

Letter to the Editor

Degenerative and Protective effects of Nicotine in Neurodegenerative diseases

Dear Editor ,

Aging is the greatest risk factor for neurodegenerative diseases. Progressive loss of structure or function of neurons, including death of neurons considered as neurodegeneration. Cell death in neurodegeneration is due to apoptosis and most commonly through the intrinsic mitochondrial pathway genetic mutations. Neurodegenerative (ND) Diseases are chronic, progressively debilitating, and incurable. There are different types of neurodegenerative diseases like Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD). Among different types of Neurodegenerative diseases, Alzheimer's disease appears to be the most common. The disabling effects for individuals with Neurodegenerative diseases may continue for years or even decades. The prevalence of Neurodegenerative diseases has increased globally, most noticeably in the aging populations of the developed world.

Alzheimer's disease is characterised by loss of neurons and synapses in the cerebral cortex and certain subcortical regions which results in gross atrophy of the affected regions, including degeneration in the temporal lobe and parietal lobe, and parts of the frontal cortex and cingulate gyrus. Parkinson's disease is known degenerative CNS disorder that affects movement.

The non motor symptoms like reduced ability to smell, rapid eye movement during sleep, gastrointestinal and urogenital functions, and postural hypotension are the other systemic alterations which are common in Parkinson's disease. Pneumonia, heart conditions, and muscle weakness are the other systemic alterations in Huntington's disease

Recent literatures stated that activation of nicotine receptor by lower doses of nicotine results in apoptotic cell death in demonstrating the ability of nicotine in mediating apoptosis .

Exposure to nicotine during the brain growth spurt period in rats has been associated with significant reduction of the Purkinje cell numbers in the cerebellar vermis. Chronic nicotine exposure during adolescence produced long-lasting impairments in contextual learning during adulthood.

Nicotine may be useful for treating movement problems associated with L-dopa therapy in Parkinson's disease, as nicotine reduces L-dopa-induced abnormal involuntary movements in different Parkinsonian animal models. If nicotine would not lead to a complete termination of progression of CNS disorders, it could at least slow down the progression and eventually attenuate the symptoms of the disease.

Early detection of various Neurodegenerative diseases is urgently needed to improve patients' quality of life and to provide necessary health and social care.

References

1. DiMauro S, Schon EA (2008). "Mitochondrial disorders in the nervous system". *Annual Review of Neuroscience*. 31:91-123.
2. Quik M, Huang LZ, Parameswaran N, Bordia T, Campos C, Perez XA (2009). Multiple roles for nicotine in Parkinson's disease. *Biochem Pharmacol*. 78:677-685.
3. Balfour D. J. K., Fagfrstrom K.O(1996). Pharmacology of nicotine and its therapeutic use in smoking cessation and neurodegenerative disorders. *Pharmacol. Ther.* 72: 51-81, 1996.
4. Chen A, Russell B. Edwards, Ronald D. Romero (2003). Long term nicotine exposure reduces Purkinje cell number in the adult rat cerebellar vermis. *Neurotoxicology & Teratology*. 25: 325-334.
5. Francois Berger, Fred H. Gage, and Sukumar Vijayraghavan (1998). Nicotinic Receptor-Induced Apoptotic Cell Death of Hippocampal Progenitor Cells. *The Journal of Neuroscience*. 18(17):6871-688

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