

SII-3-3

ADAPTIVE RESPONSE OF FISSION YEAST TO OXIDATIVE STRESS

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The fission yeast, *Schizosaccharomyces pombe* showed an adaptive response to hydrogen peroxide and menadione (MD), a redox cycling agent. This adaptation was accompanied with differential induction of antioxidant enzymes such as catalase, superoxide dismutase (SOD), glutathione reductase (GR), peroxidase, and glucose-6-phosphate dehydrogenase. Also *S. pombe* cells at the stationary phase showed higher resistance against oxidants, which could be related with remarkable induction of catalase, GR, and G6PD at this phase.

The *pgr1*⁺ gene encoding GR was isolated and characterized. Overexpression of the GR in *S. pombe* conferred resistance against MD stress to the cell. Its mRNA was induced by oxidants, salt, heat as well as starvation. This induction was mainly dependent on the presence of the functional copy of Pap1, a transcriptional regulator of *S. pombe*. Surprisingly, *pgr1*⁺ was revealed to be an essential gene for normal aerobic growth, which was uniquely different from those of *Escherichia coli* and *Saccharomyces cerevisiae*. To examine the role of GR in cell cycle control, a conditional repressible allele *nmt-pgr1* was constructed. This mutant was not able to grow on thiamine-containing plate, again indicating the obligatory requirement of GR activity.

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EXPRESSION OF TWO SUPEROXIDE DISMUTASES IN *STREPTOMYCES COELICOLOR*

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Streptomyces coelicolor contains two distinct superoxide dismutase (SOD) activities: NiSOD and FeZnSOD. The presence of nickel at micromolar concentrations in growth media induced the expression of NiSOD, whereas the expression of FeZnSOD was repressed. The changes in SOD activities were positively correlated with the amount of each enzyme as determined by immunoblotting, suggesting that nickel modulate the amount of each protein. The genes encoding NiSOD (*sodN*) and FeZnSOD (*sodF*) were isolated from *S. coelicolor* DNA using degenerate oligonucleotides corresponding to the N-terminal peptide sequences of the two SODs. DNA sequencing revealed that *sodN* gene encoded a novel protein whereas the *sodF*-encoded polypeptide was similar to other known FeSODs and MnSODs. As determined by analysis of RNA transcripts, the expression of *sodN* and *sodF* genes was transcriptionally regulated by nickel. In NiSOD-overproducing cells, the precursor form of NiSOD subunit was detected and the apoprotein was found to be activated by nickel *in vitro*, suggesting that the expression of *sodN* gene might be regulated by nickel at the posttranslational level as well.