Effects of Toxins on Neurological Function and Structure: Their Role in Parkinson's Disease

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Parkinson’s Disease (PD) is an actively researched disease since the underlying cause is still unknown. After a substantial loss of dopaminergic neurons in the brain a patient will experience motor deficits unique to PD. One of the risk factors for the development of PD is the exposure to environmental toxins, such as pesticides. This project was developed to test the effects of the neurotoxins paraquat, rotenone, and MPP+ in the neuronal development of neuroblastoma cells. These cells grow phenotypically similar to dopaminergic neurons after exposure to 9-cis retinoic acid (RA). The RA-induced differentiation can be followed by measuring the extension of cellular neurites which grow similarly to the dendrites of dopaminergic neurons. After exposure to non-lethal dose of neurotoxins, the neuroblastoma cells were differentiated with RA. Length of dendrites in the neuroblastoma cells were measured using a program called ImageJ. The results showed that the neurotoxin paraquat had less of an effect on the cells than rotenone and MPP+. This could be due to the fact paraquat causes a generic oxidative stress to dopaminergic neurons whereas rotenone and MPP+ inhibits Complex 1 of the mitochondria in the cells. The neuroblastoma research lays the groundwork to study the different effects of oxidative stress and the inhibition of complex 1 not only in this completed in vitro experiment but in a future in vivo experiment using the fruit fly .The comparison of the results from the completed and proposed experiment will shed light on if oxidative stress or inhibition of the mitochondrial complex 1 will have a greater detriment to the development of dopaminergic neurons, increasing the risk of developing PD.