OPINION ARTICLE

Cause and Symptoms of Age-Related Neurodegenerative Diseases

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DESCRIPTION

With longer life expectancies, the prevalence of age-related neurodegenerative diseases has increased. Unfortunately, the neurodegenerative state of such conditions can often only be diagnosed when symptoms are clearly present and the neurodegeneration status is advanced. Although age-related neurodegeneration is a regular occurrence in living organisms, the understanding of the cellular and molecular processes behind it is still lacking. Typically, mitochondrial stress serves as a common starting point for neurodegenerative pathways, making it a possible target for early detection and therapy. It's interesting to note that the mitochondrial prohibitin complex, a major regulator of ageing and metabolism that has been linked to neurological disorders, has undergone evolutionary conservation. Its function in neurodegeneration, however, is yet poorly known. In many genetic and physiological situations, the PHB complex exhibits both protective and harmful effects and mitochondrial and cellular stress simultaneously up and down-regulate the PHB expression. Understanding PHB's function in neurodegeneration will advance knowledge of how neurons decline as individuals grow and make it easier to spot early molecular indicators of mitochondrial stress.

The prevalence of neurodegenerative disorder is the main cause of impairment globally and one of the biggest public health issues has increased with ageing. The study of the early processes of neurodegeneration will be an important focus of research in the coming years, especially for the identification of new therapeutic targets and molecular markers of disease. This is because the majority of ND is only diagnosed after the nervous system has suffered significant damage and the symptoms become obvious. The various ND are often categorised according to their symptomatology and the region of the nervous system where the neurodegeneration takes place. For instance, Parkinson's Disease (PD), which is marked by tremor and bradykinesia as well as increasing stiffness, is brought on by the loss of dopaminergic neurons in the substantia nigra and the development of Lewy bodies. Similar to Parkinson's disease, Alzheimer's Disease (AD) is characterised by a gradual deterioration beginning in the cortical region and extending to other brain areas, such as the amygdala and hippocampus, as well as the appearance of senile plaques. Additionally, chorea, dementia, and psychiatric issues are brought on by the death of neurons in the brain and striatum caused by the increase of CAG repeats in the huntingtin gene in Huntington's Disease (HD). Muscle weakness and atrophy result from the loss of motor neurons in the cortex, brainstem, and spinal cord in Amyotrophic Lateral Sclerosis (ALS). Finally, behavioural and motor issues linked to frontotemporal dementia cause neurodegeneration in the temporal and frontal cortices. The cause of the neurodegeneration in the aforementioned and other CNS illnesses, however, may differ greatly from one another yet point to mitochondria. Indeed, changes in mitochondrial homeostasis resulting from environmental, genetic, or spontaneous factors may result in mitochondrial stress, which results in DNA, protein, or lipid damage that activates quality-control mechanisms to repair the damage. Although the causes of ND and its subtypes vary, the early stages of neurodegeneration in most of them share pathways connected to early mitochondrial stress.

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