The 1st Seminar
Parallel Brain Cross-Modal Research Division

本講演は公開セミナーです。学生の参加も大歓迎です。

Differential subcellular distribution and regulatory role of Ca_V1 and Ca_V2 calcium channels in hippocampal neurons

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Location: Room K201, Lecture Build, Noda

Calcium (Ca^{2+}) signaling controls a variety of fundamental processes and reaction pathways including excitability, excitation-transcription coupling, synaptic plasticity and release of neurotransmitters in both glutamatergic and GABAergic neurons in the central nervous system. These processes are switched on by influx of Ca^{2+} into the cytosol, partially through voltage-gated Ca^{2+} (Ca_V) channels that are activated by action potentials and/or subthreshold depolarizing signals and localized to both axonal and somato-dendritic compartments of hippocampal neurons. In one project, we investigated the nano-scale organization of Ca_V2 channels in axon terminals of dentate gyrus granule cells contacting with different cell types. Their axons form three types of synapses with distinct functional and morphological properties, depending on the nature of their postsynaptic targets, and feature a highly precise control of their glutamate release. Using quantitative SDS-FRL immunoelectron microscopy we found that (i) $Ca_V2.1$ (P/Q-type) and $Ca_V2.2$ (N-type) channels were organized in clusters in active zones MF boutons, whereas $Ca_V2.3$ (R-type) channels were scattered over the entire surface of boutons, (ii) the density of $Ca_V2.1$ was consistently higher than that of $Ca_V2.2$ in synapses, and (iii) $Ca_V2.1$ and $Ca_V2.2$ channels showed different distribution pattern at distinct MFB synapses. These data suggest differential contribution of Ca_V channels to the precise timing of presynaptic Ca^{2+} inflow and glutamate release that is important for temporal information processing in the hippocampus.

In another study, we investigated the function of $Ca_V1.2$ (L-type) on dendritic membranes of CA1 somatostatin interneurons (SOM-INs). We found that $Ca_V1.2$ channels were present over the surface of dendrites and clustered with $GABA_B$ receptors ($GABA_BRs$), which inhibited them resulting in reduced dendritic Ca^{2+} influx and block of long-term potentiation at excitatory synapses onto SOM-INs. This effect precludes synaptic strengthening during network activation, a mechanism by which $GABA_BRs$ can contribute to a long-term alteration of excitation and inhibition balance in the network.

An online stream via Teams will be available. Details will be announced later.

主催: パラレル脳クロスモーダル研究部門

To establish a brain research platform Tokyo University of Science, the Brain Interdisciplinary Research Division (BIRD) was launched in 2016. In 2021, it evolved into the Parallel Brain Interaction Division, and in 2026, it was further reorganized as the Parallel Brain Cross-Modal Research Division to advance its research foundations. The division promotes collaborative research both within the university and with external institutions to further advance brain research.

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