

Neuroscience Colloquium Winter Semester 2011/2012

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The role of translational mechanisms in the homeostatic control of synaptic function

Homeostatic mechanisms operate to stabilize synaptic function; however, we know little about how they are regulated. Exploiting *Drosophila* genetics, we have uncovered a critical role for the target of rapamycin (TOR) in the regulation of synaptic homeostasis at the *Drosophila* larval neuromuscular junction. Loss of postsynaptic TOR disrupts a retrograde compensatory enhancement in neurotransmitter release that is normally triggered by a reduction in postsynaptic glutamate receptor activity. Moreover, postsynaptic overexpression of TOR or a phosphomimetic form of S6 ribosomal protein kinase, a common target of TOR, can trigger a strong retrograde increase in neurotransmitter release. Interestingly, heterozygosity for eIF4E, a critical component of the cap-binding protein complex, blocks the retrograde signal in all these cases. Our findings suggest that cap-dependent translation under the control of TOR plays a critical role in establishing the activity dependent homeostatic response at the NMJ.

Location: BCCN lecture theater,
Bernstein Center for Computational Neuroscience
Humboldt-Universität zu Berlin
Philippstr. 13, Haus 6

Date: Friday, November 25, 4:00 p.m.

Host: Stephan Sigrist

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SFB 665 "Developmental Disturbances in the Nervous System";

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

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