Human genome consists of approximately 25 000 genes which contain instructions required for development and functioning of a living organism. However, even the smallest change in this genetic information may cause a disease with serious symptoms, which may eventually lead to death. To investigate molecular alterations caused by these changes and to search for an effective therapy, other species are used for modelling a particular disease, with rodents as the most iconic example. However, simple organisms without obvious relation to human, such as fruit fly or yeast, are also in use in laboratory work. They allow to simplify procedures, reduce research costs and apply tools unavailable in higher organisms. This approach is justified because of presence of corresponding genes between the species. It may be surprising, but it is estimated that around 25% of human genes have an equivalent gene in yeast genome and proteins encoded by these genes are expected to perform similar functions. Moreover, yeast and human cells have similar structure and physiology. Thus, it is possible to use yeast for modelling of various diseases. For example, yeast are used in research on Alzheimer's, Parkinson's and other neurodegenerative disease, metabolic disorders, mitochondrial diseases or even cancer.

In our group, we focus on Vps13 proteins. Mutations in genes encoding these proteins are linked with several neurodegenerative diseases, including chorea-acanthocytosis (ChAc) - a rare neurological disorder. Symptoms occur in adult age, progress since appearance and lead to reduced lifespan. Patients suffer from movement disorders, seizures and cognitive impairment. Currently, there is no effective therapy and treatment is limited only to palliative care. This results from limited knowledge about Vps13 proteins function and unknown molecular pathology of ChAc. Since Vps13 protein is present in yeast, this organism can be used as a model of ChAc. We use *vps13*Δ yeast strain, in which gene encoding Vps13 protein was deleted in order to mimic molecular alteration present in ChAc patients. In particular, we try to bypass defects caused by the gene deletion and thus, indicate a potential target for therapeutic intervention. In our experiments we show improvement in some of the defects of *vps13*Δ by increasing copper levels in mutant yeast cells. Copper is an essential microelement which plays a role in maintaining cellular respiration and redox balance. In this project, we would like to answer questions, if and how copper metabolism is disturbed in *vps13*Δ and why increasing copper levels improves functioning of *vps13*Δ. Copper alterations have not been reported before in research on ChAc. Findings in this project may broaden the knowledge on Vps13 function in cell, suggest study direction in research on higher organism and point at a potential therapeutic targets in ChAc patients.