Preferential repair of UV damage in highly transcribed DNA diminishes UV-induced intrachromosomal recombination in mammalian cells.

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Abstract

The relationships among transcription, recombination, DNA damage, and repair in mammalian cells were investigated. We monitored the effects of transcription on UV-induced intrachromosomal recombination between neomycin repeats including a promoterless allele and an inducible heteroallele regulated by the mouse mammary tumor virus promoter. Although transcription and UV light separately stimulated recombination, increasing transcription levels reduced UV-induced recombination. Preferential repair of UV damage in transcribed strands was shown in highly transcribed DNA, suggesting that recombination is stimulated by unrepaired UV damage and that increased DNA repair in highly transcribed alleles removes recombinogenic lesions. This study indicates that the genetic consequences of DNA damage depend on transcriptional states and provides a basis for understanding tissue- and gene-specific responses to DNA-damaging agents.