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.
