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# Microhomology-Mediated End Joining: Good, Bad and Ugly

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## Abstract

DNA double-strand breaks (DSBs) are induced by a variety of genotoxic agents, including ionizing radiation and chemotherapy drugs for treating cancers. The elimination of DSBs proceeds via distinctive error-free and error-prone pathways. Repair by homologous recombination (HR) is largely error-free and mediated by RAD51/BRCA2 gene products. Classical non-homologous end joining (C-NHEJ) requires the Ku heterodimer and can efficiently rejoin breaks, with occasional loss or gain of DNA information. Recently, evidence has unveiled another DNA end-joining mechanism that is independent of recombination factors and Ku proteins, termed alternative non-homologous end joining (A-NHEJ). While A-NHEJ-mediated repair does not require homology, in a subtype of A-NHEJ, DSB breaks are sealed by microhomology (MH)-mediated base-pairing of DNA single strands, followed by nucleolytic trimming of DNA flaps, DNA gap filling, and DNA ligation, yielding products that are always associated with DNA deletion. This highly error-prone DSB repair pathway is termed