COMMENTARY

DNA Damage in Genetic and Behavioral Anomalies in Aged Human Neurons

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DESCRIPTION

The ability for post-mitotic neurons in the human brain to remain viable throughout an individual's lifetime, genetic integrity must be preserved. The brain has an extraordinarily high degree of mitochondrial metabolic activity because of continuous synaptic plasticity and electrical communication between cells. Reactive oxygen species are produced as a result of this action, with 8-oxo-7,8-dihydroguanine (8-oxoG) being one of the most common oxidation products in the cell. A low basal level of 8-oxoG is critical for the epigenetic regulation of neurodevelopment and synaptic plasticity, whereas an uncontrolled increase in 8-oxoG damages the genome and causes somatic mutations and transcription errors. 8-oxoG is essential for the maintenance and transfer of genetic information into proper gene expression. In the context of rising cellular 8-oxoG, a gradual but continuous build-up of DNA damage has been linked to both normal ageing and neurological diseases like Alzheimer's and Parkinson's. The loss of physiological processes brought on by ageing, a multi-modal complicated process, increases the risk of disease and mortality. For sporadic neurodegenerative diseases like Alzheimer's and Parkinson's, ageing is the most important risk factor.

Cellular damage is dangerous because DNA is the framework for all cellular operations, and is one of the several biological components that are theorised to contribute to age-induced neurological abnormalities. In the 1930s, it was suggested that an organism's mutations may have a detrimental impact on fitness. Since then, a number of causes have been identified as causing DNA mutational damage, including radiation, environmental carcinogens, spontaneous deamination because nucleic acids are inherently unstable, and oxidative metabolic by-products. In actuality, the human genome can experience up to 120k lesions every day. DNA damage that is sustained over time can result in genotoxic stress signalling, which causes cell death. The cell utilizes highly conserved systems to detect DNA damage and harmful agents to repair DNA lesions to prevent irreversible alterations in order to defend against this continuous genomic risk. For optimal homeostasis of all cellular activities, even in highly demanding neurons, the long-term maintenance of genomic integrity is essential.

Since neurons need around 80% of the energy in the brain to sustain their signalling activity, they are among the most metabolically demanding cells in the body. Because neurons are post-mitotic and cannot be replaced, they are particularly vulnerable to age-related damage and must endure for the duration of an organism. Increased neuronal activity-dependent brain regions, such as those with strong synaptic connections, have higher energy needs. As a result, neurons have high oxidative metabolic needs and are therefore more vulnerable to severe oxidative damage. The free radical theory of ageing, in the 1950s, asserts that age-dependent accumulation of oxidative damage to cellular macromolecules results in a progressive deterioration of cells, tissues, and organs required for the function of the organism. Oxidative damage to a cell has long been linked to ageing. Throughout a person's lifespan, hydrolysis, interaction with reactive metabolites, or exposure to environmental pollutants can cause DNA damage. Heat-induced frequent de-purination causes human neurons to lose 108 purines, or 3% of their total purine residues, from their DNA during the course of a person's lifetime. Although pyrimidines are more resistant to these pressures, they are nevertheless vulnerable to free radicals.

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