

OsakaMito2026



Spring Workshop on Mitochondrial Sciences

Date: 15:30-18:00 on Monday, April 20

Place: 1F 101 Big Hall | Center for Infectious Disease Education and Research (CiDER) | The University of Osaka, Suita, Japan

Organizer: **Koji Okamoto** (The University of Osaka, Suita, Japan)

Invited Speakers



15:30-16:10

Yuhei Araiso

Kanazawa University, Japan

Structures and dynamics of molecular machineries regulating mitochondrial morphology



16:10-16:50

Sho Aki

The University of Osaka, Japan

Phosphoinositides link organelle trafficking to mitochondrial fusion

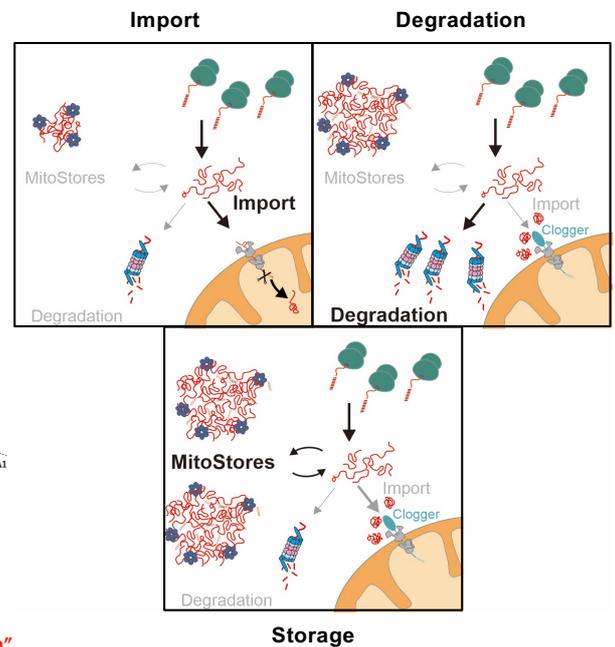
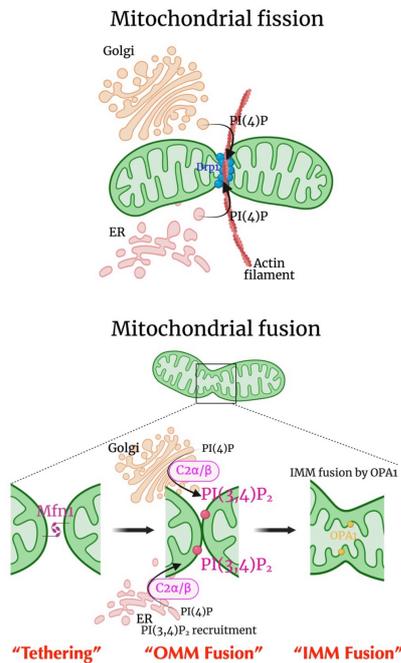
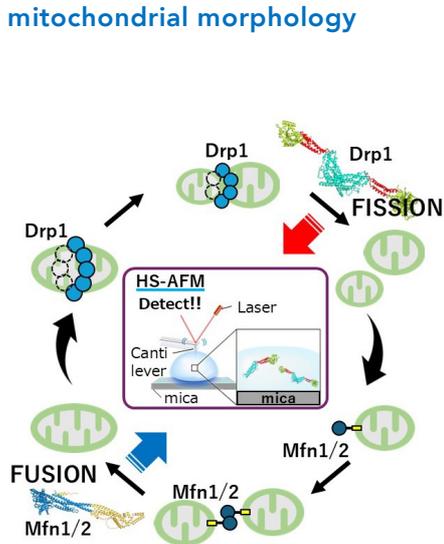


17:00-18:00

Johannes M. Herrmann

University of Kaiserslautern, Germany

Protein targeting to mitochondria – a big challenge for eukaryotic cells



This workshop is a credit-awarding seminar for Group B (Elective Subjects) credits for FBS graduate students. Please submit the original of your report to your supervisor and a copy to the FBS Educational Affairs Section.

No registration is required. For inquiry, please contact Koji Okamoto (Tel: 06-6879-7970 Email: okamoto.koji.fbs@osaka-u.ac.jp)

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Dr. Yuhei Araiso

Associate Professor

Department of Clinical Laboratory Science, Division of Health Sciences, Graduate School of Medical Sciences, Kanazawa University, 5-11-80 Kodatsuno, Kanazawa, Ishikawa, 920-0942, Japan

Structures and dynamics of molecular machineries regulating mitochondrial morphology

Mitochondrial dynamics is finely regulated by dynamin GTPase family proteins such as Drp1, Mfn1/2 and Opa1. To elucidate the detailed molecular mechanisms underlying membrane remodeling, we analyzed the molecular dynamics of these dynamin proteins using high-speed atomic force microscopy. Furthermore, we succeeded in visualizing the direct interactions between these dynamin proteins and the phospholipid bilayers. Here we present and discuss the fusion and fission mechanisms revealed by single-molecule analysis.

16:10-16:50

Dr. Sho Aki

Lecturer

Organelle dynamics modeling, Premium Research Institute for Human Metaverse Medicine (WPI-PRIME), The University of Osaka 2-2, Yamadaoka, Suita, Osaka, 565-0871, Japan

Division of Nutriomics and Oncology, RCAST, The University of Tokyo, 4-6-1 Komaba, Meguro-ku, Tokyo 153-8904, Japan

Phosphoinositides link organelle trafficking to mitochondrial fusion

Mitochondria continuously remodel their morphology through coordinated fusion and division, yet how mitochondrial fusion is spatially regulated within the organelle network remains unclear. Here we uncover a previously unrecognized role for phosphatidylinositol 3,4-bisphosphate [PI(3,4)P₂]-enriched trans-Golgi network (TGN) vesicles in mitochondrial fusion. These vesicles are recruited to ER-marked fusion sites downstream of mitofusin-dependent docking, suggesting that lipid signaling and organelle trafficking cooperate to drive mitochondrial membrane remodeling. Loss of class II PI3-kinases (PI3K-C2α/β), which generate PI(3,4)P₂, disrupts mitochondrial fusion in mammalian cells and cardiomyocytes, leading to mitochondrial dysfunction and heart failure in mice. Our findings highlight lipid-mediated organelle communication as a key regulator of mitochondrial dynamics.

17:00-18:00

Prof. Dr. Johannes M. Herrmann

Professor

Cell Biology, University of Kaiserslautern, Erwin-Schrödinger-Strasse 13, 67663 Kaiserslautern, Germany

Protein targeting to mitochondria – a big challenge for eukaryotic cells

Most mitochondrial proteins are synthesized as cytosolic precursor proteins before they are imported into mitochondria. Recent *in vivo* studies showed that mitochondrial precursor proteins have a surprisingly complex biology before they reach the mitochondrial surface. We realized that mitochondrial precursor proteins explore the cytosol, get in contact with components of the proteasome and chaperone network and often end up on the surface or even the lumen of other cellular compartments. In my talk, I will give an overview about our current knowledge of these early reactions in mitochondrial protein targeting and present recent data from our team.

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