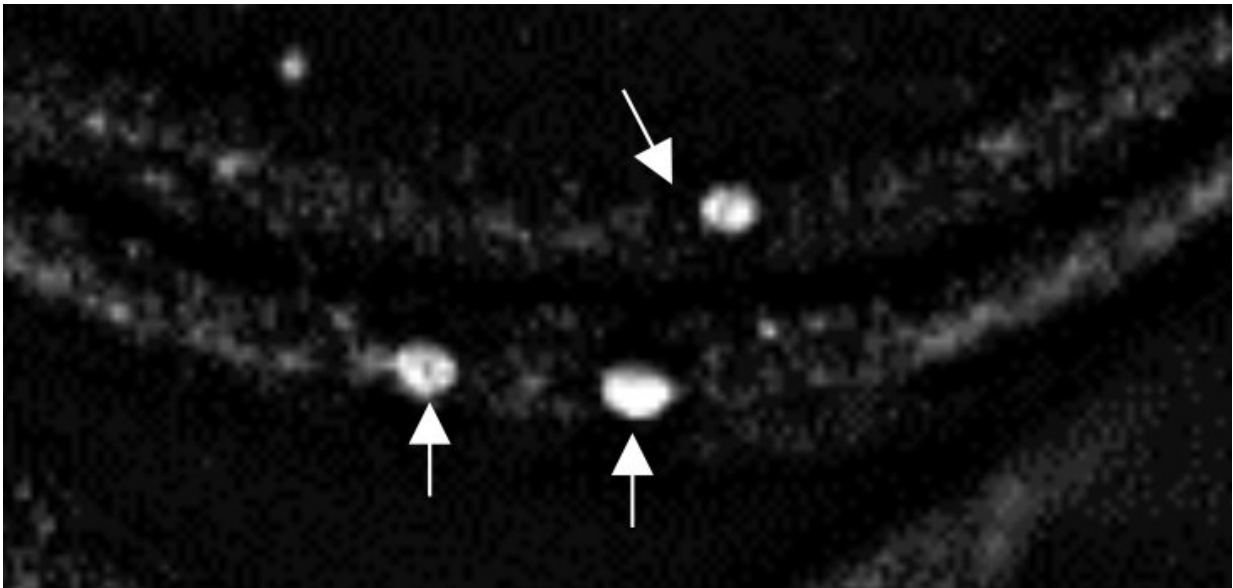


Study unravels new insights into a Parkinson's disease protein

January 16 2020, by Charlotte Hsu



An epifluorescence microscope image shows clumps of human alpha-synuclein aggregating in the neurons of a fruit fly larva. Credit: Anderson, Hirpa, Zheng, Banerjee and Gunawardena, *Frontiers in Cellular Neuroscience*, Jan. 2020. The image is cropped from a graphic published in the journal article, which is distributed under Creative Commons Attribution License CC BY 4.0 (creativecommons.org/licenses/by/4.0/)

Research by University at Buffalo biologists is providing new insights into alpha-synuclein, a small acidic protein associated with Parkinson's disease.

Alpha-synuclein is known to form abnormal clumps in the brains of patients with Parkinson's, but scientists are still trying to understand how and why this happens.

The new study explores [alpha-synuclein](#)'s basic properties, with a focus on a section of the protein known as the non-amyloidal component (NAC). The research was done on [fruit fly larvae](#) that were genetically engineered to produce both normal and mutated forms of human alpha-synuclein.

The study, led by University at Buffalo biologist Shermali Gunawardena, was published on Jan. 10 in the journal *Frontiers in Cellular Neuroscience*.

Some key findings:

- The NAC region appears to aid alpha-synuclein in moving through pathways called axons that run from one area of a neuron to another. When the NAC region was missing, alpha-synuclein did not move within axons.
- Alpha-synuclein that's missing the NAC region may help to prevent unwanted aggregates of the protein. In experiments, Gunawardena's team showed that it's possible—at least in [fruit flies](#)—to prevent some key problems that occur when too much alpha-synuclein is produced: clumping of the protein; abnormalities in the structure of synapses, which form connections between neurons; and a decrease in the speed at which [larvae](#) crawl. The scientists found that when the larvae are engineered to produce both excess alpha-synuclein and a version of alpha-synuclein with the NAC region missing, the larvae crawl normally, the [protein](#) doesn't aggregate, and the synapses are normal.

"We show that in fruit fly larvae, we're able to prevent some problems mimicking symptoms of Parkinson's [disease](#), such as accumulation of alpha-synuclein in neurons," says Gunawardena, Ph.D., associate professor of biological sciences in the UB College of Arts and Sciences.

"Our work highlights a potential early treatment strategy for Parkinson's disease that would leverage the use of deletion of the NAC [region](#)," Gunawardena adds. "One reason this study is important is because it shows rescue of alpha-synuclein aggregates, synaptic morphological defects and locomotion defects seen in Parkinson's disease in the context of a whole organism."

More information: Eric N. Anderson et al, The Non-amyloid Component Region of α -Synuclein Is Important for α -Synuclein Transport Within Axons, *Frontiers in Cellular Neuroscience* (2020). [DOI: 10.3389/fncel.2019.00540](https://doi.org/10.3389/fncel.2019.00540)

Provided by University at Buffalo

Citation: Study unravels new insights into a Parkinson's disease protein (2020, January 16) retrieved 25 April 2024 from

<https://medicalxpress.com/news/2020-01-unravels-insights-parkinson-disease-protein.html>

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