

Isolation and characterization of *HCI*: a novel human DNA repair gene

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ABSTRACT. Nucleotide excision repair (NER) acts on a broad spectrum of large lesions, while base excision repair removes individual modified bases. Although both processes have been well studied in human cells, novel genes involved in these DNA repair pathways have been described. Using a heterologous complementation approach, we identified a fetal human cDNA that complemented two *Escherichia coli* mutants that are defective in 3-methyl adenine glycosylase and in three endonucleases, all of which are enzymes with important roles in base excision repair. The central cDNA open reading frame complemented NER mutant strains and promoted an increase in survival rate of bacteria exposed to UV light. The corresponding protein was able to restore nucleotide-excision-repair activity when added to a cell extract from Chinese hamster ovary cells deficient in the ERCC1 protein, an enzyme known to promote incision at the 5' end of the lesion during NER. In contrast, that protein was not able to complement XPG Chinese hamster ovary cells deficient in the 3' incision step of NER. These data indicate a new human repair gene, which we named *HCI*; it is involved

in the recognition of two kinds of DNA lesions and it contributes to the 5' DNA incision step in NER.

Key words: DNA repair; Base excision repair; Nucleotide excision; Heterologous complementation; Genome maintenance; DNA damage