

# Neuroscience Colloquium

## Special presentation

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### A new branch in synaptic vesicle cycling: long-distance vesicle dynamics

Polarized trafficking of synaptic proteins to axons and dendrites is crucial to neuronal function. Through forward genetic analysis in *C. elegans*, we identified a cyclin (CYY-1) and a cyclin-dependent kinase (PCT-1) necessary for targeting presynaptic components to the axon. Another cyclin-dependent kinase, CDK-5, and its activator p35, act in parallel to and partially redundantly with the CYY-1/PCT-1 pathway. Synaptic vesicles and active zone proteins mostly mislocalize to dendrites in animals defective for both PCT-1 and CDK-5 pathways. Unlike the kinesin-3 motor, *unc-104/Kif1a* mutant, *cyy-1 cdk-5* double mutants have no reduction in anterogradely moving synaptic vesicle precursors (SVPs) as observed by dynamic imaging. Instead, the number of retrogradely moving SVPs is dramatically increased. Furthermore, this mislocalization defect is suppressed by disrupting the retrograde motor, the cytoplasmic dynein complex. Thus, PCT-1 and CDK-5 pathways direct polarized trafficking of presynaptic components by inhibiting dynein-mediated retrograde transport and setting the balance between anterograde and retrograde motors.

Unpublished results will also be presented that indicate CDK-5 suppresses the commitment of endocytosed synaptic vesicles to a retrograde trafficking pathway at presynaptic terminals. This, in turn, prevents the localization of synaptic vesicles to the cell body and dendrites.

**Location:** Hörsaal Innere Medizin  
Sauerbruchweg 2, CCM

**Date:** Monday, October 11<sup>th</sup>, 4:00 p.m.

**Host:** Stephan Sigrist

Supported by:

**SFB 665** "Developmental Disturbances in the Nervous System"

**GRK 1123** "Cellular Mechanisms of Learning and Memory Consolidation in the Hippocampal Formation"

**SFB-TRR 43** "The brain as a target of inflammatory processes"

**NeuroCure**

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