



Blocking Mitochondrial Fission as Potential Therapy for Parkinson's Disease

Biomolecular Sciences Institute

Mitochondrial dysfunction is a common pathogenic mechanism in both familial and sporadic Parkinson's disease (PD). However, effective therapy targeting this pathway is currently inadequate. Recent studies suggest that manipulating the processes of mitochondrial fission and fusion has considerable potential for treating human diseases.

To determine the therapeutic impact of targeting these pathways on PD, Dr. Kim Tieu's laboratory uses complementary cell and rodent models of PD. This data shows that blocking mitochondrial fission is neuroprotective in these experimental models. Specifically, it shows that inhibition of the mitochondrial fission GTPase dynamin-related protein-1 (Drp1) using gene-based and small-molecule approaches attenuates mitochondrial dysfunction, neurotoxicity and synaptic release deficits. These results suggest Drp1 inhibition as a potential novel treatment for PD.



Featuring
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Tuesday, March 7th, 2017
12:00 p.m. to 1:00 p.m.
Venue: AHC4-402

The event is free and open to the public.

Co-sponsored by the Robert Stempel College of Public Health & Social Work and the School of Integrated Science and Humanity.