**Abstract**

Parkinson’s disease, the second-most common neurological disorder, is characterized by degeneration of dopaminergic neurons and synuclein containing inclusions known as Lewy bodies. As with many neurological disorders, the underlying mechanism(s) for Parkinson’s is not known, but are thought to involve mitochondrial dysfunction and oxidative stress. Current therapeutics suppress symptoms, but neuroprotective treatments have not yet been translated to the clinic. In an attempt to speed up the discovery of neuroprotective drugs, a promising strategy is to “reposition” existing drugs that are used for other indications. A recent study reported that the muscle relaxer Dantrolene was neuroprotective in a mouse model of Alzheimer Disease by reducing amyloid accumulation. Because Dantrolene had neuroprotective effects on Alzheimer’s models, it could have a similar effect in Parkinson’s disease because of shared mechanisms between the two diseases. To test this hypothesis, *Caenorhabditis elegans (C. elegans)* were used to model Parkinson’s. *C. elegans* are non-parasitic nematodes that have short lifecycles. They are a good model organism because their genome is 40% homologous with humans. Another advantage is their transparency and ease of tracking cell linage and cell death because they are comprised of only 959 cells that have been extensively mapped. For this study, *C. elegans* strain BZ555, in which the dopamine transporter (DAT) is fluorescently tagged with GFP, was used. The worms were pre-treated with Dantrolene followed by chronic exposure to MPP+, a neurotoxin commonly used to model dopaminergic neuronal loss similar to that of Parkinson’s disease. There were 5 treatment groups with 5000 worms per treatment in duplicate and the experiment was repeated twice. Fluorescent images were taken of at least 3 worms per treatment group (n > 3). The fluorescent images were then analyzed using ImageJ for fluorescence intensity, which was normalized to control.