Abstract

The synthesis of iron-sulfur clusters is an essential cellular process, which depends on complex biosynthetic pathways. In model eukaryotes, these pathways are the ISC pathway in the mitochondria and the CIA pathway in the cytosol. A recent genome and transcriptome analysis showed, that an amitochondriate protist *Monocercomonoides exilis* lacks the canonical ISC pathway, which has been replaced by a bacterial SUF pathway. A close free-living relative of *M. exilis*, *Paratrimastix pyriformis* possesses a mitochondrion-related organelle, yet also possesses a SUF pathway instead of ISC. The acquisition of the SUF pathway has been suggested as the primordial cause for mitochondrial loss in *M. exilis,* which is the first documented eukaryotic organism without a mitochondrion.

The SUF pathway has been the subject of numerous studies in bacteria, however, its role as the core provider of iron-sulfur clusters for eukaryotic cells has been reported in merely a handful of eukaryotes and was based predominantly on genomic data. This thesis focuses on the putative ATPase SufC and the putative scaffold protein SufB. Both proteins were successfully produced in recombinant forms. SufC has been found to possess ATPase activity *in vitro*, which was increased upon interaction with SufB. The conditions for the ATPase activity of SufC have been standardized. The recombinant form of SufC has been used to prepare an antibody, which was utilized to attempt the localization of the SUF pathway in the cell of both *M. exilis* and *P. pyriformis* with little success. An *in vitro* interaction between SufB and SufC from *M. exilis* has been proved using size exclusion chromatography, suggesting an *in vitro* formation of a complex with a size corresponding to either a $SufB_2C$ or $SufBC_2$ stoichiometry and its potential dimerization in the presence of ATP.