**The effect of mitochondrial DNA mutations on oxidative protein folding and drug toxicity**

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The electrons from the mitochondrial oxidative folding machinery (composed of MIA40 and ALR) are channelled to the mitochondrial electron transfer chain (mETC) via the protein ALR. The depletion of mtDNA and mETC caused the increased expresion of ALR at protein level. The regulatory role of ATP and ROS levels on the protein level of ALR was ruled out. The effect of mtDNA depletion on the protein level of ALR has been proved not to be liver specific since the phenomenon could be observed in the case of two other, non-hepatic cell lines.

A manuscript has been prepared from the above results and has been submitted to BBRC.

The highly reactive metabolite of acetaminophen, NAPQI reacts rapidly with GSH causing extensive GSH depletion. NAPQI formation and protein binding, especially to mitochondrial proteins, is an important initiating event of cell death. In acetaminophen treated primary mouse hepatocytes the significant elevation of cell viability could be observed upon ferrostatin-1 treatment. Our results suggest that beyond necroptosis and apoptosis a third programmed cell death, ferroptosis is also involved in acetaminophen induced cell death in primary hepatocytes.

A manuscript has been prepared from the above results and accepted by Pathology and Oncology Research.

MedinProt has been mentioned in the acknowledgement of both manuscript.