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Surprising findings in mitochondrial biology change long-standing ideas on the protein MTERF1

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New findings in mitochondrial biology thoroughly change the idea scientists had for 20 years on the role and importance of the protein MTERF1. For the first time, Max Planck researcher Mügen Terzioglu and her colleagues in Germany and Sweden investigated in vivo what was up to now only explored in cell culture. Using the mouse as a model organism, she made a surprising discovery: MTERF1 does after all not play the key role in mitochondrial transcription and translation that was hitherto ascribed to it.

Dr Terzioglu's findings will change the way we look at the regulation of mitochondrial function in the cell. With her study, the young researcher demonstrates the way science often works: Long-standing research findings might be overthrown by surprising new insights, thus necessitating future projects with regard to related questions to take on a whole new direction.

Mügen Terzioglu is a researcher in the department of Mitochondrial Biology, headed by Director Nils-Göran Larsson, at the Max Planck Institute (MPI) for Biology of Ageing in Cologne. She carried out her project in with an international team of scientists at the MPI and at the Karolinska Institutet in Stockholm, Sweden.

Proteins are the 'work horses' of an organism. They perform a variety of different processes, for instance regulating genes, controlling

metabolism or making cells perform specialized functions. MTERF1 is such a work horse, carrying out its tasks in the mitochondria, also known as the 'power houses' of the cell since they supply energy by converting components in the food we eat to ATP.

And while the genetic blueprint of a living organism is largely held in the cell nucleus, the mitochondria carry their own hereditary information, also in the form of DNA. In this context, MTERF1's special role is to act as a so-called mitochondrial transcription termination factor: It defines the right stopping place when it comes to transcribe a section on the DNA into RNA. RNA in turn delivers the genetic information to the ribosomes, which can be described as the 'protein-making factories' of the cell. They synthesize proteins according to the instructions held by the RNA.

"Up to now, the role of MTERF1 was only investigated in vitro, using cell culture. And for two decades, this protein was thought to play a crucial role in the regulation of transcription, eventually acting as a key regulator for mitochondrial protein synthesis in mammals", explains Mügen Terzioglu. "However, by engineering an appropriate mouse model for the first time, we have now learned that this is not the case. That was actually quite a surprise to us. It also illustrates the fact that in vitro systems like cell culture can only to a certain extent represent a natural physiological condition. Consequently, the insights gained in vitro must always be verified in vivo. "

The findings of Mügen Terzioglu and her colleagues will change the way we look at proteins and understand their roles in the cell. In particular, a new perspective opens up to better understand the regulation in mitochondrial transcription and translation as well as the stability of the mitochondrial transcripts and their metabolism.

More information: Mügen Terzioglu, et al. 'MTERF1 Binds mtDNA

to Prevent Transcriptional Interference at the Light-Strand Promoter but Is Dispensable for rRNA Gene Transcription Regulation', *Cell Metabolism*, epub ahead of print 2 April 2013. dx.doi.org/10.1016/j.cmet.2013.03.006