OIST SEMINAR

Hosted by Cellular and Molecular Synaptic Function Unit

Speaker: Dr. Yasunori Hayashi

Senior team leader, Memory mechanisms,
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Date: Friday, December 16
Time: 16:00 - 17:00
Venue: Meeting Room D015, Level D, Lab1

Title:

"**Molecular Mechanisms of Hippocampal Synaptic Plasticity**"

Abstract:

Upon induction of long-term potentiation (LTP), a cellular signaling cascade triggered by the transient Ca^{2+} influx through NMDAR leads to a persistent increase in synaptic transmission and an enlargement of the dendritic spines. There must be a mechanism within the cascade where the transient Ca^{2+} raise is converted to a persistent biochemical signaling. We propose mutually activating kinase-effector complex as a novel mechanism involved in this process. The formation of a stable heterooligomer between “T-site” of CaMKII and a RacGEF protein TIAM1, locks CaMKII in an active conformation mimicking T286 autophosphorylation, which, in turn, results in a persistent phosphorylation of TIAM1. This mutually activating complex persistently activates Rac required for the maintenance of LTP. We conclude that CaMKII, through this mutually activating mechanism, acts as a signaling hub, which, once activated by Ca^{2+}, can persistently activate TIAM1 and possibly other signaling molecules in the vicinity.

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