

## Science News

from research organizations

# Study reports repair of mitochondrial recycling defect linked to Parkinson's disease

*Date:* August 3, 2021

*Source:* eLife

*Summary:* A study in mice shows that an experimental small molecule helped restore the removal of damaged mitochondria from dopamine-producing brain cells.

*Share:* [!\[\]\(17413706fd4997a1a4bdf85c6864eee1\_img.jpg\)](#) [!\[\]\(f419710cbe076aa30a9c6c031b5cbe84\_img.jpg\)](#) [!\[\]\(2726020a4107bdc9042b257034f90eb3\_img.jpg\)](#) [!\[\]\(9459655bf14a84f4d775e8d814cca8c9\_img.jpg\)](#) [!\[\]\(de47dbdca34225b222a4a87ac0e499b3\_img.jpg\)](#)

### FULL STORY

Treating mice that have a Parkinson's disease-causing mutation with a small molecule compound restores the removal of damaged mitochondria from their brain cells, shows a study published today in *eLife*.

The findings may help explain what goes wrong in dopamine-producing brain cells in people with mutations that cause Parkinson's disease.

Parkinson's disease is caused by the progressive loss of brain cells that produce dopamine. This causes the hallmark symptoms of the disease, including tremors, rigid movements, sleep problems and dementia.

"Scientists believe the death of these cells in people with Parkinson's disease is caused, in part, by the failure of a quality control mechanism that removes damaged energy-producing structures in the cells called mitochondria," explains first author Francois Singh, Postdoctoral Research Assistant at the Medical Research Council Protein Phosphorylation and Ubiquitylation Unit (MRC PPU), University of Dundee, Scotland, UK. "This failure to recycle damaged mitochondria is detrimental to the health of brain cells."

To learn more, Singh and colleagues teamed up with scientists from the Division of Signal Transduction Therapy, a consortium of academia and pharmaceutical companies. Together they used cutting-edge techniques to observe mitochondria recycling in the brains of mice that have the most common Parkinson's disease-causing mutation in a gene called LRRK2.

Their experiments showed that damaged mitochondria are not efficiently removed in the animals' dopamine-producing brain cells, and that damaged components in other types of brain cells are recycled. This may explain why dopamine-producing brain cells are selectively lost in Parkinson's disease and why the symptoms are all linked to a lack of dopamine.

The mutation in the LRRK2 gene results in the production of a hyperactive version of the protein. Given this, the team treated the mice with a small molecule that inhibits the hyperactive protein and found that it restored mitochondria recycling in the animals' dopamine-producing brain cells.

The authors say these results are an exciting step forward in the quest to understand mechanisms responsible for this currently incurable disease. These results should help drive and focus research in this area.

"Not only have we discovered new biology, but we have also shown that an LRRK2 inhibitor can rescue a mitochondrial defect related to Parkinson's disease," concludes senior author Ian Ganley, MRC Investigator and Scientific Programme Leader at MRC PPU, University of Dundee. "These findings highlight the significant benefit of academic-industrial collaborations that will hopefully accelerate the development of new treatments for Parkinson's disease."

---

### Story Source:

Materials provided by **eLife**. *Note: Content may be edited for style and length.*

---

### Journal Reference:

1. Francois Singh, Alan R Prescott, Philippa Rosewell, Graeme Ball, Alastair D Reith, Ian G Ganley. **Pharmacological rescue of impaired mitophagy in Parkinson's disease-related LRRK2 G2019S knock-in mice.** *eLife*, 2021; 10 DOI: 10.7554/eLife.67604

---

### Cite This Page:

MLA

APA

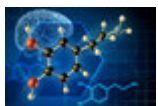
Chicago

---

eLife. "Study reports repair of mitochondrial recycling defect linked to Parkinson's disease." ScienceDaily. ScienceDaily, 3 August 2021. <[www.sciencedaily.com/releases/2021/08/210803142725.htm](http://www.sciencedaily.com/releases/2021/08/210803142725.htm)>.

---

### RELATED STORIES



#### 'Feel Good' Brain Messenger Can Be Willfully Controlled, New Study Reveals

July 23, 2021 — Researchers have discovered that spontaneous impulses of dopamine, the neurological messenger known as the brain's 'feel good' chemical, occur in the brain of mice. The study found that mice can ...

#### Becoming a Nerve Cell: Timing Is of the Essence

Aug. 13, 2020 — Researchers find that mitochondria regulate a key event during brain development: how neural stem cells become nerve cells. Mitochondria influence this cell fate switch during a precise period that ...

#### Study Shows Promise in Repairing Damaged Myelin

Apr. 18, 2019 — A new study shows that a synthetic molecule stimulates repair of the protective sheath that covers nerve cells in the brain and spinal cord. The study demonstrates in mice that a synthetic molecule ...

#### New Treatment Offers Potentially Promising Results for the Possibility of Slowing, Stopping, or Even Reversing Parkinson's Disease

Feb. 27, 2019 — A pioneering clinical trials program that delivered an experimental treatment directly to the brain offers hope that it may be possible to restore the cells damaged in Parkinson's disease. The study ...

## FROM AROUND THE WEB

---

*ScienceDaily shares links with sites in the TrendMD network and earns revenue from third-party advertisers, where indicated.*

## Free Subscriptions

---

Get the latest science news with ScienceDaily's free email newsletters, updated daily and weekly. Or view hourly updated newsfeeds in your RSS reader:

 [Email Newsletters](#)

 [RSS Feeds](#)

## Follow Us

---

Keep up to date with the latest news from ScienceDaily via social networks:

 [Facebook](#)

 [Twitter](#)

 [LinkedIn](#)

## Have Feedback?

---

Tell us what you think of ScienceDaily -- we welcome both positive and negative comments. Have any problems using the site? Questions?

 [Leave Feedback](#)

 [Contact Us](#)

[About This Site](#) | [Staff](#) | [Reviews](#) | [Contribute](#) | [Advertise](#) | [Privacy Policy](#) | [Editorial Policy](#) | [Terms of Use](#)

Copyright 2021 ScienceDaily or by other parties, where indicated. All rights controlled by their respective owners.

Content on this website is for information only. It is not intended to provide medical or other professional advice.

Views expressed here do not necessarily reflect those of ScienceDaily, its staff, its contributors, or its partners.

Financial support for ScienceDaily comes from advertisements and referral programs, where indicated.

— [CCPA: Do Not Sell My Information](#) — — [GDPR: Privacy Settings](#) —