PARKINSON DISEASE

Pesticides implicated as an environmental factor in PD

A case–control study of 50 patients with Parkinson disease (PD) suggests that exposure to the pesticide β-hexachlorocyclohexane (β-HCH) could be a risk factor for PD. "Most studies associating pesticide exposure with PD are based on patient questionnaires; this is one of the few to measure serum levels of organochlorine pesticides," points out lead author Jason Richardson of the Environmental and Occupational Health Sciences Institute, Piscataway, NJ, USA.

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Organochlorine pesticides are known to be neurotoxic, and research in rodent models has shown that they can have devastating effects on the dopaminergic system in the brain. These compounds, used widely from the 1950s until the 1970s, still persist in the environment. Four previous small studies that measured pesticide levels in postmortem brain samples hinted at a link between organochlorines and PD, prompting Richardson and colleagues to investigate further.

The link suggested by this research has been corroborated by a similar study carried out in the Faroe Islands, currently in press. "This population has very high exposure to multiple persistent pollutants based on their diet, and the levels of β-HCH in the controls were equivalent to those present in our PD patients," reports Richardson. Although a purely genetic component exists in PD, the vast majority of cases are idiopathic, and Richardson believes that the growing evidence for the involvement of pesticides in PD could help focus research towards looking at gene–environment interactions. "If there was a single or very potent environmental factor contributing to PD, researchers would have found it by now; PD is more likely to result from a combination of genetic and environmental factors," he says.

Further research could reveal whether detection of elevated β-HCH levels could help identify those at risk of developing PD. Earlier diagnosis is highly desirable, since current methods tend to diagnose PD after significant neuron loss has already occurred, which could explain why many of the clinical trials of neuroprotective agents have been unsuccessful. The group also wants to explore the effects of genetic polymorphisms in enzymes that metabolize the pesticides. "In this study, we only measured β-HCH in blood and do not know whether this mirrors the chemical compounds observed in the brain. It is possible that the β-HCH is a 'marker' for exposure to other pesticides that actually cause the damage," says Richardson.

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