous system, RIMs have two major activities that contribute to the fidelity of baseline synaptic transmission: they concentrate calcium channels at the active zone and facilitate synaptic vesicle docking/priming. Here we
confirm that RIM has an evolutionarily conserved function at the Drosophila neuromuscular junction and then define a novel role for RIM during homeostatic synaptic plasticity. We show that loss of RIM disrupts baseline vesicle release, diminishes presynaptic calcium influx, and diminishes the size of the readily-releasable pool (RRP) of synaptic vesicles, consistent with known activities of RIM. However, loss of RIM also completely blocks the homeostatic enhancement of presynaptic neurotransmitter release that normally occurs after inhibition of postsynaptic glutamate receptors, a process termed synaptic homeostasis. It is established that synaptic homeostasis requires enhanced presynaptic calcium influx as a mechanism to potentiate vesicle release. However, despite a defect in baseline calcium influx in rim mutants, the homeostatic modulation of calcium influx proceeds normally. Synaptic homeostasis is also correlated with an increase in the size of the RRP of synaptic vesicles, although the mechanism remains unknown. Here we demonstrate that the homeostatic modulation of the RRP is blocked in the rim mutant background. Therefore, RIM-dependent modulation of the RRP is a required step during homeostatic plasticity. By extension, homeostatic plasticity appears to require two genetically separable processes, the enhancement of presynaptic calcium influx and a RIM-dependent modulation of the RRP.

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