THE ROLE OF HOG MAPK SIGNALING PATHWAY DURING OSMOTIC STRESS IN *SACCHAROMYCES CEREVISIAE*

Budding yeast (*Saccharomyces cerevisiae*) cells utilize a conserved mitogen-activated protein kinase (MAPK) signaling cascade (the high-osmolarity glycerol or HOG pathway) during conditions of increased external osmolarity. It evokes cellular responses necessary to permit continued growth. Activation of HOG pathway with Hog1 MAP kinase results in production of glycerol to prevent dehydration and up regulation of other Hog1 dependent genes because of cell adaptation. We were trying to find difference in translation between wild-type cells and two mutants of *hog1* gene before and after 0,4 M NaCl osmotic stress (2, 6, 30 min). We used deletion mutant *hog1Δ* and *hog1-as* mutant with point mutation which allows inhibition of Hog1 MAPK during presence of specific AS inhibitor. We tested AS inhibitor by plate test and have found optimal concentration of 5 μM for blocking Hog1 MAPK in *hog1-as* mutant. Translation profiling proves that osmotic stress decreases translation in general. *Hog1Δ* mutant and *hog1-as* AS inhibited mutant behave similarly and their translation recovers slower than the wild-type's. That confirms that *HOG1* gene is important for cell recovery from the osmotic stress.

Microarray analysis shows that Hog1 dependent genes in wild-type are induced under osmotic stress but not in *hog1* mutants. Genes up regulated after osmotic stress include genes responding to stimulus (stress), desiccation, water deprivation and sodium ion transport. After osmotic stress ribosomal genes are suppressed in wild-type cells but not in *hog1* mutants.

Saccharomyces cerevisiae, MAP kinase, osmotic stress, NaCl, adaptation, glycerol, HOG pathway, Hog1, polysomal profiling analysis, DNA microarray

Saccharomyces cerevisiae, MAP kináza, osmotický stres, NaCl, adaptace, HOG signální dráha, glycerol, Hog1, polyzomální profily, DNA čip