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Antioxidant Therapeutic Strategies in Neurodegenerative Diseases

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Introduction

The pathogenic features of neurodegenerative diseases include mitochondrial dysfunction and the generation of derived reactive oxygen species. The neural tissue is extremely sensitive to oxidative stress, which is a major cause of both chronic and acute neurodegeneration. Based on this, therapeutic strategies that use antioxidant molecules to achieve redox equilibrium have been widely used to treat a variety of brain pathologies. Polyphenols, carotenes, and vitamins are among the most common exogenous antioxidant agents that have been tested as adjunctive therapies in neurodegeneration. Other types of antioxidants, such as hormones like the widely used melatonin, are also considered neuroprotective agents and have been used in various neurodegenerative contexts [1].

Description

Neurodegenerative diseases are a diverse group of disorders characterised by the progressive loss of function and death of specific groups of neurons, which results in the disease's clinical manifestation. Changes in specific proteins cause dysfunction of various cellular pathways, including increased numbers of reactive oxygen species resulting from mitochondrial dysfunction, excitotoxicity, and synaptic dysfunction, impairment of protein degradation systems, endoplasmic reticulum stress, DNA damage, inflammation, and cell cycle reentry. Their complex interaction makes understanding the mechanisms that cause neurotoxicity and cell death difficult, as well as finding an effective treatment [2].

The human brain, which accounts for about 2% of total body weight, receives 15% of cardiac output and consumes about 20% of total basal oxygen. According to the hypothesis that neurodegeneration has a mitochondrial basis, oxidative tissues with high energy demands are the most vulnerable to oxidative phosphorylation system defects. Furthermore, the most metabolically active areas of the brain (the cortex, particularly the motor cortex and thalami, which receive three times the blood flow of white matter) are the most susceptible to hypoxic ischemic encephalopathy. According to this, antioxidant therapeutic strategies for the treatment of various brain pathologies in order to restore redox equilibrium by scavenging free ROS are a promising approach [3].

The inner mitochondrial membrane is highly folded and impervious to almost all molecules and ions, forming the mitochondrial cristae in which the OXPHOS enzymatic complexes are embedded. Through the transfer of electrons by the five mitochondrial enzymatic complexes, OXPHOS enables the synthesis of ATP coupled to oxygen consumption. Electrons flow through the mitochondrial respiratory chain via oxidation-reduction reactions, eventually arriving at complex IV, where oxygen serves as the final receptor for the electrons and is reduced to H2O. Thus, oxygen is consumed in the OXPHOS, and an electrochemical gradient is established, driving ATP synthesis. When OXPHOS fails, intermediate reactive metabolites derived from oxygen, known as ROS, are produced, making mitochondria the primary source of ROS [3].

Under physiological conditions, all antioxidant mechanisms reduce ROS production and thus act as oxidative stress defence systems. However, in the presence of mitochondrial dysfunction, ROS production may exceed the detox threshold, jeopardising cell viability. Furthermore, even modest endogenous antioxidant defence makes the brain more vulnerable to oxidative stress. That is, the brain's comparatively low endogenous antioxidant defence in comparison to many tissues makes it vulnerable to disrupted redox homeostasis. Beyond the protective endogenous antioxidant enzymatic defence cell mechanisms, exogenous antioxidants, including those administered through diet, such as polyphenols, carotenes, and vitamins, have been widely described in the literature to play a role in redox balance, including in the context of neurodegeneration [4,5].

Conclusion

In conclusion,exogenous adjunctive interventions, the majority of the antioxidant agents recently tested in the randomised controlled clinical trials discussed herein belong to the polyphenol, carotenes, fatty acid, and vitamin families. However, the antioxidant and neuroprotective power of these groups is not limited to these groups, as other antioxidant molecules such as nucleosides, hydrogen gas, or hormones have been tested in clinical trials against neurodegenerative disorders, primarily Parkinson's disease, in the last year. Melatonin, a hormone, is one of the most frequently studied antioxidant molecules in a wide range of diseases, including neurodegenerative disorders. Two of the most recent randomised clinical trials using antioxidants in neurodegenerative diseases found in this review used melatonin in different neurodegenerative contexts.

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Conflict of Interest

There are no conflicts of interest by author.

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