Drosophila twinfilin is required for cell migration and synaptic endocytosis

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Summary

Precise actin regulation is essential for vital cellular processes. *twinfilin* (*twf*) encodes an actin monomer sequestering protein, but its in vivo functions are unclear. In this study, we characterized *twf* null mutants in a metazoan for the first time and found that *Drosophila twf* negatively regulates F-actin formation in subcellular regions of rapid actin turnover in three different systems, namely neuromuscular junction (NMJ) synapses, migratory border cells, and epithelial follicle cells. Loss of *twf* function resulted in defects in axonal growth in the brain and border cell migration in the ovary. Additionally, we found that the post-synaptic level of the glutamate receptor GluRIIA was specifically reduced in *twf* mutants. More importantly, we showed that *twf* mutations caused endocytic defects at NMJ synapses as detected by the FM1-43 uptake assay, a pharmacological assay with treatment of the F-actin destabilizing drug latrunculin A, and electrophysiological analysis. Together, our results suggest that twinfilin promotes actin turnover in multiple cellular and developmental processes that are highly dependent on actin dynamics.