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RecF, UvrD, RecX and RecN proteins suppress DNA degradation at DNA double-strand breaks in *Escherichia coli*

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ABSTRACT

Double strand breaks (DSBs) in *E. coli* chromosome (such as those induced by gamma rays) are repaired by recombination repair, during which a certain amount of DNA gets degraded. We monitored DNA degradation in gamma-irradiated cells to assess processing of DSBs. DNA degradation in irradiated cells is regulated by RecA protein concentration and its affinity of ssDNA binding, as well as by exonucleases that trim 3'-terminated ss tails. Here we determined the effects of proteins that affect formation and stability of RecA nucleofilaments on DNA degradation and cell survival. RecF and UvrD suppressed DNA degradation through RecA protein function and SOS induction, while also improving gamma survival. RecF and UvrD function in one pathway. Acting along with RecF, RecX suppressed DNA degradation and stimulated gamma-survival, which also depends on RecA protein and SOS induction. Furthermore, we determined a role in DNA degradation of several proteins that participate in DSB repair. RecN was required for DNA repair and for degradation suppression, acting on the RecBCD pathway. Furthermore, we show that SSB protein overproduction did not affect DNA degradation. Inactivation of RecG and RuvABC, proteins that catalyze the postsynaptic phase of recombination repair of DSBs, also did not affect DNA degradation, suggesting that once formed, recombination intermediates are not subject to DNA degradation, and that the postsynaptic phase is an irreversible, single-round process, unlike the presynaptic phase, which is mostly repetitive.

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