



Do Pesticides Cause Parkinson's Disease?

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We use many chemical substances to protect our food crops from attacks by insect pests—in Marin County, more than 54,000 pounds of pesticides were used in 2000, according to the Department of Pesticide Regulation. But what do those chemicals do to us? Recent studies have detailed the effects pesticides might have on our bodies, and in particular, researchers at the Novato-based Buck Institute have shown a possible relationship between certain pesticides and Parkinson's disease.

In the United States alone, at least 500,000 people suffer from Parkinson's disease. As one of the most common neurodegenerative diseases, it haunts 1 percent of people over 65 years of age.

Parkinson's disease has been documented by different cultures for over 4000 years, and yet the cause of the disease is still unknown. What is known is that it slowly destroys essential brain cells that produce dopamine, a chemical in the brain that is important in the functioning of muscles. The disorder progresses slowly, beginning with trembling of the limbs and eventually causing difficulty and slowness in moving, stiff limbs, and problems with balance. Another feature crucial to the diagnosis of Parkinson's disease is protein deposits in dying brain cells that are called Lewy bodies.

"We can relieve the symptoms, but we cannot treat the disease," said Dr. Donato Di Monte, director of basic research at the Parkinson's Institute in Sunnyvale, California. "Over time, the symptoms become more and more severe and the therapy becomes less and less effective," he said.

Recently, studies have been published linking environmental factors to Parkinson's disease. In the past few years, interest has focused on people living in rural communities that had been exposed to pesticides seemed to be at greater risk for developing Parkinson's. At Emory University in Georgia, Dr. Tim Greenamyre found that rotenone, an organic insecticide used in fisheries, created the main symptoms of Parkinson's disease, also known as Parkinsonism, in rats.

At the Buck Institute in Novato, Dr. Julie Andersen is currently researching the effects of paraquat on brains of mice and its relation to Parkinson's disease. The herbicide is used on corn, soybeans, cotton and fruit.

"Paraquat converts oxygen to damaging oxidative compounds called reactive oxygen species or ROS, such as superoxide and hydrogen peroxide. These compounds can interact with cells and produce damage," said Andersen. "Epidemiological evidence, including that of our colleagues Dr. William Langston and Dr. Carol Tanner at the Parkinson's Institute, has suggested that exposure to herbicides such as paraquat is a risk factor for the disease," she said.

Research by Di Monte has also shown that exposure to paraquat kills the neurons in the brain that direct dopamine, as well as creating Lewy bodies. Now Dr. Andersen and scientists at the Parkinson's Institute are teaming up to uncover what other factors are involved with paraquat and its dangerous effects on the brain.

"We are looking at the combined effects of both paraquat and iron in Lewy body formation and accompanying dopaminergic cell death as a model of Parkinson's disease," said Andersen. Higher levels of iron are present in the brains of Parkinson's patients. Iron contributes to the increased production of ROS compounds, and it has also been shown to increase the presence of alpha synuclein, a protein found in Lewy bodies.

Because iron uptake usually happens in the early stages of life, the scientists are looking at whether exposure to iron during infancy can cause Parkinson's later in life. Likewise, paraquat also changes oxygen into dangerous oxidative compounds, and as Di Monte's research shows, it may also assist in the development of Lewy bodies.

Andersen at the Buck Institute recently joined scientists at the Parkinson's Institute to investigate the role of pesticides in combination with other mechanisms. Andersen previously worked mostly with the MPTP model to study Parkinson's. MPTP was used in the 1980s as a substitute for heroin, and the synthetic drug's users developed symptoms of Parkinsonism. MPTP has also been found to be chemically similar to some pesticides, like paraquat and rotenone. Although MPTP reproduces all the symptoms of Parkinson's disease and is the closest model researchers have so far, it does not mimic the slow and progressive nature of the disease.

Preliminary studies using paraquat suggest that the model may be very useful, but more work must be done. "I want to emphasize that because we can reproduce pathological features of Parkinson's disease in animal models with these pesticides, it is not conclusive evidence that pesticides are involved in Parkinson's disease," said Di Monte. The problem is more complex because of the variety of environmental agents to which people are exposed and their interactions.

Genetic abnormalities in combination with environmental toxins—not necessarily pesticides—could potentially cause Parkinson's disease. By understanding the mechanisms that affect the brain, treatments could be created to counteract the degeneration of the cells.

"With the new acquisition of the new information of the past couple of years and the new technology that is available now to pursue some of these studies, I think we are as close as we have ever been to making a major breakthrough in this disease," said Di Monte.

RESOURCES

Buck Institute: www.buckinstitute.org

Parkinson's Institute: www.parkinsonsinstitute.org

National Institutes of Health: www.nih.gov