

Neurodegenerative Diseases Symptoms and Treatment

Chapter 3

Neurobiology of Parkinson's Disease: An Insight into Cholesterol Involvement

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1. Introduction

Parkinson's disease (PD) is the second most prevalent neurodegenerative disorder, after Alzheimer's disease (AD). Globally 0.01% of total population are suffering from PD. In India, the prevalence frequency for PD is 0.07%, which is higher than the global trend of PD occurrence [1]. Recent studies have shown that occurrence of PD is in increasing inclination, which is a great concern to human health [2,3]. Clinically PD is characterized by four classical symptoms namely: bradykinesia, rigidity, resting tremor, and postural instability [4,5]. These four cardinal symptoms are like a TRAP, which makes the patient to end up the rest of his life on wheelchair. Behavioural impairment in PD has been associated with the loss of nigrostriatal dopaminergic (DAergic) functions in a correlated manner [6-9]. Substantia nigra (SN) is the region of mid-brain have been found to be linked with PD [10]. The DAergic neurons of SN are known to secrete dopamine (DA) [11,12], which regulates the goal oriented behaviour, reward, and overall neuromuscular coordination in body [13-15]. Neuronal loss in SN creates a scarcity in DA level which precipitates in the behaviour of PD patients [16]. The hallmark pathological features of PD is the presence of 'Lewy Body' (LB), which is nothing but aggregation of α -synuclein with some other aggregate prone proteins [17]. It has been reported that, the function of α -synuclein is to assist the pre-synaptic

vesicles during the process of neurotransmission and after the process α -synuclein degrades with proper assistance of lysosome [18]. Degradation of α -synuclein maintains the optimum level of α -synuclein in cytosol [19]. However, in diseased condition, it has been found that the α -synuclein is becoming aggregate prone and increasing its level due to impairment in its degradation pathway [20].