MedicalXpress

Unstable proteins can cause premature ageing

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(PhysOrg.com) -- The normal ageing process has long been linked to problems with cell respiration, the process through which the cells extract energy from nutrients. Researchers at the Swedish medical university Karolinska Institutet have now shown how certain proteins that are synthesised in the cellular mitochondria - popularly known as the cells' power plants - become unstable and disintegrate, which in turn can impair cell respiration and cause premature ageing.

Every time we inhale, the blood transports the oxygen from our lungs to our cells' mitochondria, where it is used to convert the nutrients in our food into a form of energy that the body can use. Problems with this process, called cell respiration, have been linked to numerous conditions, from rare genetic diseases to diabetes, cancer, Parkinson's disease and the normal ageing process.

For cell respiration to function properly, it needs proteins synthesised outside and then imported into the mitochondria, and proteins synthesised within the mitochondria themselves from their own DNA (mtDNA). It has long been known that an accumulation of harmful mutations of mtDNA can cause premature ageing, but just how this happens has remained something of a mystery.

Scientists at Karolinska Institutet in Stockholm have now shown through studies on mice that changes in mtDNA can cause ageing by introducing errors into the proteins manufactured by the mitochondria. The amount of protein is normal, but the proteins are rendered unstable and quickly

disintegrate, leading eventually to the breakdown of cell respiration.

"Our results show that premature ageing is caused by point mutations in the mtDNA, which cause the mitochondrial proteins to become unstable and disintegrate," says Aleksandra Trifunovic, one of the scientists involved in the study.

According to the team, the study, which is presented in the scientific journal Cell Metabolism, provides a better understanding of the interaction between mitochondrial function and the ageing process, and improves the chances of one day finding an efficacious treatment for mitochondrial disorders, something that is currently lacking.

More information: "Random Point Mutations with Major Effects on Protein-Coding Genes Are the Driving Force behind Premature Aging in mtDNA Mutator Mice", Daniel Edgar, Irina Shabalina, Yolanda Camara, Anna Wredenberg, Maria Antoinette Calvaruso, Leo Nijtmans, Jan Nedergaard, Barbara Cannon, Nils-Göran Larsson and Aleksandra Trifunovic, *Cell Metabolism*, 6 August 2009.

Source: Karolinska Institutet (news : web)