Title:	Essential Role for an Isoform of Escherichia coli Translation Initiation Factor IF2 in Repair of Two-Ended DNA Double-Strand Breaks
Authors:	Mallikarjun, Jillella (/jspui/browse?type=author&value=Mallikarjun%2C+Jillella) Gowrishankar, J. (/jspui/browse?type=author&value=Gowrishankar%2C+J.)
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Abstract:	In Escherichia coli, three isoforms of the essential translation initiation factor IF2 (IF2-1, IF2-2, and IF2-3) are generated from separate in-frame initiation codons in infB. The isoforms have earlier been suggested to additionally participate in DNA damage repair and replication restart. It is also known that the proteins RecA and RecBCD are needed for repair of DNA double-strand breaks (DSBs) in E. coli. Here, we show that strains lacking IF2-1 are profoundly sensitive to two-ended DSBs in DNA generated by radiomimetic agents phleomycin or bleomycin, or by endonuclease I-SceI. However, these strains remained tolerant to other DSB-generating genotoxic agents or perturbations to which recA and recBC mutants remained sensitive, such as to mitomycin C, type-2 DNA topoisomerase inhibitors, or DSB caused by palindrome cleavage behind a replication fork. Data from genome-wide copy number analyses following I-SceI cleavage at a single chromosomal locus suggested that, in a strain lacking IF2-1, the magnitude of recombination-dependent replication through replication restart mechanisms is largely preserved but the extent of DNA resection around the DSB site is reduced. We propose that in the absence of IF2-1 it is the synapsis of a RecA nucleoprotein filament to its homologous target that is weakened, which in turn leads to a specific failure in assembly of Ter-to-oriC directed replisomes needed for consummation of two-ended DSB repair.
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