Parkinson’s disease is the second most prevalent neurodegenerative disease, affecting approximately seven to ten million people worldwide. As the disease progresses, the speed of dopaminergic neuron degeneration in the substantia nigra increases, as well as the accumulation of Lewy bodies. The loss of dopaminergic neurons leads to a decrease of dopamine, which results in the impairment of the motor system. Symptoms may begin with minor tremors which gradually advance into stiffness or bradykinesia and ultimately, the loss of movement. Aside from motor symptoms, patients with Parkinson’s disease may experience cognitive problems with attention, speed of mental processing, memory, speech, as well as perception. To reduce motor symptoms, Parkinson patients are given Levodopa (L-dopa), a precursor of dopamine, which crosses the blood brain barrier and converts to replace lost dopamine. However, with long term use of Levodopa, the efficacy diminishes and often induces dyskinesia. To manage motor symptoms, patients undergo deep brain stimulation (DBS) to implant electrodes in the basal ganglia.

When diagnosing and treating Parkinson’s, people tend to focus on the more obvious motor symptoms, but often overlook the cognitive symptoms. Recent studies have shown that Parkinson’s patients, including those treated with Levodopa and deep brain stimulation still have decision-making impairments. Patients with Parkinson’s disease have exhibited problems using previously learned information and sensory information to decide on actions to take.

We used the Contracting Basal Ganglia (CBG) model to examine the effects of Parkinson’s disease. This python based model is an abstract network of the interactions between rate based neurons in different components of the basal ganglia. Since the cells in the model are rate-based neurons, their activity levels are not directly equivalent to the membrane potential, and so it has a different range of possible values than a cell’s membrane potential would have. The network is composed of neurons, which are further divided into channels, of the thalamus, cortex, and the basal ganglia components (Figure 2). Every channel has a salience value, which represents the propensity for the channel to be chosen.

We modified the model to account for the changes in dopamine signals and transmittance in the basal ganglia to model a brain affected by Parkinson’s disease. We ran simulations varying the dopamine signal for each of the six components in the basal ganglia, incrementing the parameter by 0.1 from 0 to 2 and by 1 from 2 to 10. Then, we compared the neurons’ corresponding salience values over time in different parts of the basal ganglia between different dopamine levels. We compared varied dopamine levels to the control, a healthy brain, through the use of graphs.

Conclusions

The control (Figure 4), which models a normal, healthy brain, shows that the channels’ saliences are distinct from one another. When dopamine signals were adjusted to lower strengths (Figure 3), similar to that of those with Parkinson’s disease, the saliences become more similar amongst cell channels. When looking at the GPe cell between a brain affected Parkinson’s and a healthy brain, Channel 1 and 2 in the brain with Parkinson’s has salience values a lot closer to that of Channel 6. The GPs in particular is responsible for voluntary movement, and so the closer salience values suggest that people with Parkinson’s may have trouble with decision making, as they are unable to prioritize actions to take.

On the other hand, when the dopamine levels were adjusted to be higher than normal (Figure 5), simulating the effects of administering too much L-Dopa, one individual channel had an extremely high level of salience. Channel 6 had high values of salience throughout while Channel 1 and 2 had much smaller values, suggesting that patients with too much dopamine may exhibit recklessness.

Conclusions from biological studies examining the decision-making impairments in patients with Parkinson’s disease support the results shown by the graphs generated by our model. While our model suggests that dopamine levels affects one’s decision making skills, since it is a simplified model, there are other variables that we did not account for. For example, genetics, age, and gender were not included in our model. Also, since it is an abstract network model, the biological aspects of the CBG model may not be accurate. In the future, we hope to conduct studies on patients in order to verify the validity of our model. Also, although Parkinson’s patients take L-Dopa to artificially stimulate the production of dopamine in their bodies, the drug’s effects wane after it is ingested for a long period of time. We can further modify our model by increasing the length of time it models and include the effects of L-Dopa wearing off. This would likely give us a better understanding of the long term cognitive effects of Parkinson’s disease.