



Evolution and Circumstances of Parkinson's Disease

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DESCRIPTION

Parkinson's disease is one of the main neurological disorders affecting the aged. It is an environmentally influenced, neurodegenerative disease that is characterized by tremors, stiffness of limbs and trunk, slowness of voluntary movements called Bradykinesia, postural instability and a distinctive shuffling gait with a stooped position called "Parkinson's gait". According to one estimate more than 1% of the population suffers from Parkinson's disease after reaching 55 years of age. Most of the movement-related symptoms of Parkinson's disease are caused by a lack of dopamine due to the loss of dopamine-producing cells in the substantia nigra pars compacta of the brain. Although it is well known that lack of dopamine causes the motor symptoms of Parkinson's disease; it is not clear why the dopamine producing brain cells deteriorate. Genetic and pathological studies have revealed that various dysfunctional cellular processes, inflammation, and stress can all contribute to cell damage. In addition, abnormal clumps called Lewy bodies, which contain the protein alpha-synuclein, are found in many brain cells of individuals with Parkinson's disease. The role of these clumps in regards to Parkinson's disease is not understood. In general, scientists suspect that dopamine loss is due to a combination of genetic and environmental factors.

There have been numerous studies linking Parkinson's disease to environmental factors. A positive correlation between Parkinson's disease and industrialization has been well documented in the literature implicating pesticides, herbicides and heavy metals as contributory factors to the development of Parkinson's, the "environmental disease". These studies further support a holistic approach to this disease which focuses on reduction and removal of the individual's overall exposure. This augments the conventional treatment, which focuses on symptoms thus providing more holistic choices for the patient. Based on epidemiological studies, occupational exposure to specific metals, manganese, copper, lead, iron, mercury, zinc and aluminium appear to be a risk factor for Parkinson's disease. An analysis of the Parkinson's disease mortality rates in Michigan (1986–1988) with relation to heavy metal exposure revealed that

counties with an industry in the paper, chemical, iron or copper related-industrial categories had significantly higher Parkinson's death rates than counties without these industries. Similarly an increased risk for Parkinson's disease from prolonged occupational exposure to heavy metals was established in Valleyfield, Quebec (1987-1989). Post mortem analysis of brain tissues from patients with Parkinson's disease gives further confirmation to the involvement of heavy metals and this disorder.

These previous studies have been done on occupational exposure and Parkinson's disease. However, in a recent study on U.S. urban communities, long-term environmental exposure was found to contribute to the development of this disease. This 2003 study examined 35,000 Parkinson's disease patients who have not changed residence since 1995 and found an increased incidence in Parkinson's disease around urban areas with metal emitting facilities. With all this evidence supporting the environmental link and Parkinson's disease, the approach to treating this disease should also address the potential underlying causal factors.

CONCLUSION

Metals are ubiquitous and play a critical role in neurobiology. Transition metals are important because they alter the redox state of the physical environment. Biologically, transition metals catalyze redox reactions that are critical to cellular respiration, chemical detoxification, metabolism, and even neurotransmitter synthesis. Many metals such as iron, zinc, copper, and manganese are both nutrients and neurotoxins (at higher levels). Other metals, such as lead and cadmium, are metabolized to these metals, particularly iron. Oxidative stress phenomena have been allied with the onset of neurodegenerative diseases. There is a growing amount of evidence pointing to a role of mitochondrial damage as a source of free radicals involved in oxidative stress.

Among the elements that participate in the production of oxygen reactive radicals, transition metals are one of the most significant. Several reports have implicated the involvement of

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redox-active metals with the onset of different neurodegenerative diseases such as Alzheimers disease, Parkinson's disease and Amyotrophic lateral sclerosis. The diagnosis of Parkinson's disease is entirely clinical with no biochemical tests shortly available to diagnose Parkinson's disease. Current diagnosis is prepared by standard neurological examination and medical history.

The severity of the disease is characterized as stages based on the overall motor function evaluation using the Unified Parkinson's

Disease Rating Scale (UPDRS) or Hoehn and Yahr scale or Schwab and England Activities of Daily Living Scale. Three major cardinal symptoms of PD are tremor, rigidity and motor dysfunction, which significantly help in the detection of disease. However, the clinical diagnosis fails to identify the Parkinson's disease before substantial loss of dopamine neurons occurs. Hence, there is a necessity for early detection and more effective drugs to stop the progression of neural degeneration.