Blocking mitochondrial fission: An effective treatment for Parkinson's disease?

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A study led by a researcher from Plymouth University in the UK, has discovered that the inhibition of a particular mitochondrial fission protein could hold the key to potential treatment for Parkinson's Disease (PD).

The findings of the research are published today, 5th November 2014, in *Nature Communications*.

PD is a progressive neurological condition that affects movement. At present there is no cure and little understanding of why some people get the condition. In the UK one on 500 people, around 127,000, have PD.

The debilitating movement symptoms of the disease are primarily caused by the death of a type of brain cell that produces a chemical called dopamine. This brain chemical (also known as a neurotransmitter) helps nerve cells to send signals to other nerve cells. A reduction in dopamine from cell death results in a lack of communication between nerve cells, which in turn leads to difficulty in movement control. Understanding why these nerve cells die or do not work properly could lead to new therapies for PD.

Mitochondria are small structures within nerve cells that help keep the cells healthy and working properly – they are, in effect, the power generators of the cell. Mitochondria undergo frequent changes in shape, size, number and location either through mitochondrial fission (which leads to multiple, smaller mitochondria) or mitochondrial fusion (resulting in larger mitochondria). These processes are controlled mainly by their respective mitochondrial fission and fusion proteins. A balance of mitochondrial fission/fusion is critical to cell function and viability.

The research team found that when a particular mitochondrial fission protein (GTPase dynamin-related protein-1 – Drp1) was blocked using either gene-therapy or a chemical approach in experimental models of PD in mice, it reduced both cell death and the deficits in dopamine release – effectively reversing the PD process. The results suggest that finding a strategy to inhibit Drp1 could be a potential treatment for PD.

The research team is led by Dr. Kim Tieu from the Institute of Translational and Stratified Medicine, Plymouth University Peninsula Schools of Medicine and Dentistry. Dr. Tieu is a respected researcher in the field of PD. He initiated this research when he was a principal investigator at the University of Rochester School of Medicine and continued it on his move to Plymouth University in the UK.

He said: "Our findings show exciting potential for an effective treatment for PD and pave the way for future in-depth studies in this field. It's worth noting that other researchers are also targeting this mitochondrial fission/fusion pathway as potential treatments for other neurological diseases such as Alzheimer's disease, Huntington's disease and Amyotrophic Lateral Sclerosis."

Claire Bale, Research Communications Manager at Parkinson's UK, said: "We've known for decades that problems with mitochondria - the batteries of the cell - play a key role in the death of nerve cells in Parkinson's, but the research in this area hasn't yet led to new treatments.

"This study, which reveals a potential new drug target to protect mitochondria, is a promising step towards slowing down or stopping the progression of Parkinson's."

Provided by University of Plymouth