CDB SEMINAR

Speaker: **Katsunori Nakata**

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Title: **"Regulation of a DLK-1 and p38 MAP kinase pathway by the ubiquitin ligase RPM-1 is required for presynaptic development"**

Date:	**Monday, June 6**
Time:	**16:00 -17:00**
Place:	**7F Conference Room of Building A,CDB**

Summary:
Synapses display a stereotyped ultrastructural organization, commonly containing a single electron-dense presynaptic density surrounded by a cluster of synaptic vesicles. The mechanism controlling subsynaptic proportion is not understood. Loss of function in the *C. elegans rpm-1* gene, a putative RING finger/E3 ubiquitin ligase, causes disorganized presynaptic cytoarchitecture. RPM-1 is localized to the presynaptic periactive zone. We report that RPM-1 negatively regulates a p38 MAP kinase pathway composed of the dual leucine zipper-bearing MAPKKK DLK-1, the MAPKK MKK-4, and the p38 MAP kinase PMK-3. Inactivation of this pathway suppresses *rpm-1* loss of function phenotypes, whereas overexpression or constitutive activation of this pathway causes synaptic defects resembling *rpm-1(lf)* mutants. DLK-1, like RPM-1, is localized to the periactive zone. DLK-1 protein levels are elevated in *rpm-1* mutants. The RPM-1 RING finger can stimulate ubiquitination of DLK-1. Our data reveal a presynaptic role of a previously unknown p38 MAP kinase cascade.

Reference: Cell 120:407-20 (2005)

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