

Abstract

Mitophagy is a selective type of autophagy in which damaged or senescent mitochondria are removed and recycled. In recent years, the role of mitophagy in the pathophysiology of neurodegenerative diseases, including frontotemporal lobar degeneration (FTLD), has been intensively investigated. It has been shown that mitophagy is impaired at the onset of the neurodegenerative cascade, leading to the accumulation of damaged mitochondria, increased oxidative stress, triggering an inflammatory response, and reduced ATP production, which has a negative impact on energy balance and synaptic transmission ultimately leading to the neuronal death. However, some studies suggest that compromised mitophagy has a different molecular mechanism in FTLD than other neurodegenerative diseases. This bachelor thesis dealt with knowledge about mitophagy in animal and cell models of FTLD disease, especially subtypes with TDP-43 mutation (FTLD-TDP), MAPT (FTLD-tau), GRN (FTLD-GRN), and also summarizes knowledge about the pathophysiology of FTLD, clinical manifestation and current diagnostic criteria.