Your mother's genes can impact your own aging process, study finds

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As we age, our cells change and become damaged. Now, researchers at Karolinska Institutet and the Max Planck Institute for Biology of Aging have shown that aging is determined not only by the accumulation of changes during our lifetime but also by the genes we acquire from our mothers. The results of the study are published in the journal *Nature*.

There are many causes of aging that are determined by an accumulation of various kinds of changes that impair the function of bodily organs. Of particular importance in aging, however, seems to be the changes that occur in the cell's power plant – the mitochondrion. This structure is located in the cell and generates most of the cell's supply of ATP which is used as a source of chemical energy.

"The mitochondria contains their own DNA, which changes more than the DNA in the nucleus, and this has a significant impact on the aging process," said Nils-Göran Larsson, Ph.D., professor at the Karolinska Institutet and principal investigator at the Max Planck Institute for Biology of Aging, and leader of the current study alongside Lars Olson, Ph.D., professor in the Department of Neuroscience at the Karolinska Institutet. "Many mutations in the mitochondria gradually dis-able the cell's energy production," said Larsson.

For the first time, the researchers have shown that the aging process is influenced not only by the accumulation of mitochondrial DNA damage during a person's lifetime, but also by the inherited DNA from their mothers.

"Surprisingly, we also show that our mother's mitochondrial DNA seems to influence our own aging," said Larsson. "If we inherit mDNA with mutations from our mother, we age more quickly."

Normal and damaged DNA is passed down between generations. However, the question of whether it is possible to affect the degree of mDNA damage through lifestyle intervention is yet to be investigated. All that the researchers know now is that mild DNA damage transferred from the mother contributes to the aging process.

"The study also shows that low levels of mutated mDNA can have developmental effects and cause deformities of the brain," said lead author Jaime Ross, Ph.D., at the Karolinska Institutet.

"Our findings can shed more light on the aging process and prove that the mitochondria play a key part in aging; they also show that it's important to reduce the number of mutations," said Larsson.

"These findings also suggest that therapeutic interventions that target mitochondrial function may influence the time course of aging," said Barry Hoffer, M.D., Ph.D., a co-author of the study from the Department of Neurosurgery at University Hospitals Case Medical Center and Case Western Reserve University School of Medicine. He is also a visiting professor at the Karolinska Institutet. "There are various dietary manipulations and drugs that can up-regulate mitochondrial function and/or reduce mitochondrial toxicity. An example would be antioxidants. This mouse model would be a 'platform' to test these drugs/diets," said Dr. Hoffer.

The data published in the paper come from experiments on mice. The researchers now intend to continue their work on mice, and on fruit flies, to investigate whether reducing the number of mutations can extend their lifespan.

More information: "Germline mitochondrial DNA mutations aggravate ageing and can impair brain development", Jaime M. Ross, James B. Stewart, Erik Hagström, Stefan Brene, Arnaud Mourier, Giuseppe Coppotelli, Christoph Freyer, Marie Lagouge, Barry J. Hoffer, Lars Olson & Nils-Göran Larsson, *Nature*, online 21 August 2013, DOI: 10.1038/nature12474