What is the Mechanistic Evidence for Trichloroethylene as a Cause of Parkinson’s Disease?

**ABSTRACT**

Parkinson’s disease (PD) is a neurodegenerative disorder characterized by movement disorders. Clinical features include bradykinesia (reduction in movement), rigidity (stiffness and tightness of muscles), akinesia (lack of spontaneous movement), and postural instability, shuffling gait, and freezing gait.

PD is a disease that affects older adults, occurring in about 1-2% of individuals older than 60 years of age. The incidence increases with age, with nearly 1% of individuals older than 80 years of age having PD. The prevalence of PD in the United States is estimated to be 1.3 million people, with the majority of cases occurring in people older than 65 years of age.

PD is a complex and heterogeneous disease, with symptoms including tremor, rigidity, bradykinesia, and postural instability. The cause of PD is unknown, but it is believed to be a result of the gradual loss of dopaminergic neurons in the substantia nigra pars compacta (SNpc), leading to a decrease in dopamine production and release in the striatum. The exact mechanisms underlying the development of PD are not fully understood, but several theories have been proposed, including environmental, genetic, and metabolic factors.

In this study, we evaluated the hypothesis that trichloroethylene (TCE) is a causal or substantial contributing factor in human PD. We conducted a comprehensive review of the literature on the topic, focusing on toxicological evidence for the hypothesis that TCE exposure can lead to the development of PD.

**BACKGROUND**

The prevalence of PD in the United States is estimated to be 1.3 million people, with the majority of cases occurring in people older than 65 years of age. The incidence increases with age, with nearly 1% of individuals older than 80 years of age having PD. The prevalence of PD in the United States is estimated to be 1.3 million people, with the majority of cases occurring in people older than 65 years of age.

In this study, we evaluated the hypothesis that trichloroethylene (TCE) is a causal or substantial contributing factor in human PD. We conducted a comprehensive review of the literature on the topic, focusing on toxicological evidence for the hypothesis that TCE exposure can lead to the development of PD.

**OBJECTIVE**

The objective of this study was to evaluate the hypothesis that trichloroethylene (TCE) exposure can lead to the development of Parkinson’s disease (PD). We conducted a comprehensive review of the literature on the topic, focusing on toxicological evidence for the hypothesis that TCE exposure can lead to the development of PD.

**RESULTS**

Chronic, systemic, and intracerebral exposure to TCE has been associated with changes in the substantia nigra pars compacta (SNpc), the brain region that produces dopamine. These changes include decreased dopamine synthesis, decreased dopamine release, and increased alpha-synuclein expression. These findings are consistent with the pathological hallmarks of PD, including the presence of Lewy bodies, which are aggregates of alpha-synuclein.

**CONCLUSION**

The results of this study suggest that trichloroethylene is a causal or substantial contributing factor in human PD. Further research is needed to confirm these findings and to identify potential mechanisms by which TCE exposure may lead to the development of PD.

---