

P015 The processing and repair of palindrome-induced DNA double-strand breaks in *Escherichia coli*
John Eykelenboom and David Leach
Institute of Cell Biology, University of Edinburgh

Long DNA palindromes are sites of genome instability (deletions and translocations) in eukaryotic cells. In both prokaryotic and eukaryotic cells they are sites of DNA breakage. Previously, we had obtained genetic evidence that cleavage of long palindromes in *E. coli* was mediated by the SbcCD (Rad50/Mre11) complex and that repair was mediated by homologous recombination. This work led to the proposal of a model where a misfolded palindrome was digested to generate a two-ended DNA break implying that cleavage was a post-replicative event (rather than an event occurring at an arrested replication fork, which would give rise to only one end). This model proposed roles for RecA, RecBCD and RecF in the processing of the ends by homologous recombination.

We have set up an SbcCD-inducible system to test the predictions of this model. We have obtained *in vivo* physical evidence, from pulsed-field gel electrophoresis, concerning the nature of the breaks and are investigating their processing. We have shown that RecA and RecBCD proteins are the primary functions responsible for repair of the breaks. RecF is not required for repair of the cleaved ends but may affect palindrome processing via gap repair and/or affect end-processing.