Ackert Hall, Room 120
Wednesday, March 12, 2025
4:00 P.M.

Coffee and Cookies
Chalmers Hall, Room 168
3:45 P.M.

Biochemistry & Molecular Biophysics Seminar

Peroxisome-mitochondrial communication in aging

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Peroxisomes recently emerged as a novel regulator of aging. It has been shown that peroxisomal protein import (translocation) is greatly impaired in aged animals, which in turn drives many age-associated pathologies and cellular dysfunction, such as altered mitochondrial morphology and function. To further understand how peroxisomal dysfunction drives aging, we investigated the interplay between peroxisomes and mitochondria, with an emphasis on the role of peroxisome-derived ether phospholipids (plasmalogens) in age-dependent loss of mitochondrial cristae integrity. Plasmalogens comprise about 18-20% of the total phospholipid mass in animals and humans, and plasmalogen deficiency has been linked to impaired mitochondrial function and age-related diseases (e.g., Alzheimer's disease). We recently showed that the biosynthesis of plasmalogens decreases with age, consistently, plasmalogen deficiency mutants exhibit aberrant mitochondrial cristae structures and impaired mitochondrial respiration. One plausible mechanism for plasmalogen-regulated mitochondrial function would be through specific lipid-protein interactions. We performed a lipid-protein interactome analysis and identified >400 novel plasmalogen-interacting proteins, among which 33% of them are localized to mitochondria. Currently, we are carrying out functional analysis to investigate how plasmalogens interact with mitochondrial membrane proteins to maintain cristae integrity and mitochondrial metabolism during animal aging. Our studies are expected to reveal an exciting role of plasmalogen ether phospholipids in peroxisome-mitochondria crosstalk and mitochondrial membrane dynamics during aging.