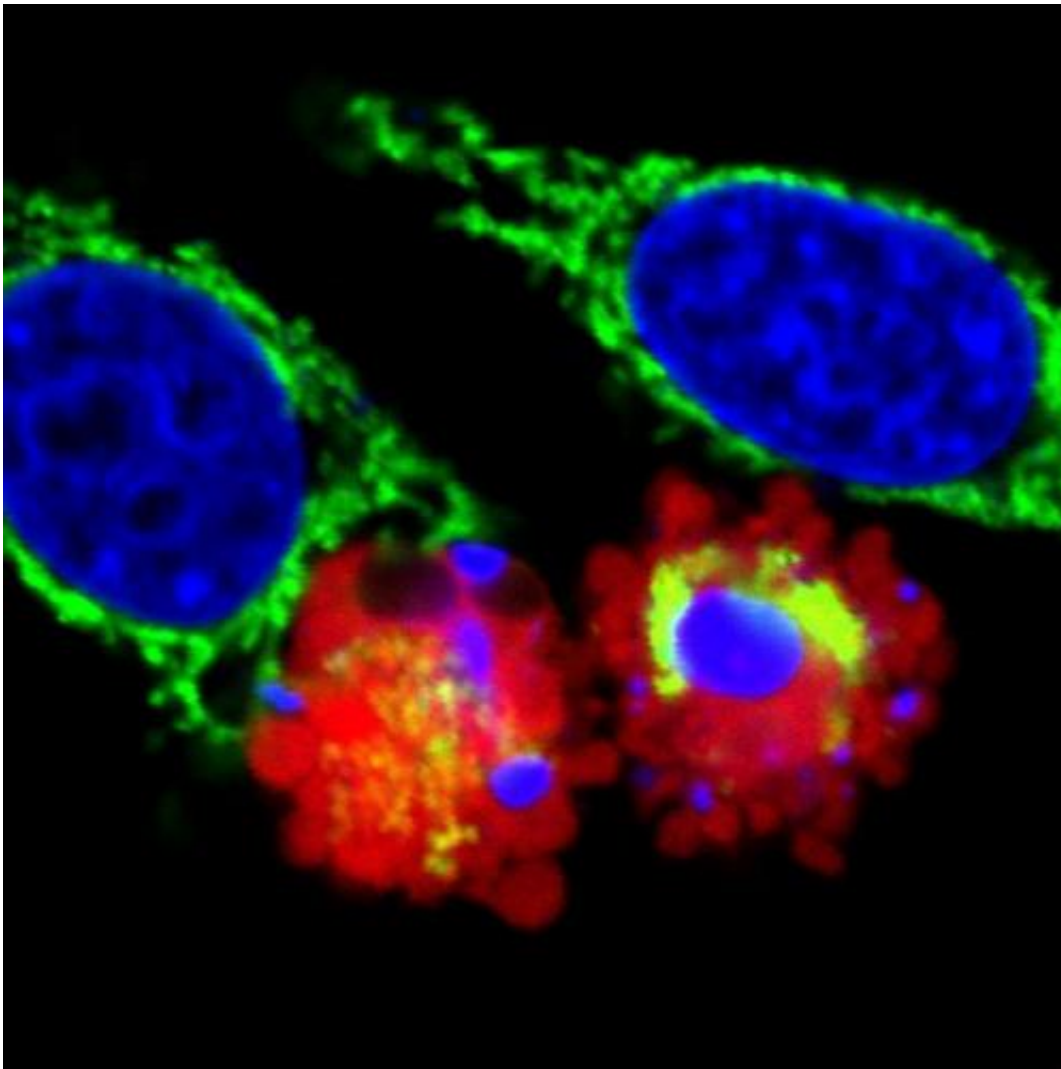


Scientists make breakthrough in understanding Parkinson's disease

November 13 2014



Parkin-expressing cells (red) are undergoing programmed cell death. Credit: Dr Emilie Hollville and Professor Seamus Martin, Trinity College Dublin

Scientists at Trinity College Dublin have made an important breakthrough in our understanding of Parkin - a protein that regulates the repair and replacement of nerve cells within the brain. This breakthrough generates a new perspective on how nerve cells die in Parkinson's disease. The Trinity research group, led by Smurfit Professor of Medical Genetics, Professor Seamus Martin, has just published its findings in the internationally renowned, peer-reviewed Cell Press journal, *Cell Reports*.

Although mutation of Parkin has been known to lead to an early onset form of Parkinson's for many years, understanding what it actually did within cells has been difficult to solve. Now, Professor Martin and colleagues have discovered that in response to specific types of cell damage, Parkin can trigger the self-destruction of 'injured' [nerve cells](#) by switching on a controlled process of 'cellular suicide' called apoptosis.

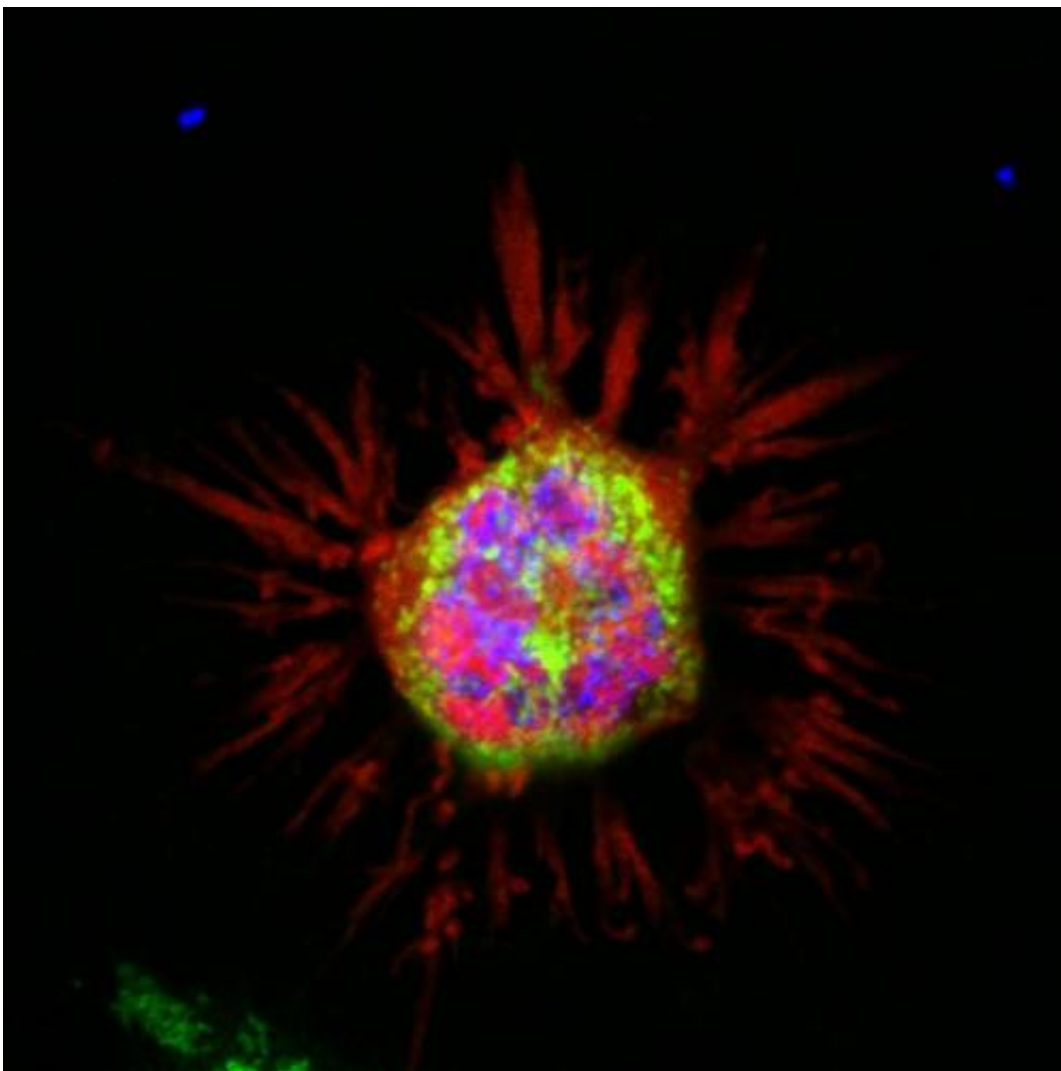
Using cutting-edge research techniques, the Martin laboratory, funded by Science Foundation Ireland, found that damage to mitochondria (which function as 'cellular battery packs') activates the Parkin protein, which results in one of two different outcomes - either self-destruction or a repair mode. Which outcome was chosen depended on the degree of damage suffered by the cellular battery packs.

Importantly, these new findings suggest that one of the problems in Parkinson's disease may be the failure to clear away sick nerve cells with faulty cellular battery packs, to make way for healthy replacements. Instead, sickly and dysfunctional nerve cells may accumulate, which effectively prevents the recruitment of fresh replacements.

Commenting on the findings, Professor Martin stated: "This discovery is surprising and turns on its head the way we thought that Parkin functions. Until now, we have thought of Parkin as a brake on cell death within nerve cells, helping to delay their death. However, our new data

suggests the contrary: Parkin may in fact help to weed out injured and sick nerve cells, which probably facilitates their replacement. This suggests that Parkinson's disease could result from the accumulation of defective neurons due to the failure of this cellular weeding process."

Professor Martin also added: "We are very grateful for the support of Science Foundation Ireland, who funded this research. This work represents an excellent example of how basic research leads to fundamental breakthroughs in our understanding of how diseases arise. Without such knowledge, it would be very difficult to develop new therapies."



Parkin-expressing cells (red) are undergoing programmed cell death. Credit: Dr Emilie Hollville and Professor Seamus Martin, Trinity College Dublin

More information: 'Parkin Sensitizes toward Apoptosis Induced by Mitochondrial Depolarization through Promoting Degradation of Mcl-1, Cell Reports, 2014.

Provided by Trinity College Dublin

Citation: Scientists make breakthrough in understanding Parkinson's disease (2014, November 13) retrieved 25 April 2024 from <https://medicalxpress.com/news/2014-11-scientists-breakthrough-parkinson-disease.html>

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