ABSTRACT

For proper function proteins should have a native conformation. If their conformation is impaired due to environmental stress or genetic mutation, proteins become prone to aggregation. There exist various types of protein aggregates. Stable non-membraneous inclusions can form which can serve for clearance of aberrant proteins from place where they can interfere with essential cellular processes. Another type of aggregates can serve as transient deposits of proteins thus protecting them from stress conditions. Stress granules (SG) are a such example of transient granules. Their formation is induced by heat shock for example. SGs contain mRNA, components of translation machinery, and other proteins. One of these proteins is Mmi1, small highly conserved protein with unknown function. Association of Mmi1 with stress granules and partial co-localization with chaperon Cdc48 and proteasom indicates Mmi1 can mediate heat stress damaged protein degradation. We have uncovered that yeast prion protein Sup35 is a component of stress granules as well. With regard to its aggregation capability there existed an assumption that prion domain of Sup35 could serve as scaffold for SG assembly. However as we show deletion of prion domain of Sup35 protein does not affect stress granules formation dynamics.

Yeast *Saccharomyces cerevisiae* is a useful model for studying evolutionary conserved cell processes including protein aggregation. Protein aggregates are found in many pathological disorders like prion and other neurodegenerative diseases like Parkinson´s disease (PD). Using *S. cerevisiae* the way that α-synuclein (protein implicated in PD) can impair cells has been elucidated. Our results show that one possible mechanism of toxic action of α-synuclein is preventing assembly of pre-autophagosomal structure (PAS) which could lead to blockage of autophagy.

Keywords: protein aggregation, stress granules, Mmi1, Sup35, α-synuclein, yeast