The results from human genome sequencing projects were published in 2001. Even 14 years later we still do not know the function of many genes identified in these projects. Mutations in many of them are leading to genetic disorders, which are individually rare, but taken together affect at least 25 million people in Europe. This is a case of *VPS13* genes. Three of four of *VPS13* genes present in human genome, were described as implicated in neurodegenerative diseases – Chorea acanthocytosis (*VPS13A*), Cohen syndrome (*VPS13B*) and frontotemporal lobar degeneration (*VPS13C*). Despite ongoing research in several laboratories we still don't know the exact role of Vps13 proteins. To analyze the function of human genes the model organisms, like yeast, were successfully used in the past. The advantage of such a strategy is due to simplicity, relatively low cost of experiments and because yeast are genetically and biochemically well characterized and various tools and strain collections are easily available. There is only one *VPS13* gene in yeast, deletion of which manifests with several different changes in cellular metabolism. We would like, based on published data and our new results on yeast Vps13, to extend the knowledge about Vps13 proteins from humans. The work published last year describes yeast Vps13 protein as a component of structures called membrane contact sites, which are implicated in exchange of small molecules (such as calcium ions) and lipids between organelles and are also engaged in signal transduction. The growing list of diseases is connected with malfunctioning of membrane contact sites, among them are neurodegenerative disorders like Niemann-Pick type C, clearly demonstrating their importance. In our project we would like to identify membrane contact sites in which Vps13 proteins are localized by finding the Vps13 interacting protein and lipid partners. We would like to proof hypothesis that due to influence on membrane contact sites Vps13 affects lipid transport and has an impact on maintaining proper lipid composition of the cell membranes. To give proof of this idea the lipid analysis will be performed in collaboration with laboratory of prof. Howard Riezman, University of Geneva, Switzerland. We will accumulate the basic knowledge about the function and regulation of membrane contact sites between organelles. Based on our results from yeast genetics we will find the proteins and pathways which compensate for Vps13 deficiency. The detailed description of Vps13 action in yeast will allow construction of the yeast model useful to screen the library of small molecules and identify potential drugs for treatment of patients suffering from Vps13 deficiency.