

Researchers increase understanding of gene's potentially protective role in Parkinson's

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Treatments for Parkinson's disease, estimated to affect 1 million Americans, have yet to prove effective in slowing the progression of the debilitating disease.

However, University of Alabama researchers have identified how a specific gene protects dopamine-producing neurons from dying in both animal models and in cultures of human neurons, according to a scientific article publishing in the Feb. 8 edition of *The Journal of Neuroscience*.

This increased understanding of the gene's neuro-protective capability is, the researchers said, another step toward the potential development of a new drug treatment.

"This gene represents a previously unexplored protein [therapeutic target](#) for Parkinson's disease," said Dr. Guy Caldwell, professor of [biological sciences](#) at The University of Alabama and a co-author of the article.

The gene, known as VPS41, was one of five [genes](#) that UA scientists showed in 2008 had protective capabilities against a hallmark trait of Parkinson's, the age-associated loss of [dopamine neurons](#).

The latest announcement reflects the better understanding since gained of how the gene functions.

The latest UA research was primarily funded by the Michael J. Fox

Foundation for Parkinson's Research. The scientific journal, published by the Society of Neuroscience, is the largest weekly journal dedicated to neuroscience discovery.

The researchers also found that specific, and rare changes in [human DNA](#) – changes sometimes also evident in non-Parkinson's patients – appear to impact how VPS41 functions.

"Mutations like these may represent previously unreported susceptibility factors for Parkinson's disease," Caldwell said.

The article's lead author is Dr. Adam Harrington, who earned his doctoral degree from UA in December 2011 while working in the Caldwell Lab. The additional UA co-author is Dr. Kim Caldwell, associate professor of biological sciences. Dr. Talene Yacoubian, a physician, and Sunny Slone, both of the University of Alabama at Birmingham, are also co-authors.

The researchers used both specific strains of tiny nematode worms as animal models for the work along with the human cultures. The genetically engineered worms contain a human protein, alpha-synuclein within their cells. Scientists have learned that people with too many copies of the code for alpha-synuclein within their DNA will contract Parkinson's.

Extra copies of alpha-synuclein can lead to repeated protein misfolding and the death of the dopamine-producing neurons in the brain. In Parkinson's patients, the death of these neurons leads to rigid and tremoring limbs, difficulty in movement and impaired reflexes.

"The main advance here is that we have mechanistically defined how VPS41 appears to convey its protective capacity to neurons – not only in worms, but also in human dopamine-producing neuron cultures," said

Caldwell.

The next phase in this research involves translating these findings into potential therapies.

"The obstacles of finding any disease-modifying therapy are diminished once protective mechanisms, like this one, become revealed and better defined," said Caldwell.

Provided by University of Alabama in Tuscaloosa

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