- **PROJECT-6 : SOS induction in *uvrD* mutants**

The starting point for this project was our finding that mutations in *uvrD*, the gene encoding DNA helicase involved in mismatch repair and nucleotide excision repair, could not be introduced into an ostensibly wild-type strain. The latter was then shown to harbour a cryptic mutation in *lon* (encoding Lon protease), and we established that the basis for the hitherto unknown *uvrD-lon* incompatibility was the occurrence of chronic SOS induction in null *uvrD* strains (1). After having demonstrated that SOS induction in uvrD mutants is not a consequence of defects in mismatch repair, nucleotide excision repair or recombination, we have proposed a model that the *UvrD* helicase is involved in chromosomal DNA replication, by helping unwind secondary structure regions on the lagging strand immediately behind the progressing replication fork *(1)*. Experiments to test this model are in progress.

1. SaiSree, L., M. Reddy and J. Gowrishankar. 2000. lon incompatibility associated with mutations causing SOS induction: null *uvrD* alleles induce an SOS response in *Escherichia coli*. J. Bacteriol. **182**: 3151-3157.

2. SaiSree, L., M. Reddy and J. Gowrishankar. 2001. IS*186* insertion at a hotspot site in *lon* promoter as basis for Lon protease deficiency of *Escherichia coli* B: identification of consensus target sequence for IS*186* transposition. (submitted).
