Two main lines of research were continued: the first one concerned the mechanisms controlling the fidelity of DNA replication in *Escherichia coli*; the second concerned cellular responses of *Saccharomyces cerevisiae* to DNA damaging agents.

We have been investigating the question whether during chromosomal DNA replication in *Escherichia coli* the two DNA strands may be replicated with differential accuracy. To address this question we set up a new system that allows the examination of mutagenesis either of the leading strand or the lagging strand. Our results suggest that the lagging strand replication of the *E. coli* chromosome may be more accurate than leading strand replication. More recently, we studied mutagenesis of the two strands in *recA730* strains which exhibit constitutive expression of the SOS system. Our results clearly indicate that in *recA730* strains there is a significant difference in the fidelity of replication between the two replicating strands. Based on our data we propose a model describing a possible mechanism of SOS mutagenesis.

To get more insight into cellular responses to DNA damage we have isolated several novel genes of *S. cerevisiae*, the transcription of which is induced by DNA lesions. Main effort was concentrated on the characterization of the *DIN7* gene. We found that Din7p specifically affects the metabolism of mitochondrial DNA (mtDNA). The elevated level of Din7p results in an increased frequency of mitochondrial petite mutants, as well as in a higher frequency of mitochondrial point mutations. Din7p affects also the stability of microsatellite sequences present in the mitochondrial genome. As expected, Din7p was found to be located in mitochondria.

In another project, we found that the *DIN8* gene isolated in our laboratory is identical with the *UMP1* gene encoding a chaperone-like protein involved in 20S proteasome maturation. Interestingly, induction of *UMP1* expression in response to DNA damage is subject to regulation independent of cell cycle checkpoint genes. Since the *din8/ump1* mutants show an increased sensitivity to UV light, we speculate that the proteasome may have a role in the repair of UV damage.

1. REGULATION OF THE FIDELITY OF DNA REPLICATION IN *E. COLI*

I. J. Fijałkowska, P. Jonczyk, M. Maliszewska-Tkaczyk, D. Gawel, M. Białoskórska

Despite progress in understanding the components of SOS mutagenesis, the mechanism of error-prone replication is still unknown. In our laboratory we have been studying mutagenesis on the *E. coli* chromosome in a *recA730* strain. *recA730* strains exhibit constitutive expression of the SOS system and a spontaneous mutator effect without the need for activation of the system by DNA damage. We measured possible differences in the fidelity of synthesis of the leading and lagging strands of DNA during SOS-modified chromosomal replication *in vivo*. The possibility of differential replication fidelity for the two strands arises from the distinct modes of replication of