

## Cell protein discovery points to healthier aging

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Researchers at The University of Queensland have found an anti-aging function in a protein deep within human cells.



Associate Professor Steven Zuryn and Dr. Michael Dai at the Queensland Brain Institute have discovered that a <u>protein</u> called ATSF-1 controls a fine balance between the creation of new mitochondria and the repair of damaged mitochondria. The research was published in *Nature Cell Biology*.

Mitochondria, with their own DNA, produce energy within cells to power <u>biological functions</u> but the toxic by-products of this process contribute to the rate at which the cell ages.

"In conditions of stress, when mitochondrial DNA has been damaged, the ATSF-1 protein prioritizes repair which promotes cellular health and longevity," Dr. Zuryn said.

As an analogy, Dr. Zuryn likened the relationship to a race car needing a pitstop. "ATSF-1 makes the call that a pitstop is needed for the cell when mitochondria need repairs," he said.

"We studied ATFS-1 in C. elegans, or round worms and saw that enhancing its function promoted cellular health, meaning the worms became more agile for longer. They didn't live longer, but they were healthier as they aged."

"Mitochondrial dysfunction lies at the core of many human diseases, including common age-related diseases such as dementias and Parkinson's.

"Our finding could have exciting implications for healthy aging and for people with inherited mitochondrial diseases."

Understanding how cells promote repair is an important step towards identifying possible interventions to prevent mitochondrial damage.



"Our goal is to prolong the tissue and organ functions that typically decline during aging by understanding how deteriorating <u>mitochondria</u> contribute to this process," Dr. Dai said.

"We may ultimately design interventions that keep mitochondrial DNA healthier for longer, improving our quality of life."

**More information:** Chuan-Yang Dai et al, ATFS-1 counteracts mitochondrial DNA damage by promoting repair over transcription, *Nature Cell Biology* (2023). DOI: 10.1038/s41556-023-01192-y

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