

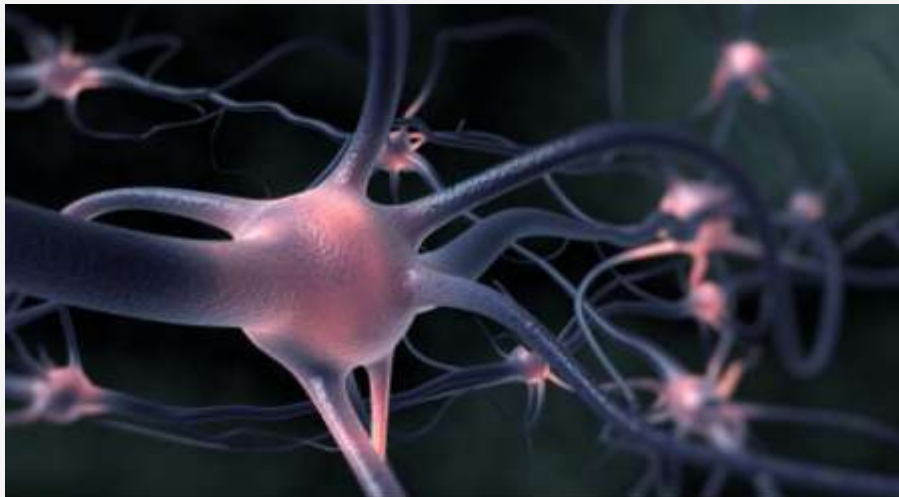
Researchers identify biological process that appears to trigger Parkinson's disease

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New research has identified a biological 'trigger' for Parkinson's disease. The findings, published in the journal *Cell*, may lead the way to early diagnostic tools and treatments that could shut down the disease before symptoms progress in patients.

Using both human neurons and fruit flies, researchers at Johns Hopkins have identified a protein, α 5, that triggers a common form of Parkinson's disease. The protein is enabled by an enzyme - leucine-rich repeat kinase 2 (LRRK2) - which then causes neurodegeneration.

Previous research has shown that mutations in LRRK2 are linked with neurodegeneration, and therefore, the progression of Parkinson's disease. LRRK2 had been previously identified to be a protein kinase, which means it is a type of enzyme that adds phosphates on to other proteins and either turns proteins on or off or changes the protein's activity. However, the proteins LRRK2 was acting on were unknown—until now.

"How mutations in [LRRK2] cause Parkinson's disease aren't well known, and what this study does is provides a pretty convincing set of data on how mutations in LRRK2 cause Parkinson's disease," study author Dr. Ted Dawson, professor of neurology and director of the Johns Hopkins Institute for Cell Engineering, told FoxNews.com.

The study's findings suggest that inhibiting s15 and LRRK2 could prevent the loss of dopamine neurons and the onset of Parkinson's disease. LRRK2 inhibitors exist, but have not been tested in patient trials. Studies are needed to identify inhibitors of s15, Dawson said.

Parkinson's disease is a neurodegenerative disorder that affects nearly one million people in the U.S., according to the Parkinson's Disease Foundation (PDF). With the disease, the brain loses dopamine-producing cells— dopamine sends messages to the brain control movement and coordination.

Symptoms vary from person to person, and are progressive, meaning they continue to worsen over time. According to the PDF, they can include tremor of the hands, legs and face, slowness of movement, stiffness of the limbs, and impaired balance and coordination.

Currently, the drug levodopa is used to treat the symptoms of Parkinson's. While effective, it doesn't treat all the symptoms, has side effects and doesn't prevent degeneration. Researchers hope their study of LRRK2 could lead to an earlier diagnosis and better treatments for Parkinson's.

There is a genetic test for Parkinson's, but it's only being used for research purposes because there aren't therapies for patients yet, Dawson said.

"The general idea now is that... the degenerative process is starting many, many years before people start to manifest with symptoms," Dawson said. "Once we do have [medications] that slow the progression, there are people who have [mutations in LRRK2] who you'd think would want to know and would want to take it."

Development of consumer-ready drugs could take years; Dawson estimated 3 to 5 years for LRRK2 trials with patients and another 10 years for s15 inhibitors.

"If you look at neurodegenerative diseases, they're a major and substantial health problem for the U.S. and it's only going to get worse, but the U.S. is not investing the required resources to really make substantial headway into treating not only Parkinson's but other neurodegenerative disorders," Dawson said.