博士論文（要約）

Study of Plasticity by Motor Protein KIF17

(モータータンパク質 KIF17 による可塑性についての研究)

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Kinesin superfamily proteins (KIFs) are microtubule-based motor proteins that play a fundamental role in intracellular transport. They are consisted of 45 *Kif* genes in mouse genome and implicated in neuronal morphogenesis, function, and survival by transporting a variety of cargos. To date, it has been revealed that several KIFs are involved in synaptic plasticity and higher brain function. In this thesis, I focused on KIF17 that predominantly transports N-methyl-D-aspartate receptor subunit 2B (NR2B) in dendrites. Overexpression of KIF17 enhances learning and memory. On the other hand, disruption of KIF17 expression or its capacity of NR2B loading/releasing leads to various types of memory impairment. KIF17 also maintains NR2A/2B balance at synapses thereby underlying synaptic plasticity. The genetic deletion of KIF17 disrupts long-term potentiation (LTP) and long-term depression (LTD) because of a decrease in the level of synaptic N-methyl-D-aspartate (NMDA) receptors. Additionally, *Kif17* gene has been proposed as one of the candidate genes for schizophrenia, and suggested to relate with the pathogenesis of amyotrophic lateral sclerosis (ALS). However, precise role of KIF17 in brain functions and its underlying molecular mechanisms have been still elusive. In the present study by performing a series of molecular cell biological analyses, I found a novel crucial role of KIF17 in memory processes via NMDA receptor activation.