

**DESCRIPTION FOR THE GENERAL PUBLIC (IN ENGLISH)** (State the objective of the project, describe the research to be carried out, and present reasons for choosing the research topic - max. 1 standard type-written page)

Some yeast species, known as flavinogenic ones, overproduce riboflavin (vitamin B<sub>2</sub>) under iron starvation. There are also some bacteria and plants (e.g. sunflower and tobacco) which overproduce riboflavin in the media with low iron content. The physiological significance and mechanisms of such regulation remain unknown. These questions are planned to be elucidated in the frame of current project on the model of the most flavinogenic yeast known, *Candida famata*. Earlier we identified gene *SEF1* coding for a putative transcriptional factor involved in regulation of riboflavin synthesis. Deletions and point mutations in this gene eliminate oversynthesis of riboflavin under iron starvation whereas its overexpression increases riboflavin production. However, mechanisms of Sef1 actions remain unknown. Besides, other components of regulatory cascade circuit presumably involved in regulation of riboflavin synthesis are still waiting for identification. Previously, several methods of molecular studies have been developed by us for *C. famata*. Further progress in this field still is slow due to lack of the data on complete genome sequence and the modern methods for genome editing. In the frame of this proposal, we plan to annotate recently sequenced by us genome of *C. famata*. Using genome sequence data and efficient new methodology of genome manipulation, we plan to delete and overexpress putative genes involved in positive (*MET2*) and negative (*SFU1*, *HAP43*, *VMA1*) control of riboflavin synthesis by iron ions. Their characteristics will be studied. Mutants with knock out of *SEF1* gene will be transformed with homologs of this gene from other flavinogenic and non-flavinogenic yeasts with the aim to establish which component of *SEF1* gene, coding sequence or promoter, is responsible for riboflavin overproduction. Besides, the genes coding for putative riboflavin excretase will be isolated, deleted and overexpressed in *C. famata*. To study interactions between genes of positive and negative control, corresponding mutations will be combined in one genome in different combinations to study possible interaction between these genes and their products. The most interesting strains isolated in the frame of this work will be checked for maximal riboflavin production under bioreactor cultivation. The main result of this work will be the model of the regulatory circuit involved in regulation of riboflavin synthesis by iron ions.