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OPINION ARTICLE

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A General View on Neurodegenerative Disease

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Description

The progressive loss of structure or function of neurons, known as neurodegeneration, is the cause of a neurodegenerative illness. Cell death could result from such neural injury. Amyotrophic lateral sclerosis, multiple sclerosis, Parkinson's disease, Alzheimer's disease, Huntington's disease, multiple system atrophy, and prion disorders are examples of neurodegenerative diseases. Neurodegeneration can be detected at several levels of neuronal circuitry in the brain, ranging from molecular to systemic. These disorders are considered incurable since there is no known means to halt the steady degradation of neurons; nonetheless, research has demonstrated that oxidative stress and inflammation are two key contributors to neurodegeneration. At the subatomic level, biomedical research has shown several parallels between these disorders, including aberrant protein assemblages and triggered cell death. Because of these commonalities, therapeutic improvements in one neurodegenerative disease may benefit other diseases as well.

Alzheimer's disease

Alzheimer's disease is a chronic neurodegenerative illness that causes the loss of neurons and synapses in the cerebral cortex and some subcortical structures, resulting in temporal lobe, parietal lobe, frontal cortex, and cingulate gyrus atrophy. It is the most frequent kind of neurodegeneration. Despite billions of dollars spent on research, no effective cures for Alzheimer's disease have been discovered. Clinical trials, on the other hand, have generated specific molecules that could transform the future of Alzheimer's disease therapy.

Parkinson's disease

The death of dopaminergic neurons in the substantia nigra, an area of the midbrain, is the hallmark of Parkinson's disease. The cause of this cell death that is selective is unknown. Within afflicted neurons, alpha-synuclein-ubiquitin complexes and aggregates have been found to accumulate in Lewy bodies. Defects in protein transport machinery and regulation, such as RAB1, are thought to be involved in the disease pathogenesis. Alpha-synuclein buildup in Lewy bodies may be caused by impaired axonal transport. Experiments indicated that both wild-type and two familial Parkinson's disease-associated mutant alpha-synucleins move at lower rates across the axons of cultured neurons. Another Parkinson's disease mechanism could be alpha-synuclein-induced membrane damage.

Huntington's disease

Huntington's disease is a neurological ailment caused by mutations in the huntingtin gene. It is a rare autosomal dominant disorder. Loss of medium spiny neurons and astrogliosis are hallmarks of HD. The striatum is the first brain region to be significantly impacted, followed by degeneration of the frontal and temporal cortices. Subthalamic nuclei in the striatum give control impulses to the globus pallidus, which begins and controls motion. As a result of the decreased impulses from the subthalamic nuclei, movement initiation and modulation are diminished, resulting in the disorder's signature movements, particularly chorea.

Multiple sclerosis

Multiple sclerosis is a devastating central nervous system demyelinating disease characterised by an autoimmune response that results in the progressive loss of myelin sheath on neuronal axons. Depending on the site of the lesion, the resulting decrease in signal transduction speed causes a loss of functionality that encompasses both cognitive and motor disability. MS progresses as a result of increased inflammation, which is thought to be caused by the release of antigens like myelin oligodendrocyte glycoprotein, myelin basic protein, and proteolipid protein, which trigger an autoimmune reaction.

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