The UvrB protein is the central recognition protein in bacterial nucleotide excision repair. We have shown previously that the highly conserved \( \beta \)-hairpin motif in *Bacillus caldotenax* UvrB is essential for DNA binding, damage recognition, and UvrC-mediated incision, as deletion of the upper part of the \( \beta \)-hairpin (residues 97–112) results in the inability of UvrB to be loaded onto damaged DNA, defective incision, and the lack of stranddestabilizing activity. In this work, we have further examined the role of the \( \beta \)-hairpin motif of UvrB by a mutational analysis of 13 amino acids within or in the vicinity of the \( \beta \)-hairpin. These amino acids are predicted to be important for the interaction of UvrB with both damaged and non-damaged DNA strands as well as the formation of salt bridges between the \( \beta \)-hairpin and domain 1b of UvrB. The resulting mutants were characterized by standard functional assays such as oligonucleotide incision, electrophoretic mobility shift, stranddestabilizing, and ATPase assays. Our data indicated a direct role of Tyr\(^{93}\), Glu\(^{99}\), and Arg\(^{123}\) in damage-specific DNA binding. In addition, Tyr\(^{96}\) plays an important but less essential role in DNA binding by UvrB. Finally, the formation of salt bridges between the \( \beta \)-hairpin and domain 1b, involving amino acids Lys\(^{111}\) bound to Glu\(^{307}\) and Glu\(^{99}\) bound to Arg\(^{289}\) or Arg\(^{289}\), are important but not essential for the function of UvrB.

Nucleotide excision repair (NER)\(^1\) is a universal and highly conserved DNA repair pathway found in bacteria and eukaryotic cells. NER is unique because of its broad substrate specificity indicating that the chemical nature of the damage is not solely responsible for damage recognition. Rather a local change in the conformation of the DNA itself resulting from the damage may be required. Damage recognition during NER in bacteria is a multistep process accomplished by the UvrA and UvrB proteins. Recently solved crystal structures of the thermophilic UvrB proteins (1–3) contributed to our understanding of how UvrB may recognize and process DNA lesions.

The \( \beta \)-hairpin motif of UvrB, which extrudes from domain 1a and contacts domain 1b, is highly conserved in all bacterial species and absolutely required in the process of damage recognition during NER (4). In our padlock binding model for the UvrB-DNA preincision complex, we have proposed that the \( \beta \)-hairpin is inserted between the two DNA strands, and the non-damaged strand is locked between the \( \beta \)-hairpin and domain 1b (3). It is believed that the \( \beta \)-hairpin is held in place with respect to domain 1b by two salt bridges and hydrophobic interactions at both the base and the tip of the hairpin (4, 5).

The role of the \( \beta \)-hairpin in the damage recognition process has been studied by generating the \( \beta \)-hairpin deletion and several point mutants and analyzing them using functional assays. We have shown that the \( \beta \)-hairpin deletion mutant (\( \Delta \)hb) of UvrB still binds to UvrA, forms the UvrAB-DNA complex, and has significantly higher ATPase activity compared with WT UvrB. However, the \( \Delta \)hb mutant does not have any strand-separating activity, is not loaded onto the site of the damage, and consequently, does not support incision (4).

Goosen and colleagues (5) have probed the role of hydrophobic residues in the \( \beta \)-hairpin of *E. coli* UvrB through a series of double mutants to show that (a) Tyr\(^{92}\)/Tyr\(^{93}\) function in damage recognition by preventing UvrB binding to non-damaged sites, (b) Tyr\(^{96}\)/Tyr\(^{99}\) at the base of \( \beta \)-hairpin have a direct role in damage recognition and are positioned in the vicinity of the lesion in the UvrB-DNA complex, and (c) Tyr\(^{103}\) and Phe\(^{189}\) in the tip of \( \beta \)-hairpin are important for the strand-separating activity of UvrB. More recently, Zou and co-workers (6) have also demonstrated an important role of Tyr\(^{95}\) in damage recognition. In this work, we further examined the role of amino acid residues in UvrB, which we predicted to be important in the process of damage recognition, namely in the interaction of UvrB with both the damaged and non-damaged DNA strands, and in the formation of salt bridges between the \( \beta \)-hairpin and domain 1b.

**MATERIALS AND METHODS**

**Enzymes**—UvrA and UvrB proteins from *Bacillus caldotenax* and UvrC protein from *Thermotoga maritima* were purified by standard procedures (NEB IMPACT™ T7 system manual) (4). T4 polynucleotide kinase was purchased from Invitrogen. Pfu-Ultra DNA polymerase was purchased from Stratagene.

**Construction of Mutant UvrB**—The construction of single amino acid substitution mutants of UvrB was performed by PCR (QuikChange™ Site-Directed Mutagenesis Kit, Stratagene) using UCA18uvrB\(^{–}\) as the template DNA, sense and antisense mutagenic oligonucleotides specific for each mutant as PCR primers, and Pfu-Ultra DNA polymerase. The

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* The abbreviations used are: NER, nucleotide excision repair; ds, double-stranded; ΔH, delta \( \beta \) hairpin (UvrB lacking amino acids 97–112); WT, wild type; PIC, preincision complex.
DNA Damage Processing by UvrB

DNA Substrates—Sigma Genosys synthesized the fluorescein-containing DNA substrates. The DNA sequence of a 50-bp dsDNA substrate (M13F26-M13mp19) is shown in Fig. 2. The fluorescein-containing 50-base oligonucleotide was a generous gift from Nora Goosen (University of Leiden, the Netherlands) and was prepared as described in Ref. 4 for the F<sub>50</sub>-50 dsDNA.

The DNA sequence of the helicase substrate (M13F26-M13mp19) is shown in Fig. 2B. Five pmol of a 26-mer containing an internal fluorescein adduct (M13F26) were labeled at the 5′ terminus under the same conditions as the F<sub>50</sub>-50 top strand. The helicase substrate was constructed by hybridizing 0.4 pmol of 5′-labeled M13F26 oligonucleotide with equimolar amounts of M13mp19(+)-strand (Invitrogen).

Electrophoretic Mobility Shift Assay—Binding reactions were performed with 20 mM B. caldotenax UvrA and 60 mM B. caldotenax UvrB in 20 μl of UvrABC buffer (50 mM Tris-HCl (pH 7.5), 10 mM MgCl<sub>2</sub>, 50 mM KCl, 1 mM ATP, 5 mM dithiothreitol) for 20 min at 55 °C. Glycerol was then added to the reaction (8% v/v), and the reaction mixture was loaded onto a 4% native polyacrylamide gel (80:1). Both the gel and the Tris borate EDTA buffer (TBE) running buffer (89 mM Tris, 89 mM boric acid, 2.5 mM EDTA) contained 1 mM ATP and 10 mM MgCl<sub>2</sub>. Electrophoresis was performed for 3 h at 100 V at room temperature. The gel was dried and exposed to a Storage Phosphor Screen (Molecular Dynamics) overnight at room temperature.

UvrABC Incision and Oligonucleotide-releasing Assay—These assays were performed as described previously (4).

ATP Hydrolysis Assay—This assay was done as described previously (7), with minor modifications. ATP (1 mM) was added to the reaction to initiate the assay.

RESULTS

Incision of the Fluorescein- or Cholesterol-containing 50-mer dsDNA Substrate—We have shown previously that the β-hairpin motif in UvrB is essential for UvrC-mediated incision (4). In this study, we delved further into the role of the β-hairpin by analyzing 13 mutations strategically incorporated in and around this region of UvrB. We first investigated the effect of the individual UvrB mutations on the incision efficiencies of two defined DNA substrates, namely the fluorescein-(F<sub>50</sub>-50) and cholesterol-(Ch<sub>50</sub>-50) containing 50-mer dsDNA with radioactively labeled 5′ termini. The results are shown in Fig. 3, A and B, respectively. Incision efficiencies are expressed relative to WT UvrABC and are summarized in Fig. 3C. Based on the results of the oligonucleotide incision assay, we divided the mutants into three categories according to the severity of their phenotype (i.e., ability to affect UvrC-mediated incision of a damaged DNA substrate).

Category I mutants are defined as those that exhibit the most severe phenotype-failure to support UvrC-mediated incision of either substrate tested. UvrB mutants Y96A, E99A, and R123A fall into this category (Fig. 3, A and B, lanes 5, 9, and 6, respectively). Alanine acid Tyr<sup>367</sup> is 100% conserved among all known UvrB proteins. According to our model, it is located at the base of the β-hairpin facing the damaged DNA strand and is the last amino acid residue of our β-hairpin deletion mutant (Δβh UvrB-deletion of residues 97–112) that did not support incision of the F<sub>50</sub>-50 dsDNA substrate.

Both Glu<sup>99</sup> and Arg<sup>123</sup> are also 100% conserved. Glu<sup>99</sup> is located on the side of β-hairpin where it forms contacts with the domain 1b (Arg<sup>123</sup> is not involved in contacts to domain 1b.) The WT UvrB structure indicated the formation of a salt bridge between Glu<sup>99</sup> and Arg<sup>367</sup>, thus bridging the β-hairpin to domain 1b. Surprisingly, the R367A mutation had a moderate effect on incision efficiency (Fig. 3, A and B, lane 13), suggesting that Glu<sup>99</sup> might have an additional function such as the interaction with the DNA substrate rather than just the formation of a salt bridge with Arg<sup>367</sup>. Arg<sup>123</sup>, which is still present in our Δβh mutant, is located at the very base of the β-hairpin and seems to have an essential role in the interaction of UvrB with the DNA in the UvrB-DNA preincision complex, as an alanine substitution results in extremely low, if any, incision of either site-specific substrate (Fig. 3, A and B, lane 6).

Category II mutants are those with a relatively mild phenotype, reaching only ~50–80% of wild-type UvrABC endonuclease activity. This reduced activity was observed for the following mutations: Y92A, Y93A, R289A/R367A, R367A, E307A, and F527A (Fig. 3, A and B, lanes 3, 4, and 12–15, respectively). The WT UvrB model had shown that Lys<sup>111</sup> forms a salt bridge with Glu<sup>307</sup>. We also hypothesized that His<sup>124</sup> and Phe<sup>249</sup> might be involved in DNA binding.

Loading of UvrB Mutants onto the Site of the Lesion—The ability of UvrB to efficiently recognize DNA damage and present it to the UvrC subunit is critically important in the overall reaction mechanism for the UvrABC system. To test whether these mutant UvrB proteins are loaded onto the site of the damage, i.e., whether they form a stable UvrB-DNA preincision complex, we used the electrophoretic mobility shift assay. The results are shown in Fig. 4. We used a cholesterol-containing 50-mer dsDNA labeled with <sup>32</sup>P at the 5′-end of the damaged strand as the DNA substrate. The results of the electrophoretic mobility shift assay correlate well with the incision data obtained for mutants in category I. Those that failed to promote an incision of the defined substrates, such as mutants Y96A, R123A, and E99A, did not form a stable UvrB-DNA PIC (Fig. 4, lanes 5, 6, and 9, respectively).

Although many of the category II mutants displayed near WT levels of UvrB-DNA complex formation, a few showed remarkable diversity in the quantity of stable UvrB-DNA complex. This is especially evident when comparing mutants Y92A and Y93A (Fig. 4, lanes 3 and 4, respectively). The Y92A mutant reaches UvrB-DNA complex formation levels close to WT UvrB, whereas Y93A is significantly lower. It is interesting to note that despite the difference in UvrB-DNA complex formation, the incision activities of these two mutants do not differ as greatly (82 and 68% relative to wild-type UvrB, respectively). Furthermore, although the F527A mutant is slightly deficient in incision, it appears to be loaded onto damaged DNA better than WT.
All category III mutants (no phenotype in incision assay), i.e. K111A, H124A, and F249A, are proficient in UvrB-DNA complex formation. Each promotes the formation of the UvrB-DNA complex as well as WT UvrB (Fig. 4, lanes 7, and 8, respectively).

**Strand-destabilizing Activity of UvrB Mutants**—A limited strand opening around the lesion by the UvrAB complex has previously been shown to be important for dynamic recognition of damaged DNA (8, 9). To examine the effect of mutations in UvrB on this limited helix-destabilizing activity of the UvrAB complex, we evaluated these mutants in the strand-destabilizing assay (4). This assay measures the release of a radioactively labeled 26-mer oligonucleotide containing a site-specific fluorescein from M13mp19(+) single-stranded circular DNA. These results are shown in Fig. 5A, and the quantitation is summarized in Fig. 5B.

Category I mutants Y96A, R123A, and E99A exhibit defects in strand-destabilizing activity (Fig. 5A, lanes 8, 10, and 16, respectively) similar to those seen with the Δ3β UvrB mutant (4). Among the category II mutants, it is interesting that mutant Y93A, which showed only a mild reduction in incision efficiency and a very significant decrease in UvrB-DNA complex formation, also exhibited a significant loss of strand-destabilizing activity compared with WT UvrB (Fig. 5A, lane 2 versus 6). In addition, mutants R289A/R367A and E307A (Fig. 5A, lanes 24 and 20, respectively) exhibited a more detrimental effect on the strand-destabilizing activity of UvrB than observed for either UvrB-DNA complex formation or incision. Again it is interesting to note that F527A displays 100% of WT activity in this assay (Fig. 5A, lane 28). The category III mutants, H124A, F249A, and K111A show strand-destabilizing activity comparable with that of WT UvrB (Fig. 5A, lanes 12, 14, and 18, respectively. versus lane 2).

**ATPase Activity of Selected UvrB Mutants**—It has been shown previously that ATP binding and hydrolysis are absolutely required for NER (10, 11). However, the precise step(s) during damage processing that requires ATP binding and or hydrolysis remains unclear. The situation is further complicated by the fact that UvrA possesses two ABC ATPase domains, is an ATPase, and UvrB has cryptic ATPase activity, which is activated in the presence of UvrA and damaged DNA. We have proposed that the free energy available either from ATP hydrolysis or UvrA-UvrB complex formation might be crucial for a conformational change in UvrB prior to the binding of DNA (4, 12). According to our padlock model, a conformational change of the β-hairpin transforms UvrB from a relatively poor DNA-binding protein into a highly specific damaged DNA-binding/recognition protein. To test whether the altered DNA binding properties of selected mutants are because of an altered ATPase activity, we examined their ATPase activity in the presence or absence of UV light-irradiated DNA alone (Fig. 6A) or in combination with UvrA (Fig. 6B). These data suggest that the ATPase activity of UvrB and the incision activity are not well correlated as seen also in the β-hairpin deletion mutant (4), which displayed no incision activity but elevated ATPase activity (4, 7). Y96A showed a similar phenotype with very little incision activity but almost full ATPase activity (Fig. 6B). The other category I mutants, R123A and E99A, showed 43 and 72% of the WT UvrB ATPase activity in the presence of UvrA and damaged DNA. For the category II mutations, Y92A displayed 20% more activity than WT UvrB, whereas the other mutants in this category showed a 20–50% reduction in UvrA/DNA damage-stimulated ATPase activity.

The category III mutants, H124A, F249A, and K111A, had variable ATPase activities ranging from 46% (K111A) to 92% (H124A) of the WT UvrB activity.

**DISCUSSION**

The aim of this work was to identify residues within and adjacent to the β-hairpin that might be directly involved in binding the damaged and non-damaged DNA strands during NER. We also tested the importance of the salt bridges that are formed between the β-hairpin motif and domain 1b; Glu99 forms a salt bridge to Arg267 or Arg258, and Lys911 forms the second salt bridge to Glu307 (Fig. 1) as seen in the WT UvrB structure (3).

The first group of residues we studied were the highly conserved tyrosines at positions 92, 93, and 96. Tyr96 is absolutely conserved among bacterial UvrB species, and the Y96A UvrB mutant displayed a defect in incision, failed to form a stable UvrB-DNA preincision complex, and had a very low level of strand-destabilizing activity. These data strongly support the results obtained for the Y95A/Y96A double mutant reported previously (5). Together, the data lead to the conclusion that Tyr96 plays a direct role in damage-specific DNA binding by UvrB. Furthermore, Zou and co-workers (6) prepared the Y95W mutant of UvrB and showed that it retained full activity, suggesting a less important role for Y95. Moolenaar et al. (5) suggested two mechanisms whereby UvrB might accomplish its task of damage recognition. First, they suggested that a clash between a non-damaged nucleotide and Tyr92 and/or...
Tyr93 prevents the binding of UvrB to a non-damaged DNA site. Secondly, they proposed that the damaged nucleotide would be flipped out of the DNA helix, allowing Tyr92 and/or Tyr93 to occupy the vacated space. Our data suggest that the Tyr92 and Tyr93 might be functionally redundant and that the presence of one is sufficient for proper damage-dependent activity, in which case Tyr92 or Tyr93 could slip into the vacated base position. However, from the phenotype of the single mutants it is clear that Tyr93 has a more important role in DNA recognition than Tyr92, because the Y93A mutant is reproducibly more impaired in all the functional assays as compared with the Y92A mutant.

Two amino acid residues at the base of β-hairpin, Arg123 and His124, and one amino acid located on domain 2, Phe249, were mutated as well. All of these residues are on the side of UvrB that is proposed to interact with the non-damaged DNA strand. The R123A mutant exhibited a very severe defect in incision, preincision complex formation, and strand-destabilizing activity (category I). This would suggest that Arg123 is absolutely required for UvrB DNA binding and that it either directly

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**Fig. 3. Incision of a lesion-containing 50-bp duplex.** A 50-bp duplex (2 nM), containing either fluorescein or cholesterol (sequences of both substrates shown in Fig. 2), both with a 5’ terminally labeled modified strand, was incubated with 20 nM *B. caldotenax* UvrA, 60 nM *B. caldotenax* UvrB, and 50 nM *T. maritima* UvrC at 55 °C for 1 h. The samples were analyzed by polyacrylamide gel electrophoresis under denaturing conditions. A, incision of F26-50 ds substrate; B, incision of Ch2650 dsDNA substrate; C, graphic comparison of incision of F26-50 and Ch2650 dsDNA substrates.
contacts the DNA or promotes structural changes that stabilize DNA binding. On the other hand, the H124A and F249A mutants exhibit WT-like phenotypes.

Mutants suspected to disrupt salt bridge formation exhibited phenotypes ranging from severe to no effect. However, the majority of salt bridge mutants examined in this study display a mild phenotype. These mutated amino acid residues, R367A, R289A/R367A, and E307A, are all located on domain 1b oppo-
FIG. 7. Hypothetical model for UvrB-mediated DNA processing. Using the convention first developed for monomeric helicases by Wigley and co-workers (13), the bases along the damaged DNA strand are shown in green and the adducted base is shown as a red hexagon. The black bar across the DNA strand indicates that the point of interaction between Phe527 (F527), and the DNA is several bases downstream (more 3') of the incision site. We have depicted, in various colors, the UvrB amino acid side chains that are thought to be important for DNA damage recognition and processing (5, 6, 14). A, UvrB has engaged the DNA adduct (red) using Tyr95 and Tyr96 (Y95/96). These residues either stack into the double helix displacing the damaged base or stabilize the displaced damaged base (5). B, the domain 3 motion of UvrB, possibly linked to ATP binding, pushes Phe527, and/or another yet to be identified residue into the double helix causing the damaged strand to be pushed up into the 3'-nuclease center of UvrC leading to incision. Arg123 (R123) and Glu99 (E99) are proposed to hold the DNA in place through attractive and repulsive forces, respectively. See "Discussion" for additional details.
site the β-hairpin. Alternatively, salt bridge mutations localized within the β-hairpin belong to the other two categories. For example, the β-intermediate mutant E99A had the most severe phenotype as it was shown to be defective in all functional assays (incision, loading onto the site of the damage, strand-destabilizing activity). Although Glu99 is thought to be essential for proper UvrB function, mutations of its proposed salt bridge partners, Arg367 or Arg289 to alanines, showed only a mild phenotype. This suggests that the salt bridge between Glu99 and Arg367 (or Arg289) may not be as important as we predicted, or Glu99 may form an alternative salt bridge or serve a different purpose altogether. The formation of salt bridges between the β-hairpin and domain 1b, between amino acids Lys111 and Glu307 and between Glu99 and Arg367 (or Arg289), might be redundant such that the crippling of one can be offset by the activity of the others. It is also possible that Glu99 might play another role in damage processing other than formation of a salt bridge.

The final mutant evaluated was Phe527, located in domain 3 (close to domain 1a). We chose to mutate this residue because of its high degree of conservation with monomeric helicases (13) and its potential to intercalate between the DNA bases. The homologous amino acid in PcrA, Phe626, has been shown to intercalate into DNA and is proposed to be involved in binding and releasing DNA bases in response to ATP hydrolysis (13). We hypothesized that Phe527 of UvrB might be involved in coupling domain 3 motions with the movement of DNA across the DNA binding surface of UvrB to properly align the DNA damage with the damage recognition elements of UvrB, i.e. Tyr96. We discovered that the F527A protein is more proficient than WT UvrB at generating stable UvrB-DNA complexes yet possesses reduced incision activity; therefore we propose that it is defective in the presentation of DNA to UvrC.

A hypothetical model for the roles of Tyr92, Tyr93, Tyr95, Tyr96, Glu99, Arg123, and Phe527 in UvrB-mediated DNA damage processing is shown in Fig. 7. UvrB is believed to first lock the non-damaged strand (14) into place by the interactions between the β-hairpin and domain 1b (see Fig. 1). Following this initial step, UvrB grasps the damaged strand by making intimate contacts between the adducted base and Tyr95 and Tyr96 (Fig. 7A). Once the damaged strand has been fully engaged, it is believed that UvrB uses its helicase fold to close domain 3 down on the damaged strand, possibly through ATP binding. This domain motion uses Phe527, and most likely another yet to be identified amino acid, to force the damaged strand up into the 3′-nuclease active center so that UvrC can cleave the phosphodiester backbone (Fig. 7B). Once incision has occurred, UvrB hydrolizes ATP to return domain 3 to its original position. This model explains why the β-hairpin deletion mutant hydrolizes more ATP as compared with the WT UvrB (4, 7). Each round of ATP binding mediates domain 3 closure and is met with DNA strand slippage away from the deleted β-hairpin, which still maintains the aromatic bases at the stump but loses its ability to lock down the non-damaged strand.

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