**The interrelationship of programmed cell deaths and their key proteins: ferroptosis and autophagy in focus**

Our joint research aimed at the determination of the position of the novel cell death form ferroptosis within the other cell death types such as necroptosis, apoptosis and autophagy. According to this plan HepG2 cells were treated by the ferroptosis inducer erastin and samples were collected for protein analysis and GSH determination regularly. The level of autophagy markers has shown a transient increase during erastin treatment, meanwhile the induction of both necroptosis and apoptosis was detected at the end of treatment. However mTOR activation was not observed. Measured intracellular reduced GSH levels showed a significant drop to 50% just after 2h, 12% after 4h, 5% after 6h of treatment and continued to drop below quantification limit through the treatment. Furthermore parallel with the depletion of GSH level, the phosphorylation of JNK could also be observed due to erastin treatment. Interestingly LDH release was not significantly altered possibly because during the cell death process the cellular membrane remains more or less intact. All these results suggest a connection between the necroptotic, autophagic and ferroptotic cell death forms. The level of necroptosis marker RIP3 was elevated by apoptosis inductor treatment in the apoptosis resistant, mtDNA depleted HepG2 Rho0 cells. This observation suggests a switch from apoptosis to necroptosis in these cells.