SUPEROXIDE DISMUTASE ACTIVATION DURING OXIDATIVE STRESS IN *S. CEREVISIAE* REQUIRES GLUTATHIONE

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It has been thought that activation of cytosolic superoxide dismutase in *Saccharomyces cerevisiae* is only dependent on chaperone Ccs1, responsible for insertion of copper into the catalytic center of the enzyme. In this work we addressed another mechanism for Sod1 activation that requires glutathione (GSH) and its role in yeast lifespan. Exponential cells were treated or not with 0.5 mM menadione (a source of superoxide radicals) during 1h and thereafter growth was arrested. The effect of oxidative stress pre-treatment on chronological lifespan was measured in wild type (WT) and glutathione deficient strains. The results showed that menadione-induced extension of longevity in the WT strain was suppressed in the glutathione deficient strain. Interestingly enough, menadione treatment increased the expression of *SOD1*, *SOD2* and *CCS1* in both strains. However, although these strains showed the same Sod1 activity before treatment, only the WT presented an increase of Sod activity after menadione exposition. In addition, no Sod activity was observed in a *ccs1D* strain, showing that even if glutathione is required for superoxide dismutase activation, the chaperone is still essential for this process. These results suggest that, in an oxidative stress environment, glutathione-dependent full activation of Sod1 may be required for lifespan extension.