Neurodegeneration of dopaminergic neurons is a pathological hallmark of Parkinson's Disease, the second most common neurodegenerative disease and most common movement disorder. The unregulated production of reactive oxygen species (ROS) by dysfunctional mitochondria is one of the contributing factors leading to the death of dopaminergic neurons. One mechanism that the cell can utilize to counter the stress caused by ROS is the transcriptionally encoded antioxidant response initiated by Nuclear factor E2-related factor 2 (Nrf2). Hundreds of cytoprotective genes are regulated whose protein products have roles in detoxifying and removing ROS from the cell. Under basal conditions, Nrf2 is sequestered in the cytoplasm by an actin-associated protein, Keap1. Keap1 is oxidatively modified under reactive oxygen stress, changing the conformation of Keap1 and thereby allowing Nrf2 to enter the nucleus and activate the antioxidant response pathway through binding to the antioxidant response element (ARE).

Evidence suggests that omega-3 fatty acids can act as exogenous activators of the Nrf2/antioxidant pathway and may be able to provide protection from Parkinson's pathogenesis. In this research, we hope to determine if omega-3 fatty acids can activate the Nrf2/antioxidant response, the mechanism by which this occurs and how this may impact neurodegenerative indices both in vitro and in vivo.