Graduate School Event

Thesis Defense: The ER complex SUTU-7/MACO-1 regulates the fate of mRNAs encoding GPCRs

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This thesis elucidates functions for two previously uncharacterized proteins, suppressor of tumorigenicity 7 (SUTU-7) and macoilin (MACO-1). Mutants defective in these proteins were identified in a forward genetic screen for animals that lost the ability to aggregate, a behavior linked to oxygen sensing. Aggregation is controlled by a hub-and-spoke circuit that consists of the RMG hub interneurons and an array of sensory neurons linked to RMG via electrical and/or chemical synapses. SUTU-7 is a membrane protein of unknown function conserved across metazoa. Mutants of sutu-7 are healthy but have the signature behavioral phenotypes associated with low activity in the RMG circuit: defects in escape from 21% O2, elevated escape from CO2, and robust escape from hypoxia. We found that SUTU-7 shows a broad and predominantly neuronal expression pattern and resides in the endoplasmic reticulum (ER). SUTU-7 forms a complex with MACO-1, which recruits the deadenylation complex CCR4-Not to the ER. Our data suggest that SUTU-7 interacts with membrane proteins, including GPCRs, as they are made in the ER. The O2 response defects of sutu-7 mutants reflect stabilization of mRNA encoding the GPCR NPR-1, which inhibits the RMG interneurons. A series of qPCR experiments suggest that SUTU-7 destabilizes mRNAs encoding most C. elegans GPCRs. Tunicamycin treatment, which disrupts protein folding by inhibiting N-linked glycosylation, broadens the number of mRNAs negatively regulated by SUTU-7. Our data suggest SUTU-7/MACO-1 form an ER quality control complex that destabilizes mRNAs encoding wild type GPCRs, likely in response to aberrant receptor biogenesis.

Tuesday, June 24, 2025 02:00pm - 03:00pm

Sunstone Bldg / Ground floor / Big Seminar Room A (I23.EG.102) and Zoom

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https://ista.ac.at/en/campus/how-to-get-here/ The ISTA Shuttle bus is marked ISTA Shuttle (#142) and has the Institute Logo printed on the side.