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New Parkinson's Drug Could Slow Disease Progression

A family of compounds has recently been developed that may slow the progression of Parkinson's disease.

Parkinson's affects both men and women and usually develops after the age of 50. It is one of the most prevalent neurodegenerative diseases, which results from the death of dopamine neurons, eventually leading to rigidity, tremors, and difficulty moving.

There are existing medications that can alleviate the symptoms, but unfortunately they do not have an impact on the development of the disease.

The new compounds were developed by Richard B. Silverman, the John Evans Professor of Chemistry at the Weinberg College of Arts and Sciences and inventor of the molecule that became the well-known drug Lyrica, and D. James Surmeier, chair of physiology at Northwestern University Feinberg School of Medicine. The findings were published in the journal *Nature Communications*.

The compounds shut out a fairly rare membrane protein, so that calcium cannot enter into dopamine neurons. A different study by Surmeier indicated that premature aging and death could result from calcium entry through this protein, which strains dopamine neurons. **The exact protein involved, the Vav1.3 channel, was also recognized through his research**.

Surmeier explained:

> "These are the first compounds to selectively target this channel. By shutting down the channel, we should be able to slow the progression of the disease or significantly reduce the risk that anyone would get Parkinson's disease if they take this drug early enough."

"We've developed a molecule that could be an entirely new mechanism for arresting Parkinson's disease, rather than just treating the symptoms," Silverman added.

The way the compounds work is comparable to the way the drug isradipine, which successfully passed a phase 2 national clinical trial led by Northwestern Medicine neurologist Tanya Simuni, M.D., works.

However, isradipine cannot be used in a high enough concentration to be beneficial enough for patients with Parkinson's disease because it interacts with other channels found in the walls of blood vessels.

Silverman needed to create new compounds that aim for the Cav1.3 channel, instead of those that are abundant in blood vessels. Sixty-thousand existing compounds were tested through high-throughput screening. However, none of them did what they were looking for.

"We didn't want to give up," Silverman explained. Some compounds he had developed in his lab were then tested for other neurodegenerative diseases. After he recognized one compound as a potential drug, a postdoctoral associate in his lab, Soosung Kang, refined the molecules until they were able to efficiently close only the Cav1.3 channel.

Graduate student Gary Cooper tested the drug, developed by Silverman and Kang in Surmeier's lab, in regions of a mouse brain that was comprised of dopamine neurons. The drug did exactly what was expected, with no evident side effects. **The stress on the cells was alleviated by the drug**, Surmeier pointed out.

Before the team can move to a Phase 1 clinical trial, the pharmacology of the compounds needs to be improved so that they are acceptable for humans, and then examined on animals.

"We have a long way to go before we are ready to give this drug, or a reasonable facsimile, to humans, but we are very encouraged," Surmeier concluded.

Written by Sarah Glynn
View drug information on Lyrica.