DNA interstrand cross-linking agents such as nitrogen mustards, mitomycin C, and psoralen are widely used in cancer chemotherapy because of their high cytotoxicity to dividing cells (1). A single unrepaired interstrand cross-link (ICL)1 can kill a bacterial or yeast cell, and about 40 unrepaired ICLs can kill a mammalian cell (2, 3). Moreover, DNA ICLs induce mutations and chromosomal rearrangements. The most extensive studies of cross-link repair have been carried out in *Escherichia coli*, in which the major interstrand cross-link repair pathway is well characterized, both genetically and biochemically. ICL repair in eukaryotes is less well understood, and there may be several pathways (4). In *E. coli* and in *Saccharomyces cerevisiae*, the repair of ICLs depends on both nucleotide excision repair and homologous recombination (5–7). In mammals, mutant cell lines sensitive to cross-linking agents have been useful to identify proteins that might be involved in ICL repair. XPF and ERCC1 mutant cell lines, in addition to being defective in nucleotide excision repair, are particularly sensitive to cross-linking agents (8), suggesting that these proteins play a special role in ICL repair. Similarly, mutations in the XRCC2 and XRCC3 genes, encoding proteins with sequence homology to the human RAD51 protein, confer sensitivity to cross-linking agents (9). Fanconi anemia cell lines are also particularly susceptible to such agents. Thus far, eight Fanconi anemia complementation groups have been defined, and six genes have been mapped and cloned, FANCA, FANCC, FANCD2, FANCE, FANCF, and FANCG (1). Studying Fanconi anemia will likely be of great value in understanding human ICL repair mechanisms, but the function of the FANC proteins is still unclear.

In *Drosophila melanogaster*, mutations in the *mus308* gene lead to marked sensitivity to cross-linking agents. Experiments suggested that some incision event takes place in *mus308* mutants, but full repair does not take place (10). The C-terminal portion of the *Mus308* protein encodes a DNA polymerase, whereas the N-terminal portion encodes the seven characteristic motifs found in DNA and RNA helicases (11). Sharief et al. (12) cloned a human cDNA for *POLQ*, encoding a polypeptide homologous to the *Mus308* polymerase domain but with no corresponding helicase region. A longer cDNA sequence for *POLQ*, deposited with the NCBI database by Abbas and Linn (NCBI accession number NM_006596, predicts a presumed full-length protein with the polymerase domain in the C-terminal portion and a helicase domain at the N terminus, similar to *D. melanogaster* *Mus308*.

In many DNA repair pathways, the function of DNA helicases is essential. In particular, UvD helicase is needed to repair ICLs in *E. coli* (13). Moreover, defects in DNA helicases are the causes of several human diseases. BLM and WRN, the products of the Bloom and Werner syndrome genes, are members of the RecQ family of DNA helicases (14, 15). Although their most critical roles in cells are not precisely known, they participate in pathways of DNA damage tolerance. Another member of the RecQ family of helicases, RECQ4, has been implicated in a subset of cases of Rothmund-Thompson syndrome (16). XPD and XPB helicases, two of the subunits of TFIIH transcription/repair factor, are involved in nucleotide excision repair (17), and mutations in their genes can lead to the disorders xeroderma pigmentosum and trichothiodystrophy.

With the aim of isolating new proteins implicated in DNA cross-link repair, we sought mammalian homologs of the putative helicase portion of *D. melanogaster* *Mus308*. We report
identification of new human and mouse genes and the biochemical activity of the human gene product.

**EXPERIMENTAL PROCEDURES**

**Cloning of Human and Mouse HEL308**—Human and mouse HEL308 genes were cloned by 3' and 5' rapid amplification of cDNA ends using a CLONTECH SMART RACE cDNA Amplification kit. Primers were designed from human expressed sequence tags AA625285, H08004, and R24580 in order to clone the human HEL308 coding region. cDNA was prepared from the testis cancer cell line 833K and the bladder cancer cell line MGH-U1 (18) according to the manufacturer's instructions (CLONTECH). Two fragments of 1767 bp (3' RACE) and 2167 bp (5' RACE) were combined to obtain the entire HEL308 coding region. The mutant HEL308 (500) gene was generated with the QuickChange site-directed mutagenesis kit (Stratagene); the single AAT to ATG (lysine to methionine) substitution was confirmed by sequencing. Primers were designed from mouse expressed sequence tag AA517170 to clone mouse HEL308 by RACE PCR from mouse leukemia L1210 cells RNA (19) and mouse total liver RNA (Ambion Inc.) using a CLONTECH SMART RACE cDNA Amplification kit.

**Purification of Human HEL308**—The human HEL308 open reading frame was subcloned into plasmid pFastBac HTb, and the Bac-to-Bac baculovirus expression system (Invitrogen) was used to obtain recombinant baculovirus to infect Sf9 cells. A 500-ml spinner flask of Sf9 cells was prepared from the testis cancer cell line 833K and the bladder cancer cell line MGH-U1 (18) according to the manufacturer's instructions (CLONTECH). Gel Filtration Calibration Kit (Amersham Biosciences, Inc.) equilibrated with Buffer C containing 0.1 M KCl, 20 mM Tris-HCl, pH 7.5, 4 mM MgCl2, 1 mM dithiothreitol, 50 μg/ml bovine serum albumin, 2 mM ATP, and 60 μM of substrate in a 20-μl volume. Human RPA was produced as a recombinant protein in E. coli, purified according to the method of Henricksen et al. (20), and included where indicated. Incubation was for 30 min at 37 °C. Reactions were terminated by the addition of 6 μl of gel loading buffer (0.25% bromophenol blue, 0.25% xylene cyanol, 30% glycerol, and 0.17 μM EDTA). DNA species were separated by electrophoresis through non-denaturing 10% polyacrylamide gels that were dried and analyzed by autoradiography or quantified with a Fuji phosphorimaging device.

**Human and Mouse Helicase HEL308**—With the aim of isolating proteins involved in DNA cross-link repair, we searched data bases with the DNA helicase portion of Drosophila mus308. We found three human expressed sequence tags, AA625285, H08004, and R24580 (NCBI accession numbers), homologous to motif VI of the D. melanogaster mus308 helicase domain. From this ~700-bp sequence, primers were designed to clone the whole gene by 3' and 5' rapid amplification of cDNA ends (CLONTECH SMART RACE cDNA Amplification kit). Total RNA was prepared from a testis cancer cell line (833K) and a bladder cancer cell line (MGH-U1). Two different amplification reactions were performed for each cell line, and in both cases, two fragments of 1767 and 2167 bp were amplified through 3' RACE and 5' RACE, respectively. Six different PCR products (three from the 3' RACE and three from the 5' RACE) were sequenced to exclude mistakes obtained by PCR amplification. Finally, the complete open reading frame (3303 bp) was constructed by subcloning the 5'- and 3'-fragments into NcoI and KpnI sites of plasmid pFastBac HTb. To reflect the helicase activity described below and the high homology to mus308, we designated the previously unannotated gene as HEL308.

The human HEL308 gene maps on chromosome 4q21 and encodes for a protein of 1101 amino acids with a predicted molecular size of 124.5 kDa. We also found a mouse expressed sequence tag AA517170 that allowed us to clone mouse Hel308 by 3' and 5' rapid amplification of cDNA ends (CLONTECH SMART RACE cDNA Amplification kit). Total RNA was prepared from a testis cancer cell line (833K) and a bladder cancer cell line (MGH-U1). Two different amplification reactions were performed for each cell line, and in both cases, two fragments of 1767 and 2167 bp were amplified through 3' RACE and 5' RACE, respectively. Six different PCR products (three from the 3' RACE and three from the 5' RACE) were sequenced to exclude mistakes obtained by PCR amplification. Finally, the complete open reading frame (3303 bp) was constructed by subcloning the 5'- and 3'-fragments into NcoI and KpnI sites of plasmid pFastBac HTb. To reflect the helicase activity described below and the high homology to mus308, we designated the previously unannotated gene as HEL308.

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40% identity (55% similarity) over the same region. No other human sequences were found approaching these high levels of homology to the Drosophila Mus308 helicase domain.

Purification of the Human HEL308 Protein—The HEL308 open reading frame was cloned into plasmid pFastBac HTb, linked to a hexahistidine tag at the N-terminal end. We also generated a cDNA expression construct encoding a tagged version of HEL308 with a single amino acid substitution at position 365. This conserved lysine residue is in the putative Walker A nucleotide binding motif and was changed to a methionine residue using site-directed mutagenesis. The resulting protein is referred to as HEL308K365M. For a number of ATPases, including E. coli UvrD and S. cerevisiae Rad3, mutation of the equivalent lysine residue severely impairs nucleotide triphosphate hydrolysis, although the overall structure of the protein seems to remain intact (21–23).

For protein production, both cDNAs were placed under the transcriptional control of the polyhedrin promoter in recombinant baculoviruses. These viruses were used to transfect Sf9 cells. The proteins HEL308 and HEL308K365M were detected in cell extracts by immunoblotting (data not shown). The extract was fractionated over a Ni2+/H11001-nitrilotriacetate superflow resin (Qiagen), a Mono Q column, and a Mono S column, and samples of the HEL308-containing fractions were analyzed by electrophoresis through an SDS-polyacrylamide gel (Fig. 2). A sample of the final purification step of the HEL308K365M protein, which was produced and purified in exactly the same manner as the wild type protein, is shown in Fig. 2, lane 5.

ATPase and Helicase Activity of Human HEL308—To characterize human HEL308, helicase and ATPase assays were performed on fractions along the Mono Q column gradient. Human HEL308 co-fractionated with a helicase activity and an ATPase activity (Fig. 3), though protein eluting after the peak fraction may be in a less active form. The helicase assay tested the ability to displace a 24-nt oligonucleotide from M13mp18 viral DNA (Fig. 3B). In the ATPase assay, released radiolabeled phosphate was separated from non-hydrolyzed ATP by thin layer chromatography, and the extent of hydrolysis was quantified (Fig. 3C). This ATPase activity was dependent upon the addition of single-stranded DNA to the reaction mixture, as found for other related enzymes (24).

Human HEL308 was further purified through a Mono S column. HEL308 ATPase activity was strongly stimulated by single-stranded DNA but not by double-stranded plasmid DNA (Fig. 4A). Unwinding of the 40-bp partial duplex substrate catalyzed by HEL308 required a nucleotide cofactor (Fig. 4B), as expected. Substituting 4 mM Mn2+ instead of Mg2+ gave barely detectable helicase activity (Fig. 4B, lane 1). A non-hydrolyzable ATP analog, AMP-PNP, could not support the helicase activity (Fig. 4B, lane 2). To determine whether nucleo-

Fig. 1. Sequence alignment of human and mouse HEL308 with A. thaliana MUS308, human POLQ, D. melanogaster Mus308, and C. elegans MUS-1 and with human WRN and BLM DNA helicases and E. coli Dead RNA helicase. Roman numerals indicate regions conserved in DNA and RNA helicases, and the bars mark their approximate extent. The arrow indicates the lysine that was mutated to methionine in mutant HEL308K365M. Positions with six or more identical residues are shaded. The asterisks denote residues that are conserved in the MUS308 subfamily of helicases but not in other known or putative helicases. The sequence alignment was carried out using the Clustal X program.
otide hydrolysis was needed for helicase activity, increasing amounts of AMP-PNP or ATPγS were added to reaction mixtures that contained 2 mM ATP (Fig. 4C). AMP-PNP at 10 mM and ATPγS at 2 mM were competitive inhibitors of helicase action, indicating that they bind to HEL308 in place of ATP and that nucleotide hydrolysis is necessary for activity. The result also suggests that ATPγS has a higher affinity for HEL308 nucleotide binding sites than AMP-PNP. *Drosophila* RecQ5 helicase activity is similarly inhibited by an equimolar ratio of ATPγS to ATP but not by an equimolar ratio of AMP-PNP to ATP (25). Interestingly, 2–4 mM AMP-PNP stimulated unwinding activity by about 2-fold in the presence of 2 mM ATP. This may suggest cooperative binding to an enzyme that has both catalytic and noncatalytic nucleotide binding sites, as found for the hexameric T7 gene 4 and *E. coli* Rho helicases (26, 27). T7 gene 4 helicase prefers dTTP as a nucleotide cofactor, and the non-hydrolyzable nucleotide analog dTMP-PCP can bind to a noncatalytic site with little effect on dTTPase turnover (27).

To determine the nucleotide preference of HEL308, eight nucleotides were tested for their ability to support unwinding of the 40-bp partial duplex substrate. At the 2 mM nucleotide concentration used, only ATP and dATP gave detectable activity with 76% displacement in the presence of dATP and 33% displacement with ATP (Fig. 4B). ATP was used in further experiments since the ATP concentration in the cell is higher than the dATP concentration.

**Unwinding of Longer DNA Duplexes by HEL308**—To determine whether HEL308 can unwind longer DNA duplexes, similar DNA substrates were constructed containing 60- or 70-nt fragments annealed to M13mp18. DNA helicase assays were performed with each DNA substrate in the presence of increasing amounts of HEL308 (Fig. 5). The mutant HEL308K365M could displace none of the substrates (Fig. 5, lanes 3, A–F) and had no ATPase activity (not shown), confirming that these catalytic activities are due to wild type HEL308 enzyme. The HEL308 helicase displaced both the 60- and the 70-nt fragments, although with much less efficiency than the 20- and 40-nt fragments. After 30 min, 1.4 nM HEL308 displaced 89, 32, 26, and 2.5% of the 20-, 40-, 60-, and 70-nt oligonucleotides, respectively.

Substrates with 3′- or 5′-unpaired flaps were also tested. A non-complementary stretch of (A)10 was added either 5′ or 3′ of the 40-nt fragment (Fig. 5, C and D). In neither case was unwinding activity changed, as compared with the displacement of the 40-nt fragment (Fig. 5G). We followed the unwinding reaction of the 20-bp partial duplex substrate at different time points using 0.14 nM HEL308 (Fig. 5H). After 10 min, 20% of the substrate was unwound with 50% unwinding reached after 18 min. The percentage of unwound substrate increased further with time.

**Single-stranded DNA-binding Protein RPA Stimulates the HEL308 Helicase**—One possible reason for the fact that the HEL308 helicase is less efficient in unwinding DNA duplexes of increasing length might be that the displaced single strand tends to re-annell. If this is the case, the activity of HEL308 might be stimulated by single-stranded DNA-binding proteins. To test this possibility, the helicase activity was measured in the presence of purified human RPA (Fig. 6). In reactions containing the annealed 70-nt fragment and 2.8 nM HEL308, RPA stimulated HEL308 helicase activity with maximal stimulation at ~2 nM RPA. At this concentration, RPA increased displacement of the 70-nt fragment by 2.6-fold. 7.5 nM RPA did
not stimulate HEL308 helicase activity (Fig. 6, lane 8), and concentrations of RPA equal or higher than 15 nM inhibited its activity (data not shown). These results suggest that RPA stimulates HEL308 by binding to the unwound regions produced by HEL308 helicase activity and inhibiting re-annealing. Given a binding site of about 30 nt for human RPA (28), 15 nM RPA covers approximately 25% of the M13 single-stranded DNA annealed to the 70-nt fragment. This concentration of RPA may more likely inhibit HEL308 translocation on DNA rather than its binding to DNA. The order of addition of RPA and HEL308 did not affect these results (data not shown).

The HEL308 Helicase Acts in the 3' to 5' Direction—To determine the polarity of the HEL308 helicase, the 60-nt oligonucleotide was annealed to M13mp18 DNA, cut with SalI, and labeled at the 3'-ends with $^{32}$P. This produced linear M13mp18 DNA with a 20-nt fragment annealed to its 5'-end and a 44-nt fragment annealed to its 3'-end. HEL308 displaced only the 20-nt fragment, indicating translocation in the 3' to 5' direction (Fig. 7A). Because HEL308 helicase activity decreases as a function of the size of the annealed oligonucleotide that it has to displace, a second DNA substrate was prepared. In this case, 42- and 32-nt fragments were annealed at the 5'- and 3'-end of linear M13mp18 DNA, respectively. HEL308 helicase activity displaced only the 42-nt fragment (Fig. 7B). These results confirm that the polarity of the HEL308 helicase is 3' to 5' relative to the single-stranded region.

**Gel Filtration Analysis of Human HEL308**—The oligomeric structure of a DNA helicase is an important parameter that may have implications for its mechanism of action (29). The native molecular weight of human HEL308 was estimated from gel filtration analysis. Fraction 28 of the Mono Q column was loaded onto a Superose column equilibrated in buffer that included 0.5 M KCl. Proteins were eluted, and fractions were analyzed. Immunoblotting, ATPase, and helicase activities all co-eluted at a position corresponding to a molecular size of ~600 kDa as determined from standards (Fig. 8). This would be consistent with a possible hexameric association given the predicted molecular size of 124 kDa for a HEL308 monomer. Under the same gel filtration conditions, the mutant HEL308K365M protein eluted at the same position as wild type HEL308 as determined by immunoblotting (not shown). This suggests that the point mutation K365M in the Walker A motif does not alter the quaternary structure of the protein.

**DISCUSSION**

We isolated human and mouse homologs of the helicase domain of D. melanogaster mus308 and designated them HEL308. Drosophila mus308 encodes a DNA polymerase in its C-terminal portion and a DNA helicase in its N-terminal portion (11). We found homologs of Drosophila mus308 in C. elegans and A. thaliana in addition to those in human and mouse, but we could not find any homolog in S. cerevisiae or in other yeast and bacterial species. The presumably full-length cDNA for human POLQ also has a predicted helicase domain at the N terminus, but its activity remains to be demonstrated.

The MUS308 family of helicases is part of the superfamily II of DNA and RNA helicases. Harris et al. (11) identified other sequences in GenBank™ that have sequence similarity to the helicase domain of Drossophila mus308. Besides the seven characteristic motifs of the superfamily II, these putative helicases include motifs Ib and IVa containing characteristic residues in the MUS308 subfamily. Motif I (the Walker A box) is unusual in having a serine in place of the glycine found in almost all the MUS308 subfamily. Motif I (the Walker A box) is unusual in having a serine in place of the glycine found in almost all helicases of the DEXH family, whereas motif IV has characteristics of the so-called DEXH family of helicases, which seem to combine motifs from various families. Motif II is characteristic of the so-called DEXH family of helicases, which includes many “repair/recombination” helicases, such as Rad3, Rad54, WRN, and BLM, whereas motif IV has characteristics of the DEAD family of RNA helicases.

HEL308 is a single-stranded DNA-dependent ATPase and DNA helicase. It translocates on DNA with 3' to 5' polarity and
efficiently displaces 20- to 40-mer duplex oligonucleotides. Although activity on longer substrates is lower, it can be stimulated by the single-stranded DNA-binding protein RPA. Other helicases have been shown to be stimulated by single-stranded DNA-binding proteins. For example, *S. cerevisiae* MER3 helicase efficiently unwinds a 631-nt fragment only in the presence of RPA (30). Substrates with 3' or 5'-unpaired flaps do not stimulate HEL308 activity, whereas many helicases, such as *E. coli* DnaB, phage T4 gene 41 protein, and phage T7 gene 4 protein, require a forked DNA substrate to initiate DNA unwinding *in vitro* (29). WRN helicase displaces a 40-nt oligonucleotide much more efficiently when two unpaired 10-mers are added to the 40-mer at its 3'- and 5'-ends (31). Both BLM and WRN prefer a fork DNA substrate to unwind DNA (32). Gel filtration analysis suggests that HEL308 behaves as a multimer, possibly a hexamer. A few DNA helicases studied to date operate as monomers, such as PcrA (33) and T4 Dda helicase (34). Many more helicases are active as oligomers, often as

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**FIG. 5.** Unwinding reactions on different DNA substrates catalyzed by HEL308. The unwinding activity of HEL308 DNA helicase was evaluated with six different substrates and increasing amounts of the protein HEL308 (A–F). A schematic diagram of the structure of each substrate is shown on the left of each autoradiogram. In the first two lanes, no protein was added. Lane B, boiled substrate. In the third lane, K365M mutant protein was added at a concentration of 2.8 nM. Remaining lanes, increasing amounts of purified HEL308 (in nM). G, plot of fragment displacement (%). Each point is the mean value of three different experiments. H, displacement of the 20-nt fragment from M13mp18 viral DNA at different times with 0.14 nM HEL308. In the first two lanes, no protein was added. Lane B, boiled substrate.

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**FIG. 6.** RPA Stimulates displacement of the 70-nt fragment. Displacement of the 5'-end-labeled 70-nt fragment annealed to M13mp18 single-stranded DNA was examined in the presence of increasing amounts of human RPA. Reaction mixtures contained ~6 fmol of DNA substrate. In the first two lanes, no protein was added. Lane B, boiled substrate. Remaining lanes, 2.8 nM purified HEL308 was added together with increasing amounts of RPA.
hexamers (T7 gene 4, T4 gp41, E. coli DnaB, and BLM) or dimers (E. coli Rep helicase) (29, 35). Studies by electron microscopy will reveal whether HEL308 forms multimeric structures.

Drosophila mus308 is believed to be involved in the repair of interstrand DNA cross-linking damage since mus308 mutants are hypersensitive to DNA cross-linking agents such as photo-activated psoralen, diepoxybutane, and nitrogen mustard but are not sensitive to the monofunctional alkylating agent methyl methanesulfonate (11). Not much is known about how interstrand cross-links are repaired in eukaryotes. Biochemical and genetic analyses in prokaryotes indicate that DNA interstrand cross-links are repaired by an excision-recombination mechanism (36, 37). In E. coli, removal of a DNA interstrand cross-link is initiated by the UvrABC endonuclease complex, which incises one strand on each side of the cross-link. The 5' nuclease activity of DNA polymerase I, in concert with the UvrD helicase, generates a gap at the site of incision. The resulting single-stranded region provides a substrate for binding of RecA protein and for the initiation of homologous pairing and strand exchange. The polymerase activity of DNA polymerase I can then carry out repair synthesis with an undamaged homolog as a template. Some aspects of this mechanism of interstrand cross-link repair may be conserved in eukaryotes. Genetic data suggest that both excision and recombination are involved in the repair of interstrand cross-links in eukaryotes as well. Mus308 and POLQ belong to the A family of DNA polymerases, as does E. coli DNA polymerase I. Both UvrD and HEL308 are 3'→5' multimeric DNA helicases. This direction of translocation along DNA would fit a model where coupling between a helicase and a polymerase coordinates duplex unwinding and polymerization. Gene 4 of bacteriophage T7 encodes a protein (gp4) that has both a helicase and an RNA primase domain. gp4 forms hexameric rings that can translocate along single-stranded DNA, coupling the unwinding of duplex DNA with the synthesis of short RNA primers that are elongated by T7 DNA polymerase (38). It will be interesting to analyze whether HEL308 interacts with a polymerase and forms a complex with an activity similar to T7 gp4. The existence of both HEL308 and the very similar putative helicase of POLQ raises further questions concerning the functions of these enzymes. It is possible that they function in DNA repair processes in different tissues.

Fanconi anemia cell lines are sensitive to interstrand cross-linking agents. Thus far, eight Fanconi anemia complementation groups have been defined, and six genes have been mapped and cloned (1). We are currently investigating the possibility
that HEL308 might be one of the two remaining uncloned genes, FANCB and FANCD1.

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A Human DNA Helicase Homologous to the DNA Cross-link Sensitivity Protein

Mus308

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