

How Parkinson's disease starts and spreads

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Injection of a small amount of clumped protein triggers a cascade of events leading to a Parkinson's-like disease in mice, according to an article published online this week in the *Journal of Experimental Medicine*.

Progressive accumulation of clumps of the protein alpha-synuclein in the brains of patients with Parkinson's disease coincides with the onset of [motor dysfunction](#). However, whether these clumps are sufficient to trigger neurodegeneration, and how these clumps spread throughout the brain, remained unclear.

To answer these questions, a team led by Virginia M.Y. Lee at the University of Pennsylvania School of Medicine studied mice expressing a mutated form of alpha-synuclein found in patients with Parkinson's disease. These mice show symptoms of disease around one year of age but not earlier.

Lee and colleagues found that injecting preformed clumps of human alpha-synuclein into the brains of young mice accelerated disease onset and severity. These clumps seemed to act as "seeds" that recruited even the mouse version of alpha-synuclein into new clumps, which then spread throughout the brain. The pattern of spreading from neuron to neuron suggests that the clumps may hijack the highway traveled by normal brain signals.

These findings suggest that Parkinson's disease, like other [neurodegenerative diseases](#) including Alzheimer's, may start and

progress due to abnormal aggregation and accumulation of proteins within the brain. What gets these clumps going in the first place remains unclear.

More information: Hung, L.W., et al. 2012. *J. Exp. Med.*
[doi:10.1084/jem.20112285](https://doi.org/10.1084/jem.20112285)

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