Neuroscience Foundation

Research & Graphics by
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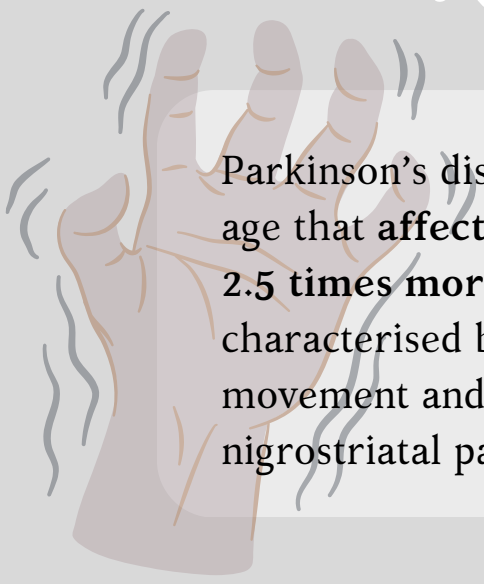
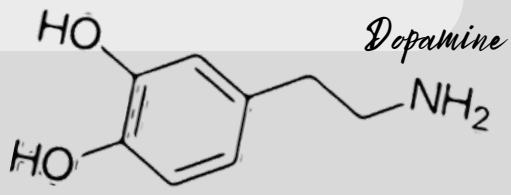
Parkinson's Disease



Parkinson's disease is a movement disorder of middle and old age that **affects 1–2% of the elderly population**. It is **about 2.5 times more prevalent in males** than in females. It is characterised by rigidity, tremors, and poverty of voluntary movement and is associated with the deterioration of the nigrostriatal pathway



Symptoms

The initial symptoms of the disease are mild—no more than a slight stiffness or tremor of the fingers—but as time passes, they inevitably increase in severity.

The most common symptoms of the full-blown disorder are:

- tremors that are pronounced during inactivity
- muscular rigidity
- a marked poverty of spontaneous movements
- difficulty initiating movement
- slowness in executing initiated movements
- a masklike face

Pain and depression often develop before the motor symptoms become severe.
Although Parkinson's patients often display cognitive deficits, dementia is not always associated with the disorder

Causes

Parkinson's disease seems to have no single cause.

The following have all been implicated in specific cases:

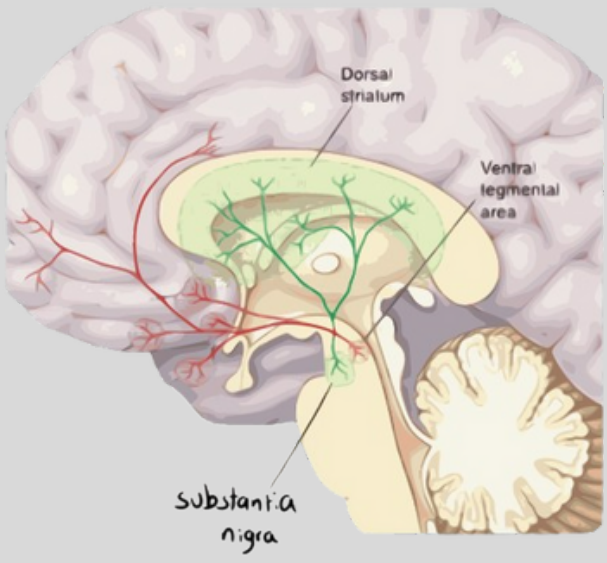
- Faulty DNA
- brain infections
- strokes
- tumours
- traumatic brain injury
- neurotoxins

However, in the majority of cases, no cause is obvious, and there is no family history of the disorder.
Numerous genes have been linked to it**

Neural basis of the disease

Parkinson's disease is associated with widespread neural degeneration, but it is particularly severe in the substantia nigra—the midbrain nucleus whose neurons project via the nigrostriatal pathway to the striatum of the basal ganglia

A small group of nerve cells called the substantia nigra (black substance) make a particular chemical called dopamine, which they deliver to another part of the brain, known as the striatum. As the cells of the substantia nigra die, the amount of dopamine they can deliver goes down. The striatum helps control movement, and to do that normally, it needs dopamine.



Treatment

Although dopamine levels are low in Parkinson's disease, dopamine is not an effective treatment because it does not readily penetrate the blood–brain barrier.

L-dopa, the chemical precursor of dopamine, serves as a treatment for Parkinson's. It readily penetrates the blood–brain barrier and is converted to dopamine once inside the brain. However, l-dopa is not a permanent solution as it loses its effectiveness with continued use, until its side effects outweigh its benefits.

Another treatment for Parkinson's disease, but a controversial one, is deep brain stimulation. It involves chronic bilateral electrical stimulation of a nucleus that lies just beneath the thalamus and is connected to the basal ganglia: the subthalamic nucleus. Once the current is turned on, symptoms are sometimes alleviated within minutes, but the effectiveness of deep brain stimulation slowly declines over the ensuing months. If the stimulation is turned off, the therapeutic improvements dissipate within an hour or two. Unfortunately, deep brain stimulation can also cause side effects such as cognitive, speech, and gait problems.

References: John P. J. Pinel (2014), Introduction to Biopsychology, ninth edition, Pearson

https://www.hopkinsmedicine.org/health/conditions-and-diseases/parkinsons-disease/myths-and-facts-7-parkinson-disease-misconceptions