PURDUE UNIVERSITY | School of Health Sciences

SPECIAL SEMINAR

THURSDAY, MAY 8, 2025 | 4:00 - 5:00 P.M. | GRIS 103

OPTINEURIN IN NEURODEGENERATIVE DISEASES: TRUE TO ITS MULTIFUNCTIONALITY?

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Abstract: Mutations in the OPTN gene have been found in amyotrophic lateral sclerosis (ALS) mporal dementia (FTD) and normal-tension glaucoma (NTG). OPTN encodes optineurin, a multirun Mutations in the OPTN gene have been found in amyotrophic lateral sclerosis (ALS), frontotemporal dementia (FTD) and normal-tension glaucoma (NTG). OPTN encodes optineurin, a multifunctional polyubiquitin-binding adaptor protein involved in regulating diverse cellular processes including inflammatory signaling, autophagy, cell death, vesicle trafficking and axonal transport of mitochondria. I will discuss our work on mouse and cellular models mimicking ALS/FTD-linked OPTN mutations, alongside computational analyses of a broader spectrum of OPTN variants found in neurodegenerative diseases. Taken together, these findings highlight the complex role of optineurin in neurodegeneration.

Overall lab focus: Our laboratory is interested in understanding the link between immunity and neurodegenerative disease. Neurodegenerative diseases such as amyotrophic lateral sclerosis (ALS), are marked by an unusually wide genetic and clinical heterogeneity. Chronic (neuro) inflammation has been singled out as a rare hallmark common to all ALS cases. It triggers neuronal damage and death by enhancing glutamate toxicity, protein aggregation and/or oxidative stress. Nevertheless, various anti-inflammatory approaches have consistently failed to stop or slow down disease progression. For this reason, it is imperative to redefine the role of the immune system in neurodegeneration. In our laboratory we focus the role of the innate immune system in active maintenance of the homeostasis in the central nervous system.