Parkinson's Disease (PD) was described by James Parkinson in his publication *An Essay on the Shaking Palsy* in 1817. Characterized by slow onset and gradual decline, the symptoms of PD include tremor, loss of muscular control, depression, sleeplessness and eventually, delirium [1].

Now, nearly 200 years later, the exact cause of PD is still unknown. Clusters of family members suffering from PD make up less than 10% of cases. Definitive inheritance of the disease is rare, and makes up less than 1% of cases, indicating that the idiopathic form of PD is unlikely to have a genetic basis.

The average age of onset is 60 years. At the age of 70 or greater, idiopathic form of PD is unlikely to have a genetic basis. Now, nearly 200 years later, the exact cause of PD is still unknown. Clusters of family members suffering from PD make up less than 10% of cases. Definitive inheritance of the disease is rare, and makes up less than 1% of cases, indicating that the idiopathic form of PD is unlikely to have a genetic basis.

The accumulation of toxins may eventually lead to cell death, upon which the cells can release their neuromelanin. Cell death in the substantia nigra means there is not enough dopamine, which is a signaling chemical the brain uses. If the brain doesn't have enough dopamine, a person can develop Parkinson’s Disease [1].

Neurons like substantia nigra cells are fastidious. Being able to grow these cells enables us to pursue many avenues of future research. Substantia nigra cells in the brain synthesize dopamine using tyrosine hydroxylase (TH) and store it for future release as a neurotransmitter. Excess dopamine can also be made into neuromelanin. Neuromelanin is a pigment similar to the pigment that might be in your skin. The neuromelanin is thought to help protect the brain by taking up toxins such as heavy metals and pesticides [5].

Future Directions
- **Assay binding constants of know PD-inducing toxins toward NM**
- **Correlate the appearance of NM with TH presence and activity**
- **Compare cell mediated toxin binding with non-cell mediated binding to NM**
- **Insert Channelrhodopsin-2 into cells and change behavior with blue light**
- **Insert voltage-sensitive fluorescent proteins to monitor electrical activity**
- **Assay for presence of tyrosine hydroxylase**

On 27-March, we began treating some of the cells with L-DOPA, which is a precursor to dopamine. It is our expectation that the neurons will make neuromelanin [4]. We will use that neuromelanin (NM) to conduct experiments related to how neuromelanin binds to metals and other chemicals. Cell death in the substantia nigra means there is not enough dopamine, which is a signaling chemical the brain uses. If the brain doesn't have enough dopamine, a person can develop Parkinson’s Disease [1].

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