

Seminar/Talk

Dynamic regulation of presynaptic function and plasticity in health and disease

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Host: Peter Jonas

The process of synaptic vesicle priming is an essential determinant of the function, strength, and plasticity of nerve cell synapses, because it maintains a pool of readily releasable vesicles at any given time and determines the time course of synaptic fatigue and recovery. The corresponding forms of synaptic short-term plasticity determine multiple complex brain functions, from sensory adaptation to working memory. Munc13s execute synaptic vesicle priming by regulating the assembly of fusogenic SNARE complexes. They are themselves regulated by three major pathways, involving (i) calcium-calmodulin signaling via dedicated amphipathic calcium-calmodulin binding sites, (ii) diacylglycerol signaling via C1 domains, and (iii) calcium-phospholipid signaling via C2 domains. We studied the functional relevance of these regulatory pathways in various synapse types and found that calcium-dependent Munc13-regulating pathways are major determinants of synaptic short-term plasticity, synapse endurance, and synaptic fidelity. I will discuss these data in the context (i) of the combined role of signaling pathways that target presynaptic function, (ii) of the role of Munc13 priming proteins in determining the unique features of regulated exocytosis at nerve cells synapses, and (iii) of the role of these processes in physiological and pathophysiological circuit function.

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Office Bldg West / Ground floor / Heinzel Seminar Room (I21.EG.101)



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