Alzheimer's disease patients show increased brain iron levels as compared to control individuals. If the iron levels exceed the iron storage capacity of the cells free iron can catalyze the formation of reactive oxygen species (ROS).

Our aim is to understand how AD shifts the normal iron homeostasis and how the iron might participate in the neuropathology.

We analyze if iron withdrawal by iron chelators Deferoxamine (DFO) and Deferiprone (DFP) can reverse morphological changes induced by Amyloid β peptide (Aβ25-35) in human microglia CHME3 cells. Analyses are performed by immunofluorescence-microscopy and fluorescence plate reader. We detect Cytochrome C, mitochondrial membrane potential by MitroTrackerRed and nucleus morphology by Hoechst staining. The proliferation study is preformed using the Alamar Blue assay.

Aβ25-35 cell treatment for 26 h induces morphological changes of the nucleus. In support, quantitative analysis shows increased Hoechst staining of Aβ25-35 treated cells. The proliferation assay shows no significant difference in cell growth of Aβ25-35 treated cells as compared to control. DFO at the concentrations of 30µM and 100 µM are toxic as demonstrated by changes in the morphology of the nucleus and decrease the mitochondrial membrane potential. However, DFP (30µM) have so far not showed any obvious sign of cell toxicity, making DFP a promising iron chelator for the following studies with Aβ25-35.