Department seminar

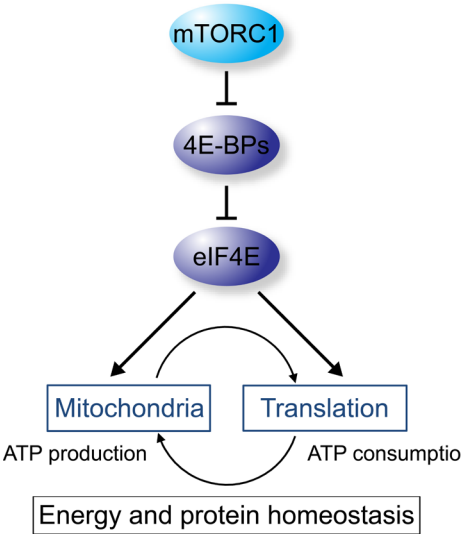
日時：12 月 20 日（水曜日）、16:00-17:00

場所：工学部 3 号館 8B04 講義室

演者：Masahiro Morita, Ph.D.
Principal Investigator and Tenure-Track Assistant Professor
Department of Molecular Medicine and Barshop Institute for Longevity and Aging Studies, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

Cross-talk between mTORC1, mRNA translation, and energy metabolism in cancer

Protein synthesis is the most energy-consuming process in the cell. Dysregulation of mRNA translation and energy metabolism is implicated in various diseases, such as cancer and metabolic disorders. However, the mechanisms governing the interplay between protein and energy homeostasis remain largely unexplored. In our recent studies, by using a combination of pharmacological, genetic, histological, and omics approaches, we identify a hitherto unrecognized link between the nutrient-sensing mTORC1 pathway, mRNA translation, mitochondrial dynamics, and cancer. Our investigation reveals that the alterations in mTORC1 activity coincide with dramatic changes in mitochondrial dynamics, characterized by hyper-fusion. mTORC1, through the phosphorylation of the translation initiation factor 4E (eIF4E)-binding proteins (4E-BPs), promotes mitochondrial fission by selectively enhancing the translation of mRNAs encoding mitochondrial fission factors. This discovery underscores the pivotal role of mTORC1 in coupling mRNA translation with mitochondrial dynamics and functions to maintain energy consumption and production. Our research further shows that the suppression of mRNA translation of mitochondrial fission factors induced by mTOR inhibition acts as a cytoprotective mechanism that prevents mitochondrial fragmentation and cell death. These findings offer a molecular foundation for enhancing the anti-tumor effects of mTOR inhibitors in clinical settings by targeting mitochondria. In addition, I will introduce our recent study on the mRNA degradation-dependent regulation of inter-organ communication and metabolic syndrome and discuss the post-transcriptional control of gene expression in the context of cancer and metabolic disorders.



References:

Morita et al., 2013, Cell Metab; Morita et al., 2017, Mol Cell; Hulea et al., 2018, Cell Metab; Morita et al., 2019, PNAS; Katsumura et al., 2022, Cell Metab

このセミナーに関する連絡先
鈴木 勉　（ts@chembio.t.u-tokyo.ac.jp）
協賛　JST-ERATO　鈴木RNA修飾生命機能プロジェクト