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Nanoplastics may help set the stage for Parkinson's risk

At a Glance

- Scientists found that tiny plastic particles can enter nerve cells, impair breakdown of structures linked to Parkinson's disease, and harm certain brain regions in mice.
- The findings point to molecular links between plastics and Parkinson's disease mechanisms that can be further explored through additional research.

The study findings give insight into how polystyrene waste may help contribute to Parkinson's disease. *Rokas Tenys / Shutterstock*

Parkinson's disease and related dementias have been on the rise worldwide. These disorders are marked by an abnormal buildup of the protein alpha-synuclein in the brain. The factors leading to this buildup of alpha-synuclein are unknown. Research points to a potential role for environmental factors.

Small bits of plastic are widely found throughout the environment, including food and water supplies. Microplastics are plastic particles smaller than 5 mm in diameter—tinier than a sesame seed; nanoplastics are less than 1 µm, too small to be seen by the human eye. At least one previous study found that particles of polystyrene and other plastics can be detected in the blood of most healthy adults. Single-use polystyrene products—like plastic cups, utensils, and foam packing—are widespread environmental waste. But despite their ubiquity, the potential health consequences of these plastics are only beginning to be studied and understood.

Previous studies found evidence that alpha-synuclein's activities can be affected by polystyrene and other particles. An international research team led by Dr. Andrew B. West of Duke University decided to take a closer look at the effects that nanoplastics might have on nerve cells and the brain. The scientists explored interactions between alpha-synuclein and polystyrene nanoplastics both in lab dishes and in mice. Results were reported on November 17, 2023, in *Science Advances.*

The researchers first showed that human alpha-synuclein binds readily to polystyrene nanoplastics in a test tube. This binding led to the formation of abnormal alpha-synuclein structures called fibrils, a hallmark of Parkinson's disease and related dementias.

The scientists next examined how alpha-synuclein fibrils and nanoplastics behave with cultured brain cells, or neurons. They found that both the fibrils and the plastics can enter neurons via endocytosis, in which the cell's outer membrane engulfs targeted items. Once inside, both the fibrils and the plastics entered the cell's lysosomes, membrane-bound organelles that serve as cellular garbage disposals. The researchers found that nanoplastics disrupted lysosome activities, slowing the breakdown of harmful clumps of alpha-synuclein.

The team next looked at how polystyrene nanoplastics and alpha-synuclein interact in the mouse brain. They found that the nanoplastics and alpha-synuclein fibrils also interacted there, which increased the spread of abnormalities across interconnected brain regions. Neurons in the brain's substantia nigra region were especially affected. This brain region helps to control movement and is damaged in Parkinson's disease and related dementias.

Taken together, these findings point to previously unrecognized interactions that could contribute to Parkinson's disease risk and progression. Further research is needed to study how these interactions affect disease development and whether other types of plastics have similar effects.

"Numerous lines of data suggest environmental factors might play a prominent role in Parkinson's disease, but such factors have for the most part not been identified," West explains. "Our study suggests that the emergence of micro and nanoplastics in the environment might represent a new toxin challenge with respect to Parkinson's disease risk and progression."

—by Vicki Contie

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References: Anionic nanoplastic contaminants promote Parkinson's disease-associated α-synuclein aggregation. Liu Z, Sokratian A, Duda AM, Xu E, Stanhope C, Fu A, Strader S, Li H, Yuan Y, Bobay BG, Sipe J, Bai K, Lundgaard I, Liu N, Hernandez B, Bowes Rickman C, Miller SE, West AB. *Sci Adv.* 2023 Nov 15;9(46):eadi8716. doi: 10.1126/sciadv.adi8716. Epub 2023 Nov 17. PMID: 37976362.

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