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Title:	Modulation of RecFORQ- and RecA-Mediated Homologous Recombination in Escherichia coli by Isoforms of Translation Initiation Factor IF2
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Abstract:	Homologous recombination (HR) is critically important for chromosomal replication, as well as DNA damage repair in all life forms. In Escherichia coli, the process of HR comprises (i) two parallel presynaptic pathways that are mediated, respectively, by proteins RecB/C/D and RecF/O/R/Q; (ii) a synaptic step mediated by RecA that leads to generation of Holliday junctions (HJs); and (iii) postsynaptic steps mediated sequentially by HJ-acting proteins RuvA/B/C followed by proteins PriA/B/C of replication restart. Combined loss of RuvA/B/C and a DNA helicase UvrD is synthetically lethal, which is attributed to toxicity caused by accumulated HJs since viability in these double mutant strains is restored by removal of the presynaptic or synaptic proteins RecF/O/R/Q or RecA, respectively. Here we show that, as in ΔuvrD strains, ruv mutations confer synthetic lethality in cells deficient for transcription termination factor Rho, and that loss of RecFORQ presynaptic pathway proteins or of RecA suppresses this lethality. Furthermore, loss of IF2-1 (which is one of three isoforms [IF2-1, IF2-2, and IF2-3] of the essential translation initiation factor IF2 that are synthesized from three in-frame initiation codons in infB) also suppressed uvrD-ruv and rho-ruv lethalities, whereas deficiency of IF2-2 and IF2-3 exacerbated the synthetic defects. Our results suggest that Rho deficiency is associated with an increased frequency of HR that is mediated by the RecFORQ pathway along with RecA. They also lend support to earlier reports that IF2 isoforms participate in DNA transactions, and we propose that they do so by modulation of HR functions.
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