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認定科目

【博士課程】

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【修士課程(医科学コース)】

選択科目「大学院セミナー」



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場所：順天堂大学 10号館 1階105号室

Neural-Specific Autophagy Regulation

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Abstract

Autophagy, a vital lysosome-mediated degradation pathway, maintains cellular homeostasis by removing damaged organelles and misfolded proteins. This is particularly crucial in neurons due to their long lifespan and high metabolic activity. Autophagy is highly specialized in neurons, as their extensive axonal and dendritic networks require precise autophagic activity regulation to sustain synaptic function and plasticity.

Mutations in autophagy genes WDR45 and WDR45B cause beta-propeller protein-associated neurodegeneration (BPAN) and intellectual disability (ID), respectively. WDR45/45B, along with WIPI1/2, are mammalian homologs for yeast Atg18, which forms a complex with Atg2, essential for autophagosome formation. However, the specific functions of WDR45/45B-ATG2 in neurons are unclear. Our study shows that WDR45/45B and ATG2A are crucial for the progression of autophagosomes into autolysosomes in neural cells. They interact with the SNARE proteins STX17, SNAP29, and VAMP8, facilitating the assembly of fusion complex. ATG2A and another tether protein, EPG5, exhibit partial redundancy in mediating autophagosome-lysosome fusion. Thus, the WDR45/45B-ATG2A complex plays a key role in neural autophagy by tethering autophagosomes with lysosomes for fusion.

Publications

Xue et al. J Cell Biol. 2026, Zheng et al. J Cell Biol. 2025, Zheng et al. Autophagy. 2025,
Li et al. J Cell Biol. 2024, Ji et al. J Cell Biol. 2022, Ji et al. Curr Biol. 2022,
Ji et al. Autophagy. 2020.