MAX PLANCK INSTITUTE FOR **BIOLOGY OF AGEING**

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A mother's genes can hasten her child's ageing process

When we age, our cells change and become damaged. Now, researchers at the Max Planck Institute for Biology of Ageing and Karolinska Institutet have shown that ageing is determined not only by the accumulation of cell damage during our lifetime but also by the genetic material we acquire from our mothers. The results of the study are published in the scientific periodical *Nature*.

There are many causes of ageing, a process that is determined by an accumulation of various kinds of cell damage that impair the function of bodily organs. Of particular importance to ageing, however, seems to be the damage that occurs in the cell's power plant – the mitochondrion.

"The mitochondrion contains its own DNA, the so-called mitochondrial DNA or mtDNA, which changes more than the DNA in the nucleus, and this has a significant impact on the ageing process," says Nils-Göran Larsson, Director at the Max Planck Institute for Biology of Ageing, also a professor in Mitochondrial Genetics at Karolinska Institutet (KI) and leader of the current study alongside Professor Lars Olson (KI). "Many mutations in the mitochondria gradually disable the cell's energy production."

Now, however, the researchers have shown that the ageing process is attributable not only to the accumulation of mtDNA damage during a person's lifetime, but also to their maternally inherited mtDNA.

"Surprisingly, we also show that our mother's mitochondrial DNA seems to influence our own ageing," says Professor Larsson. "If we inherit mtDNA with mutations from our mother, we age more quickly."

Normal and damaged DNA is passed down from generation to generation. However, the question of whether it is possible to affect the degree of mtDNA damage through, for example, lifestyle intervention

is yet to be investigated. All that the researchers know now is that mild mtDNA damage is transferred from the mother and contributes to the ageing process.

They also show in the current study that low levels of mutated mtDNA have developmental effects and may cause deformities of the brain when they are accompanied by large amounts of mtDNA mutations that occur over the lifetime.

“Our findings can shed more light on the ageing process and prove that the mitochondria play a key part in ageing. They also show that it’s important to reduce the number of mutations,” says Professor Larsson.

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.

Original publication:
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