Parkinson’s Disease and Cell Communication

EnvisionSTEM

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Cell communication is a process that allows cells to transmit signals to each other through complex pathways. Through this, cells become capable of coordinating with other cells and producing appropriate responses to their environment. A key factor in cell communication is the signal transduction pathway. This pathway is the general way a signal is transmitted through a cell and involves three major steps: reception, transduction, and response. The reception stage is when a signaling molecule, a ligand, binds to a specific receptor protein, triggering the start of the signal transmission. Transduction is when this signal is amplified through a series of reactions, known as cascades. The final action is the response, in which the signal has been effectively communicated and actions such as gene expression, enzyme activation, or cellular movement can take place.

Parkinson's Disease (PD) is a neurodegenerative disorder or a disorder of the nervous system that causes difficulty with motor skills, balance, and coordination. PD is the second most common neurodegenerative disorder and prominently affects those 60 years or older. However, around 5% of people are diagnosed with PD before they reach 50 years old (*National Institute of Neurological Disorders and Stroke*, n.d.). This disease primarily stems from the deterioration of nerve cells in the basal ganglia, an area of the brain in charge of movement (*National Institute on Aging*, n.d.). These nerve cells located in the substantia nigra are responsible for producing dopamine, a neurotransmitter essential for the coordination of movement, that also serves as a reward pathway in the human brain (*Madame Curie Bioscience Database*, 2013). The dopamine affects the striatum, which in turn affects the thalamus, a part of the brain responsible for relaying motor information. When the brain sends signals involving movement, it refines these movements using cells that need dopamine. However, as the nerve cells become damaged, dopamine levels begin to deplete and disrupt communication between the basal ganglia and other

parts of the brain, including the thalamus, ultimately accelerating PD (*Cleveland Clinic*, 2024). Additionally, in those diagnosed with PD, the majority of their brain cells contain Lewy Bodies— toxic clumps of alpha-synuclein (a protein associated with movement disorders). When these Lewy Bodies affect the substantia nigra in the ventral midbrain, symptoms such as muscle stiffness and tremors emerge (American Parkinson Disease Association, 2024). The effect of decreased dopamine levels is vital as it interferes with a cell's ability to communicate, and its symptoms go on to affect daily life and induce challenges in basic activities such as walking or even speaking.

In a normal signaling pathway, one in which Parkinson's Disease is not present, healthy neurons release dopamine into the striatum, which can trigger the direct pathway of the basal ganglia. The direct pathway involves neurons from the striatum sending inhibitory signals to the GPi, which allows the thalamus to become more active (Kenhub, 2023). The thalamus is then able to send stronger signals to the cortex, where movement control is established. In this case, the signaling molecule is dopamine, and its receptors are dopamine receptors. The basal ganglia, cortex, and thalamus then all work together, creating a cascade of reactions to convey the signal throughout the cell (Kenhub, 2023.). Finally, the signal is conveyed, and the execution of the movement is done without challenges. However, in the pathway affected by PD, this is not the case. The neurons that produce dopamine begin to die and cause a depletion in dopamine levels, affecting the reception signals throughout the rest of the cell. Without enough dopamine, communication between the basal ganglia and other parts of the brain becomes inefficient. The reduction of the triggering of responses in the transduction stage of the pathway causes abnormalities such as neuronal damage and dysfunction.

There are currently no treatments that will cure Parkinson's, but treatments, such as therapeutic services, can go a long way to relieve the symptoms of PD. The National Institute of Neurological Disorders and Stroke has been funding research to advance treatment options for PD. Dopamine replacement therapy helped reduce motor struggles in the early stages of PD, and deep brain stimulation reduced tremor and rigidity, but these therapies only provide temporary relief and not a permanent cure. Scientists believe that imbalances in the calcium found in membranes of cells lead to their dysfunction and could be an accelerated PD. A current NINDS study is testing out a neuroprotective treatment that controls the calcium levels of recently diagnosed individuals in hopes of finding a balance and slowing down Parkinson's. Another potential treatment is to deliver AAVs trained to produce GDNF proteins that produce dopamine. By stimulating the production of dopamine in the brain through this protein, scientists hope that dopamine levels will rise and restore normal conditions (*National Institute of Neurological Disorders and Stroke*, n.d.). All of these treatments have potential, but most are still in the process of being tested or will not work long-term. However, the rate of technological advancement is advancing, making it possible to see a permanent cure in the near future.

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