

Solvents and Parkinson's—Is There a Connection?

Abdullah Hafid^{1,2} and Nicholas A Kerna^{3*}

¹University of Health and Humanities, Tortola, BVI ²University of Science, Arts and Technology, Montserrat, BWI ³SMC–Medical Research, Thailand

*Corresponding Author: Nicholas A Kerna, POB47 Phatphong, Suriwongse Road, Bangkok, Thailand 10500. Contact: medpublab+drkerna@gmail.com

Received: March 23, 2020; Published: March 28, 2020

DOI: 10.31080/ecne.2020.12.00677

Keywords: Breast Milk; Parkinson's Disease; Parkinsonism; Methanol; Solvents; Toluene

Abbreviations

PD: Parkinson's Disease; PERC: Perchloroethylene; TCE: Trichloroethylene

Background

Solvents consist of a large variety of substances that dissolve other compounds. Solvents are available in a wide range of products, such as petroleum, paints, pastes, ointments, degreasers, and cleaning items [1]. Methanol, toluene, and chlorinated solvents have drawn particular attention relating to specific cognitive disorders, such as Parkinson's disease (PD) [2].

Methanol is categorized as a solvent and utilized in a variety of applications, from serving as a fossil fuel additive to a primary building block of plastic and formaldehyde [1]. While these products are near-ubiquitous in human society, humans have various routes of absorption for methanol. These absorption-routes include inhaling methanol fumes, dermal introduction to substances laden with methanol, or oral ingestion of specific fluids [3].

In humans, methanol intake or exposure can yield parkinsonian-like symptoms or parkinsonism [4], which include, but are not limited to, lethargy, anxiety, confusion, ataxia, and visual impairment, most commonly. Diminished visual-spatial aptitude, frontal lobe activity, recall memory, and increased cerebellar atrophy have been observed in people who have been exposed to a high amount of solvents, or have had low-grade but long-term exposure to solvents [5].

Toluene is a methylbenzene chemical found in paint, paint thinner, glue, or ink. "On the street", in back alleys, or schools, it is abused to "get high" or feel euphoric temporarily. Chronic users demonstrate altered brain chemistry, while acute symptoms of short-term use include cognitive changes, euphoria, headache, and ataxia—which are typically reversible when the "drug" has worn off [1].

In a meta-analysis by Goldman., *et al.* (2011), trichloroethylene (TCE) and perchloroethylene (PERC) showed a statistically-significant association with PD; TEC (OR 6.1, 95%CI 1.2–33, p = 0.034) and PERC (OR 10.5, 95%CI 0.97–113, p = 0.053), respectfully. Also, concomitant use of TEC and PERC increased PD incidence significantly; (OR 8.9, 95%CI 17–47, p = 0.01) [2].

Summary

Parkinson's disease is a degenerative disease of the substantia nigra of the central nervous system, affecting men 40–70 years of age mostly. Symptoms appear gradually with the hallmark tremor, rigidity, hypokinesia, gait disturbances, and speech and writing difficulties [5].

Methanol, toluene, TCE, PERC, or other solvents have been utilized in one form or another for nearly a century [6]. While the use and application of specific solvents are controlled and restricted, sectors of the population have been and remain vulnerable to these solvents. Even with stringent controls and restrictions for their use, detectable levels of these solvents remain throughout the environment, including over twenty-five percent of U.S. water systems—and in female breast milk [6].

Various helpful solvent inadvertently act as ecological toxins, and for those exposed to them, might play a role in parkinsonism or Parkinson's disease; however, to date, the data is inconclusive and, at times, conflicting [7]. The degree to which environmental solvents contribute to the pathophysiology of Parkinson's disease remains unclear. Numerous demographics and ecological components might be factors in the etiology of Parkinson's disease or affect the severity of the disease [1]. While age is a significant risk factor for the disease, exposure to specific solvents might be contributory.

Conflict of Interest Statement

The authors declare that this paper was written in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

References

- 1. Lock E., *et al.* "Solvents and Parkinson disease: A systematic review of toxicological and epidemiological evidence". *Toxicology and Applied Pharmacology* 266.3 (2013): 345-355. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3621032/
- 2. Goldman S., *et al.* "Solvent exposures and Parkinson disease risk in twins". *Annals of Neurology* 71.6 (2011): 776-784. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3366287/
- Ohlson C and Hogstedt C. "Parkinson's disease and occupational exposure to organic solvents, agricultural chemicals and mercury--a case-referent study". Scandinavian Journal of Work, Environment and Health 7.4 (1981): 252-256. https://www.ncbi.nlm.nih.gov/ pubmed/7347910
- Keener AM and Bordelon YM. "Parkinsonism". Seminars in Neurology 36.4 (2016): 330-334. https://www.ncbi.nlm.nih.gov/ pubmed/27643900
- 5. Van der Mark M., *et al.* "Occupational exposure to solvents, metals and welding fumes and risk of Parkinson's disease". *Parkinsonism and Related Disorders* 21.6 (2015): 635-639. https://www.ncbi.nlm.nih.gov/pubmed/25903042
- 6. Chin-Chan M., *et al.* "Environmental pollutants as risk factors for neurodegenerative disorders: Alzheimer and Parkinson diseases". *Frontiers in Cellular Neuroscience* 9 (2015): 124. https://www.ncbi.nlm.nih.gov/pubmed/25914621
- 7. Anderson F., *et al.* "Inflammasomes: An emerging mechanism translating environmental toxicant exposure into neuroinflammation in Parkinson's disease". *Toxicological Sciences* 166.1 (2018): 3-15. https://www.ncbi.nlm.nih.gov/pubmed/30203060

Volume 12 Issue 4 April 2020

©2020. All reserved by Abdullah Hafid and Nicholas A Kerna.