F310 Heat Shock and Oxidative Stress in *Schizosaccharomyces pombe*

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All organisms are sensitive to heat shock and have developed defense mechanism to survive. In cells exposed to elevated temperature, induction of heat shock proteins and accumulation of trehalose, a thermoprotectant, are major physiological changes. Trehalose 6-phosphate synthase (*tps1*$^+$) is an essential enzyme for trehalose synthesis and *S. pombe tps1* deletion muatant was more sensitive to heat shock compared with the wild type. Wild type cells cultured in glycerol medium were more resistant that those grown in glucose medium due to constitutive expression of trehalose 6-phosphate synthase in glycerol medium. However, *Δtps1* mutant did not show this resistance even when grown in glycerol medium. Trehalose may stabilize proteins in their native state under heat-shocked condition. Under heat shock cells also suffer from damage by reactive oxygen species (ROS). Heat shock produced substantial amount of peroxide detectable with the fluorescence probe 2',7'-dichlorofluorescein diacetate. Furthermore many antioxidant enzymes were induced by heat shock. To elucidate the relationship between heat shock and oxidative stress damages, we characterized the role of antioxidant enzymes in thermotolerance of *S. pombe*.

F311 The fusion of large protein to TP domain of HBV Pol inhibits the pregenomic RNA encapsidation and the maturation of HBV genome.

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HBV polymerase replicate viral DNA genome by reverse transcription of an RNA pregenome. These HBV polymerase (Pol) are multi-functional proteins that have protein priming activities, RNA and DNA-dependent elongation activities, and RNaseH activities. Here, we describe the effects of large protein fused to N-terminus of Pol protein on the viral DNA replication. A series of HBV Pol full-length and truncated mutants, which contain N-terminal fusion partner such as MBP (Maltose Binding Protein) or CBP (Calmodulin Binding Peptide), were constructed. The modified Pol proteins were tested for their ability to support viral DNA replication in a *trans*-complementation assay in which HepG2 cells were cotransfected with expression constructs coding for the respective modified Pol proteins and a 1.2 HBV mutant genome which contains frame-shift mutation in the *pol* gene. In the case of MBP-fused Pol (MBP/Pol), pregenomic RNA encapsidation and generation of mature genome were interfered whereas CBP-fused Pol (CBP/Pol) showed normal as wild type Pol. The defects in viral replication of Pol fusion proteins seemed to correlate with the size of fused protein since small protein CBP-fused Pol showed normal. Further investigation with deletion constructs suggested that the site of MBP fusion within Pol also correlated with the defects of Pol fusion protein function; TP domain blocked by MBP fusion, seemed to contribute to these defects. We provide evidence in this report that the acquisition of TP domain in HBV Pol employs another replication mechanism, which does not use packaging system of the gag-pol fusion protein into core particle as HIV-1, but packaging system of Pol protein alone.