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

The ratio of cells with mitochondrial DNA (mtDNA) mutations is higher in elder people. The elevated number of these mutations can lead to mitochondrial dysfunction. On the base of the mitochondrial cascade hypothesis the mitochondrial function and its strength is determined by the genetic background. Finally the age dependent mitochondrial functional fall is also determined by the mtDNA. When the mitochondrial function falls below a critical point characteristic senile dysfunctions appeared. To study these senile dysfunctions the establishment of a cytoplasmic hybrid cell line is needed. This would be one of our main goals. By the aid of this cybrid cell line we would like to study the oxidative protein folding. The terminal electron acceptor of the oxidative protein folding is the mitochondrial electron transfer chain. Thus the efficiency of oxidative protein folding may decline. The declined senile mitochondrial function can affect the biotransformation of drugs and the biotransformation of drugs can perturb the redox states of the ER and the mitochondrion. Thus we also aim at the study of the interaction of these processes in the established cybrid cell lines.

