Parkinson’s disease (PD) is one of the most common of the neurodegenerative disorders, affecting 1-2% of the population worldwide. Multiple lines of evidence place mitochondrial dysfunction as a central player in the pathogenesis of sporadic PD, and studies of genes associated with familial PD demonstrate convergent pathways involving oxidative stress and mitochondrial dysfunction. Two proteins commonly mutated in familial PD, PINK1 and Parkin, play a key role in maintaining mitochondrial integrity by identifying damaged mitochondria and degrading them through a selective form of autophagy termed mitophagy. Our lab investigates the molecular mechanisms of PINK1/Parkin mitophagy and how it works with mitochondrial repair pathways to maintain healthy mitochondria. Furthermore, given the fundamental importance of autophagy to cellular viability, our research also aims to understand the factors and processes that drive autophagy.

Research Projects

1. **PINK1/Parkin mitophagy**

2. **Mitochondrial quality control**

3. **Autophagy mechanisms**

Transmission electron microscopy was used to generate a 3D reconstruction of a damaged mitochondrion (red), engulfed by an autophagosome (green), surrounded by the endoplasmic reticulum (blue) during PINK1/Parkin mitophagy.

Selected significant publications:


