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THE RELATIONSHIP BETWEEN ENVIRONMENTAL TOXINS AND PARKINSON'S DISEASE

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ABSTRACT

Although Parkinson's disease does have genetic factors in the equation, it's important not to discount the importance of the environment. Some see a link to environmental toxins like pesticides and heavy metals. In a world where these toxins are in abundance, it's hard not to fear for our future health. However, direct, long-term exposure to these toxins doesn't cause Parkinson's disease on its own. In this comprehensive review, the changes in our brain as a result of long-term exposure to environmental toxins are thoroughly examined. How Parkinson's disease develops as a result of these chemical changes is described in detail. Along with showing how the problem is created, this review offers a few solutions.

Keywords: Toxins, Parkinson's disease, Pesticides, Agent Orange

Introduction

Before we dive deeper, it's important to understand what Parkinson's disease is. Parkinson's disease is a brain disorder that causes uncontrolled movements, such as shaking, stiffness, and tremors, difficulty with balance and coordination. Symptoms usually worsen over time as people may gradually have difficulty walking and talking. Other symptoms that can develop over time include mental and behavioral changes, depression, memory difficulties, sleep problems, and fatigue. People over 60 are the highest at-risk group. A notable cause of Parkinson's disease is when nerve cells in the basal ganglia, an area of the brain that controls movement, are impaired and/or die. These neurons produce dopamine, so the death of these neurons leads to less dopamine being produced.

Studies have shown that long-term exposure to toxins like pesticides/herbicides and Manganese and other metals have a correlation to developing Parkinson's disease. However, there are other toxins that we should look out for like MPTP, which is known to cause Parkinsonism, a

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syndrome similar to Parkinson's disease. Another interesting finding is that Agent Orange, a toxin used in the Vietnam War, is something the US Department of Affairs associates with Parkinson's disease even though there haven't been many cases where long-term exposure to Agent Orange has led to development of the brain disorder. The list of toxins that have already caused or may cause Parkinson's disease is long and is always developing. Therefore, this review will focus on only a few of these toxins and go into detail into how exactly they can be huge contributing factors for people to develop Parkinson's disease. It is important to understand the effects of these toxins to know how to maintain a safer environment.

Pesticides

Many pesticides contain the insecticides rotenone and permethrin (found in clothing and nets that are used to kill mosquitoes). Both of these insecticides induce the death of dopaminergic neurons. Another way that pesticides can cause neuron death involves the fact that they can be inhibitors of the oxidative phosphorylation system. Oxidative phosphorylation is a cellular process that involves the reduction of oxygen to generate more chemical energy in the form of ATP. Dysfunction of this system can negatively affect neurogenesis, leading to cell death. As of recently, scientists have been trying to gather evidence in favor of the hypothesis that early exposure to pesticides during the prenatal period can affect the nervous system and increase the chance of developing Parkinson's disease. Different rodent models have been tested and they have successfully supported this hypothesis. It is important for babies to stay away from areas where a lot of pesticides are used like farms.

The potential epigenetic consequences of pesticide exposure on brain health have also been highlighted by recent study. Gene expression changes referred to as epigenetic changes don't include changes to the underlying DNA sequence. Studies have demonstrated that specific pesticides can alter the expression of microRNA, histone changes, and DNA methylation patterns in brain cells. These epigenetic changes may affect how genes involved in neurotransmission, neurodevelopment, and inflammation are regulated, ultimately making dopaminergic neurons and the nervous system as a whole more vulnerable. Additionally, even in people who do not have a direct occupational exposure to pesticides, the cumulative effects of repeated low-level pesticide exposures over time are being looked into as a potential contributor to neurological illnesses like Parkinson's. It is crucial to push for stricter regulations and safety precautions to reduce the potential risks associated with pesticide use, especially in settings where infants and young children are present, as our understanding of the complex interactions between pesticides and neural physiology grows.

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Manganese

Manganese is known to have the ability to accumulate in the basal ganglia and may cause a parkinsonian syndrome called manganism. Prolonged occupational exposure (>1 mg/m^3) represents a big risk to Parkinson's disease, as Manganese is commonly found in mining and welding sites. Mn toxicity is characterized by motor impairments such as hypertonia, good bradykinesia, and of course, neurodegeneration that may cause Parkinson's disease. In studies done on humans and animals, Mn concentrations in cells have an inverse correlation to dopamine levels, providing further proof.

Additionally, via significant research, the mechanisms causing manganese neurotoxicity and its connection to Parkinson's disease are progressively coming into focus. In the basal ganglia, manganese can disturb cellular homeostasis and cause oxidative stress, which can accelerate the degeneration of dopaminergic neurons. The ensuing motor dysfunctions, such as bradykinesia and hypertonia, are consistent with Parkinson's disease's clinical symptoms. The levels of dopamine, a neurotransmitter essential for motor control and mood modulation, have been shown to be significantly inversely correlated with manganese concentrations within brain cells in research using both human subjects and animal models. This highlights the complex interactions between manganese exposure and the dopaminergic system and suggests that neurodegeneration brought on by manganese exposure may play a role in Parkinson's disease development.

Strategies for reducing the dangers associated with manganese are being investigated as researchers delve deeper into these linkages. One such technique entails controlling manganese absorption through dietary adjustments. One potential strategy to reduce manganese buildup is to consume enough iron. Higher iron levels may assist decrease the uptake of manganese, potentially lessening the danger of excessive manganese accumulation in the basal ganglia. Iron and manganese compete for absorption in the body. As our knowledge of the subtleties of manganese toxicity expands, it is crucial to address occupational exposures in high-risk environments like mining and welding as well as to raise public awareness about dietary practices that may help to reduce the effect of manganese on neural development.

MPTP

The most widely studied chemical that can cause parkinsonism is 1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine (MPTP). When MPTP is administered to mice, it causes oxidative stress and mitochondrial apoptosis, and this ultimately leads to dopaminergic cell death. Oxidative stress involves cells not getting enough oxygen. Prolonged exposure to this toxin leads to mitochondrial swelling and release of cytochrome C and other intermembrane space, leading to

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activation of caspase-mediated apoptosis. Administration of pargyline, a monoamine oxidase inhibitor (MAO), and/or diphenyl can help reduce symptoms of MPTP exposure.

Additionally, the identification of the complex chemical pathways underlying neurodegeneration has been made possible by the discovery of 1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine (MPTP) and its impact on Parkinsonism. When mice are given MPTP, a series of events take place that illustrate how oxidative stress and mitochondrial malfunction contribute to the death of dopaminergic cells. Oxidative stress, a circumstance when cells don't get enough oxygen, becomes a key factor, starting a number of harmful processes. Mitochondrial apoptosis, which is defined by disturbances in the delicate balance of cellular energy production, is brought on by MPTP-induced oxidative stress. Dopaminergic neurons, important participants in the regulation of movement and reward, eventually die as a result of this disturbance.

The increasing mitochondrial failure seen following long-term exposure to MPTP is a crucial component of this sequence. Notably, the toxin causes enlargement in the mitochondria and causes the discharge of substances from the intermembrane gap, including cytochrome C. This release sets off a complicated chain of events, which includes the activation of the route for programmed cell death known as caspase-mediated apoptosis. These interconnected processes eventually lead to a noticeable loss of dopaminergic neurons, mirroring the distinctive neurodegeneration found in Parkinson's disease.

The exploration of therapeutic approaches has resulted from efforts to combat the incapacitating consequences of MPTP exposure. Notably, the use of pargyline, an MAO inhibitor, and/or diphenyl has demonstrated promise in reducing the symptoms brought on by MPTP exposure. These treatments focus on particular elements of the MPTP-induced cascade with the goal of reducing oxidative stress and mitochondrial dysfunction while maintaining the health of dopaminergic neurons. These discoveries may aid in the creation of new treatment plans for Parkinson's disease and other neurodegenerative diseases as research into the complex mechanisms of MPTP-related Parkinsonism advances.

Agent Orange

Although there is limited evidence to suggest that Agent Orange can be a direct cause of Parkinson's disease, exposure to it leads to similar symptoms. Studies have shown that exposure to Agent Orange leads to symptoms like tremors, loss of balance, and muscle rigidity.This means that Agent Orange can possibly be associated with causing Parkinson’s disease because of the similar symptoms.

The shared symptomatology between exposure to this herbicide and the manifestation of Parkinson's-like symptoms is a tempting area for additional research, even if the connection

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between Agent Orange and Parkinson's disease is still a subject of current inquiry. The possibility of environmental poisons having an adverse effect on neurological health is highlighted by the occurrence of tremors, loss of balance, and rigidity in people who were exposed to Agent Orange. These signs and symptoms resemble those of Parkinson's disease, which is characterized by motor abnormalities; hence, it is unclear if exposure to Agent Orange may have influenced the underlying processes that result in the malfunctioning of dopaminergic neurons and subsequent neurodegeneration. A fuller comprehension of the probable link between Agent Orange and Parkinson's disease could have important implications as research into the complex interaction between environmental exposures and neurodegenerative illnesses continues.

Conclusion

Parkinson’s disease is one of the most debilitating diseases out there. A combination of environment and genetic factors cause this crippling disease. There are so many toxins out there in our world that the likelihoods of developing Parkinson’s disease for people are just going to go up. It is important to inform others of these findings so that they may be more careful around these toxins. The urgency of further research and increased public awareness underscores the importance of finding solutions to a debilitating disease. Finding the right atmosphere can be beneficial to all and help decrease the risk of developing Parkinson’s disease.

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