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科研費 国際共同研究加速基金（国際先導研究）セミナー
共催：順天堂大学大学院医学研究科

日時：１１月２日（月）17:00-
場所：順天堂大学 10号館 1階105号室

# Organelle Remodeling and Lipid Trafficking during Development

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## Abstract

Developmental remodeling of animal tissues requires extensive organelle turnover, yet how metabolic and signaling cues regulate the selective clearance of distinct organelles within the same cell remains unclear. Using the developing Drosophila melanogaster intestine, we identified ubiquitin signaling and sterol trafficking as interconnected mechanisms that regulate organelle-specific autophagy. We previously showed that PINK1 coordinates selective clearance of the endoplasmic reticulum (ER-phagy) and mitochondria (mitophagy) through distinct ubiquitin pathways. PINK1 promotes ER-phagy through Keap1 and the Cullin3 ubiquitin ligase, which regulate ubiquitylation of the ER-phagy receptor Rtnl1, while PINK1-dependent Parkin activity drives mitochondrial clearance.

We subsequently identified an Atg-independent ER clearance pathway mediated by ER–lysosome contacts and the lipid-transfer proteins Vap33, Osbp, and Start1, linking ER turnover to sterol transport.

Our current work reveals that dietary sterol primarily accumulate in mitochondria positioned near the intestinal brush border. During the larva-toprepupa transition, sterols are transferred from mitochondria to the ER and subsequently delivered to lysosomes through selective autophagy. The elevated ER sterol level promotes Rtnl1 ubiquitylation and stimulate Rtnl1-dependent ER-phagy.

Together, these studies establish a mechanistic connection between ubiquitin signaling, inter-organelle sterol trafficking, and selective organelleturnover. We identify sterol as both metabolic cargo and a regulatory signal for selective ER clearance during development.

## Publications

Wang et al. Cell Rep 2026, Huang et al. Autophagy 2026, Wang et al. Cell 2023,
Wang et al. Curr Biol. 2022, Wang et al. J Cell Biol. 2019.

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