Day 2　Saturday, September 12

Plenary Lecture　特別講演

2L1　A role of drebrin A in the activity-dependent trafficking of NMDA receptors to the plasma membrane

Saturday, September 12　10：00～11：00　Room A

Chairperson：Hiroshi Kiyama（木山　博資）
Functional Anatomy and Neuroscience, Anatomy and Cell Biology, Nagoya University Graduate School of Medicine（名古屋大学大学院医学系研究科機能組織学（解剖学第二））

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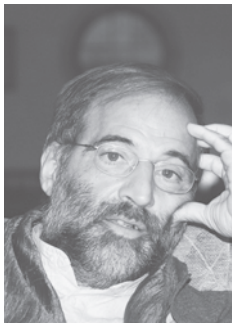
NMDA receptors (NMDARs) are central molecular agents that enable the activity-dependent modification of synaptic strengths at excitatory synapses. However, relatively little is known about the molecular mechanisms regulating NMDAR levels at each synapse. We have previously used electron microscopic (EM) immunocytochemistry (ICC) to quantify the NMDAR levels at dendritic spines and to further differentiate the proportion of NMDARs occurring specifically at the plasma membrane, where they can bind to ligands, versus the cytoplasm, reflecting their reserve pools. In 2003, we demonstrated that this approach can successfully capture the activity-dependent trafficking of NMDARs from the dendritic shaft into the spine cytoplasm and to the plasma membrane following 30 min exposure of intact pyramidal neurons within cerebral cortex to the NMDAR antagonist, D-APV. We hypothesized that decreased activation of synaptic NMDARs by D-APV may increase NMDAR trafficking to the plasma membrane by promoting the tethering of NMDAR-containing saccules along the F-actin lattice. What might be the molecular agent that translates synaptic activity to the F-actin-mediated trafficking of NMDARs? Drebrin A is a good candidate to be this agent, because drebrin A finds to F-actin and is trafficked more into the spine head in response to D-APV blockade. This idea was tested by subjecting cortices of mice with global KO of drebrin A to D-APV blockade. Drebrin A KO cortices failed to up-regulate the NMDAR level at the plasma membrane within the hemisphere treated with D-APV, relative to the vehicle-treated hemisphere (0% ± 13% increase [mean ± SEM]), while the WT cortices up-regulated NMDAR levels at spines 94% ± 28% following D-APV treatment, relative to the vehicle-treated side. Comparisons of spine head size and NMDAR levels at spines of vehicle-treated hemispheres comparisons did not reveal any difference across the KO-WT genotypes, suggesting that drebrin A is involved in the activity-dependent regulation of synaptic strength, rather than their basal levels.

Presidential Lectures　会長招聘講演



2L2　Is delivery a critical period in the pathogenesis of autism?

Saturday, September 12　11：00～11：40　Room A

Chairperson：Toru Takumi（内匠　透）
Laboratory for Mental Biology RIKEN Brain Science Institute（理化学研究所 脳科学総合研究センター 精神生物学研究チーム）

Yehezkel Ben-Ari
INMED, France

2L3　Neuronal calcium sensor proteins：contribution to the diversity of neuronal calcium signaling

Saturday, September 12　13：50～14：30　Room A

Chairperson：Masayuki Miura（三浦　正幸）
Graduate School of Pharmaceutical Sciences, Faculty of Pharmaceutical Sciences, University of Tokyo（東京大学大学院薬学系研究科・薬学部）

Ken Takamatsu（高松　研）
Dept. of Physiology, Toho University School of Medicine（東邦大学医学部生理学講座）