

Mitochondria may protect brain against Parkinson's

January 15 2018



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A Norwegian study shows that impairment in mitochondria may actually protect the brain in Parkinson's disease.

Mitochondria are microscopic power stations found inside our cells. They convert foodstuffs (nutrients) into fuel, providing our bodies with the energy they need.



In 1989, studies in <u>brain tissue</u> from individuals with Parkinson's disease showed that an essential component of the mitochondrial energy generators, called respiratory complex-I, becomes impaired in an area of the brain called the "substantia nigra" (latin for "the black substance"). Since this area is particularly vulnerable to Parkinson's disease, this observation led to the hypothesis that complex I deficiency is highly deleterious and contributes to neurodegeneration.

A new study from the University of Bergen (UiB), in Norway, in collaboration with the University of Cambridge, shows that the function of mitochondria, the microscopic powerhouses of the cell, is altered throughout the entire brain of individuals with Parkinson's disease. Ominous as this may sound, it might actually not be deleterious for patients.

"This new study shows that complex I deficiency is, in fact, a global phenomenon in the brain of persons with Parkinson's disease, and is found indiscriminately in both affected and healthy brain regions. Intriguingly, brain cells (neurons) with decreased complex I levels are significantly less likely to contain Lewy bodies, the abnormal proteinaggregates that characterize Parkinson's disease," says researcher Charalampos Tzoulis at Department of Clinical Science, UiB.

These discoveries suggest that, contrary to mainstream theory, mitochondrial complex I deficiency may not be entirely deleterious for the brain in Parkinson's disease.

"It is possible that complex I deficiency is part of a compensatory regulation attempting to protect the brain in Parkinson's disease, for instance via decreased production of oxidative free radical species. Further work will be necessary to understand why and how mitochondrial function is regulated in Parkinson's disease and whether this can be exploited for treatment," Charalampos Tsouliz exlplains.



Parkinson's disease facts:

- Parkinson's disease is one of the most common <u>brain</u> disorders. It affects approximately 130,000 people in the UK, 1 million people in the U.S and more than 10 million people worldwide.
- Parkinson's disease commonly starts after the age of 50 and causes a combination of debilitating symptoms, including shaking and other abnormal movements, loss of balance, <u>low</u> <u>blood pressure</u>, bladder and intestinal problems, sleeping disorders and dementia.
- In spite of intensive research in the field, there is no cure and patients die prematurely due to increasing disability. The research team from Bergen hope that their findings may bring us a step closer to developing a cure for Parkinson's <u>disease</u>.

More information: Irene H. Flønes et al. Neuronal complex I deficiency occurs throughout the Parkinson's disease brain, but is not associated with neurodegeneration or mitochondrial DNA damage, *Acta Neuropathologica* (2017). DOI: 10.1007/s00401-017-1794-7

Provided by University of Bergen

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