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Management of E. coli sister chromatid cohesion in response to genotoxic stress

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Aberrant DNA replication is a major source of the mutations and chromosomal rearrangements associated with pathological disorders. In bacteria, several different DNA lesions are repaired by homologous recombination, a process that involves sister chromatid pairing. Previous work in Escherichia coli has demonstrated that sister chromatid interactions (SCIs) mediated by topological links termed precatenanes, are controlled by topoisomerase IV.We have recently observed that that during the repair of mitomycin C-induced lesions, topological links are rapidly substituted by an SOS-induced sister chromatid cohesion process involving the RecN protein. The loss of sister chromatid interactions and viability defects observed in the absence of RecN were fully compensated by alterations in topoisomerase IV, demonstrating that the main role of RecN during DNA repair is to promote contacts between sister chromatids. RecN also promotes the re-mixing of chromosomes that have initiated their segregation. RecN modulates the dynamics of repair foci suggesting that SCIs significantly contribute to the repair of DNA double-stand breaks (DSBs).