Formation and plasticity of glutamatergic synapses: Characterization of the roles of beta-amyloid precursor protein, scribble, and wingless at the Drosophila neuromuscular junction

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Abstract

The flow of information through neuronal circuits relies on the ability of neurons to form synaptic connections with specific temporal and spatial properties. These properties are not static but have the ability to change, allowing synaptic connections to be strengthened or weakened. It is this plastic nature of synapses that is central to higher order processes such as learning and memory. However, major gaps remain in our understanding of this process. Throughout my dissertation work I have examined mechanisms of a form of structural synaptic plasticity by analyzing the roles of a variety of proteins that we have found serve to regulate the formation and maintenance of glutamatergic synapses at the *Drosophila* NMJ. These proteins include APPL, the *Drosophila* homolog of Alzheimer's disease-associated β-Amyloid Precursor Protein (APP), the tumor suppressor protein Scribble (Scrib), the secreted signaling molecule Wingless (Wg), and the cell adhesion molecule Fasciclin II (FasII). In this work, in collaboration with members of the labs of Dr. Vivian Budnik, Dr. Kalpana White, and Dr. Susan Cumberledge, I have demonstrated that Wingless (Wg) provides a secreted signal that is required to initiate the formation of pre- and postsynaptic structures. Further, I have also demonstrated that once synapse formation is initiated, presumably by Wg signaling, APPL regulates synaptic bouton proliferation. This process also involves signaling by FasII, a protein required for synapse maintenance, and growth. Moreover, I have also demonstrated that Scrib is a scaffolding protein that plays a key role at these synapses in influencing neurotransmitter release. ^

Subject Area

Biology, Molecular|Biology, Neuroscience|Biology, Cell

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