Distinct Neuroimaging Features Define Parkinsons Disease and Welding-Related Neurotoxicity

Lee EY1*, Lewis MM1,2, Mailman RB1,2 and Huang X1,2,3,4,5

¹Department of Neurology, Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033, USA

²Department of Pharmacology, Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033, USA

³Department of Radiology, Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033, USA

⁴Department of Neurosurgery, Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033, USA

⁵Department of Kinesiology, Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033, USA

Corresponding author: Eun-Young Lee, Department of Neurology, Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033, USA, Tel: 5736732989; E-mail: elee@pennstatehealth.psu.edu

Received date: September 19, 2017; Accepted date: September 22, 2017; Published date: September 25, 2017

Copyright: © 2017 Lee EY, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Editorial

Neurobehavioral disorders are a signif cut and growing health, problem worldwide. economic and social Age-related neurodegenerative disorders contribute signif cLht`mto this growing problem because of increased longevity in the population. Parkinson's disease (PD) is the second most common neurodegenerative disorder U er Alzheimer's disease, U ecting 1% of the population over 60 yrs [1]. It is marked clinically by motor dysfunctions, e.g., resting tremor, bradykinesia and rigidity and pathologically by -synuclein-positive Lewy bodies and nigrostriatal dopamine neuron loss in the substantia nigra pars compacta (SNpc) of the basal ganglia (BG) [2]. Despite a growing number of associated genetic factors, the exact cause(s) for PD are unknown [3]. Because the majority of PD patients do not have a family history and identical twins are o en discordant for the disease, there is increased interest in the role that environmental and occupational toxicant exposure may play in PD aetiology [48]. For example, previous studies reported that exposure to well water, pesticides, herbicides and certain metals such as manganese (Mn), iron (Fe), lead (Pb) and mercury (Hg), or occupations like welding and farming, were associated with developing Parkinsonian symptoms [6], a clinical syndrome that presents both in PD and a number of look-alike disorders

Since Couper [7] first reported Mn-induced Parkinsonism in 1837, signif cLite ort has been exerted to determine potential links between Mn-induced neurotoxicity and PD. Recent studies reported that Mnexposed workers had a higher prevalence of Parkinsonian features compared to unexposed workers [9,10] and the Parkinsonian motor symptoms exacerbated with cumulative long term Mn-exposure [11]. Welders have been among the most studied occupational groups since microstructural changes due to compromised dopaminergic systems, this MRI modality may serve as a useful long-term marker to assess welding-induced microstructural changes and help further dissociate them from PD.

Overall, the current clinical, pathological and neuroimaging f ndings suggest that welding-related neurotoxicity is distinct from PD. It is possible, however, that Mn-exposure may contribute to an atypical presentation of idiopathic PD and further research can ultimately lead to better diagnoses and treatment.

References

1. Tysnes OB, Storstein