

P020 DNA double-strand break repair in *Escherichia coli*
Laura Wardrope and David Leach
Institute of Cell Biology, University of Edinburgh

Double-strand DNA breaks are lethal unless repaired and the mechanisms that afford repair of these lesions have been conserved through evolution. *E. coli* provides an ideal model system in which to study these mechanisms and we have employed a novel method by which to induce the formation of dsDNA breaks in order to study their repair by recombination. The mutagen 2-aminopurine (2-AP) is used to create unmethylated binding sites for EcoKI, an endogenous endonuclease of *E. coli* that cleaves DNA to produce dsDNA breaks. By assaying the viability of recombination repair mutants cultured in the presence of 2-AP over time, relationships between pathways mediated by various recombination proteins at dsDNA break sites have been elucidated.

Using this assay, the propensity for the RecG and the RuvABC Holliday junction resolution pathways to form crossover products following DSB repair has been investigated. This was achieved by combining *recG* and *ruvAC* mutations with mutations in the *XerCD/dif* site-specific recombination pathway responsible for separating chromosome dimers produced by the formation of cross-over products in DSB repair.