

Team to study new gene associated with Parkinson's disease

15 June 2018, by Kelsey Schnieders Lefever

A multidisciplinary team of researchers at Purdue University and the University of Bordeaux in France has received a grant from The Michael J. Fox Foundation for Parkinson's Disease to study a new gene associated with Parkinson's disease, which was linked to the disease using novel big data methodologies.

The findings from this research could potentially be used to design new therapies to slow neurodegeneration in the brains of patients with Parkinson's disease and other related disorders.

Jean-Christophe Rochet, professor of medicinal chemistry and molecular pharmacology at Purdue; Erwan Bezdard, research director at the Institute of Neurodegenerative Diseases at the University of Bordeaux; Jason Cannon, associate professor of toxicology at Purdue; and Min Zhang, professor of statistics at Purdue, will study the neuroprotective effects of the new gene, known as NFE2L1, in Parkinson's [disease models](#). Their collaboration was helped by a nearly \$107,000 grant from the organization named for the famous actor who has Parkinson's.

Zhang and Rochet began discussing a potential collaboration 10 years ago because of their common interest in Parkinson's disease. While analyzing several data sets obtained from one of the National Institutes of Health-Designated Data Repositories, they identified a list of genes associated with the disease.

"While some of the genes on the list were already known, Chris found an interesting gene that has not been reported to be directly associated with Parkinson's disease yet," Zhang said.

NFE2L1 is a protein that controls the expression of [genes](#) involved in the differentiation and survival of dopamine neurons.

"NFE2L1 levels are reduced in [dopamine neurons](#)

in the brains of Parkinson's disease patients," Rochet said. "We recently found in a large-scale genomic study that a minor allele of NFE2L1 can lower Parkinson's risk. These observations imply that neuron death in Parkinson's disease may result in part from a loss of the neuroprotective action of NFE2L1."

The team hypothesizes that an increase of NFE2L1 can alleviate [neuron death](#) in rodent models of Parkinson's disease. The results of the study will shed light on the ability of NFE2L1 to reduce neurotoxicity throughout the brain.

Zhang and Rochet hope that the results of the study will set the stage for developing Parkinson's [disease](#) therapies aimed at increasing NFE2L1 levels in the brain. The next step toward clinical application of the results of this study will be to screen for compounds that increase NFE2L1 levels in the brain, either by stimulating the protein's expression or blocking the protein's destruction by the proteasome.

Provided by Purdue University

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