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# Effects of the *ssb-1* and *ssb-113* Mutations on Survival and DNA Repair in UV-Irradiated Δ*uvrB* strains of *Escherichia coli* K-12

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The molecular defect in DNA repair caused by *ssb* mutations (single-strand binding protein) was studied by analyzing DNA synthesis and DNA double-strand break production in UV-irradiated *Escherichia coli* Δ*uvrB* strains. The presence of the *ssb-113* mutation produced a large inhibition of DNA synthesis and led to the formation of double-strand breaks, whereas the *ssb-1* mutation produced much less inhibition of DNA synthesis and fewer double-strand breaks. We suggest that the single-strand binding protein plays an important role in the replication of damaged DNA, and that it functions by protecting single-stranded parental DNA opposite daughter-strand gaps from nuclease attack.

The *Escherichia coli* DNA single-strand binding (SSB) protein is coded by the *ssb* gene and is essential for DNA replication in vivo (6, 15) and in vitro (20, 28, 30). Two mutations affecting the SSB protein, *ssb-1* (15, 16) and *ssb-113* (previously known as *exrB* [7, 8] and *lexC113* [11]), have been described. They map very close to each other and are considered to be allelic (6). Strains containing either of these *ssb* mutations are temperature sensitive for DNA replication and they are UV radiation sensitive (6, 8, 15, 26). Therefore, in addition to its role in DNA replication, the SSB protein must also play a role in the repair of UV radiation-damaged DNA.

Two major "dark repair" systems have been described in *E. coli* for the repair of UV radiation-damaged DNA: excision repair and postreplication repair (9). Since the *uvrA* and *uvrB* strains of *E. coli* are deficient in excision repair (10, 21, 22), the major dark repair system operating in these strains is postreplication repair. Recent studies on the UV radiation survival of *ssb* mutants suggest that the SSB protein plays a role in recombination repair (i.e., postreplication repair), but not in excision repair (14, 29).

In this paper we examine the effect of *ssb* mutations on DNA synthesis and DNA double-strand break (DSB) formation in UV-irradiated *uvrB* cells. Our results suggest that the major role of the SSB protein in the repair of UV radiation-damaged DNA is to protect single-stranded parental DNA opposite daughter-strand gaps from nuclease attack.

## MATERIALS AND METHODS

**Bacterial strains.** The bacterial strains used in this work are listed in Table 1. The transduction technique used was similar to that of Miller (17). The cotransfer of the *ssb* mutation was identified by the failure of $Mal^+$ transductants to grow at 42°C.

**Media.** The media used for the growth of cells were either LB medium (10 g of Difco tryptone, 5 g of Difco yeast extract, and 10 g of NaCl per liter of water) or glucose-salts minimal medium (MM) (5) supplemented with 0.5 μg of thiamine-hydrochloride per ml and, when necessary, thymine at 10 μg/ml and L-amino acids at 1 mM. YENB agar (7.5 g of Difco yeast extract and 23 g of Difco nutrient agar per liter of water) and supplemented MM (SMM) agar (SMM solidified with 1.6% Difco Noble agar) were used to determine colony-forming units. DTM buffer, which is MM without glucose, was used for washing and resuspending cells. Cells were grown at 30°C in a shaking water bath.

**Irradiation.** UV irradiation (254 nm) and survival determination were performed as previously described (27). The stated UV radiation fluences have been corrected for cell-masking effects (31).

**DNA synthesis.** DNA synthesis was measured by the net incorporation of [$^3$H]thymine into DNA (23, 24). Cultures were grown at 30°C for at least four generations in SMM containing thymine at 2 μg/ml and [*methyl*-$^3$H]thymine (30 Ci/mmol; Amersham Corp.) at 2 μCi/ml. After a density of $10^8$ cells per ml was reached (optical density at 650 nm, 0.1; Zeiss PMQII spectrophotometer), the cells were UV irradiated while in the medium. After irradiation, the cells were again incubated at 30°C, and duplicate 0.2-ml samples were removed at intervals to test tubes containing 0.5 ml of ice-cold 10% trichloroacetic acid. The trichloroacetic acid precipitates were collected on membrane

TABLE 1. *E. coli* strains used in this work

| Stanford radiology no. | Genotype[a] | Source, derivation, or reference |
|---|---|---|
| SR248 | *leuB19 metE70 bioA2 thyA36 deo(C2?) lacZ53 malB45 rha-5 rpsL151* $Su^-$ | R. B. Helling (KH21) |
| SR839 | Δ(*uvrB-chlA*) *recA56 leuB19 thyA36 deo(C2?) lacZ53 malB45 rha-5 rpsL151 srlA300*::Tn*10*($Tc^r$)[b] | M.-s. Tang |
| SR896 | Δ(*uvrB-chlA*) *leuB19 thyA36 deo(C2?) lacZ53 malB45 rha-5 rpsL151* | (27) |
| SR963 | *ssb-113 lon sulA* | B. F. Johnson (PAM29) |
| SR978 | Δ(*uvrB-chlA*) *leuB19 thyA36 deo(C2?) lacZ53 rha-5 rpsL151* | P1::Tn*9*cts·SR963 × SR896 (select $Mal^+$)[c] |
| SR979 | Δ(*uvrB-chlA*) *ssb-113 leuB19 thyA36 deo(C2?) lacZ53 rha-5 rpsL151* | Same as SR978 |
| SR995 | *ssb-1 thy mel rha* | J. W. Chase (KLC436) |
| SR1006 | Δ(*uvrB-chlA*) *leuB19 thyA36 deo(C2?) lacZ53 rha-5 rpsL151* | P1::Tn*9*cts·SR995 × SR896 (select $Mal^+$) |
| SR1007 | Δ(*uvrB-chlA*) *ssb-1 leuB19 thyA36 deo(C2?) lacZ53 rha-5 rpsL151* | Same as SR1006 |

[a] Genotype symbols are those used by Bachmann and Low (1). Strain SR963 is a derivative of *E. coli* B/r; all the remaining strains are derivatives of *E. coli* K-12 and are $F^-$ and $\lambda^-$.

[b] $Tc^r$, Tetracycline resistance.

[c] $Mal^+$, cells can utilize maltose in place of glucose.

filters (Millipore Corp.) and prepared for the assay of radioactivity as previously described (25). Cells irradiated in SMM containing 2 μg of thymine per ml had the same survival curve as those irradiated in DTM buffer (data not shown). Therefore, no additional correction was made for the stated UV radiation fluence for the presence of the growth medium.

**DNA degradation.** DNA degradation was measured by the release of trichloroacetic acid-soluble radioactivity from DNA labeled as above. After reaching a density of about $1 \times 10^8$ to $2 \times 10^8$ cells per ml, the cells were harvested on Millipore HA membrane filters (0.45-μm pore size), washed with DTM, suspended at an optical density at 650 nm of 0.05 in SMM containing 2 μg of thymine per ml, and incubated at 30°C for 2 h before being irradiated and treated as described above for DNA synthesis.

**DNA DSB.** The neutral sucrose gradient method for determining DNA DSB was that of Tang and Smith (25). After centrifugation, the bottom of each tube was pierced, and about 30 fractions of equal volume were pumped onto Whatman no. 17 paper strips, which were then washed twice in cold 5% trichloroacetic acid and once each in 95% ethanol and acetone. After drying, the strips were cut into individual fractions, placed in vials, and assayed for radioactivity (23).

For the analysis of DSB in parental DNA, cells were grown for at least four generations at 30°C in SMM containing thymine at 2 μg/ml and [*methyl*-$^3$H]thymine at 5 μCi/ml. When the culture reached an optical density at 650 nm of 0.1, the cells were harvested by filtration, washed with DTM, and suspended at an optical density at 650 nm of 0.075 in SMM containing 2 μg of thymine per ml. The resuspended cells were incubated at 30°C for 45 min before being UV irradiated in the medium. After different times of postirradiation incubation at 30°C, 0.2-ml samples were treated with lysozyme and layered onto 4.8-ml neutral sucrose gradients (5 to 20%, wt/vol) as previously described (25).

For the analysis of DSB in DNA replicated after UV irradiation, cells were first prelabeled as described above, except that 2 μCi of [2-$^{14}$C]thymine (54.5 Ci/mol; New England Nuclear Corp.) per ml was used. After irradiation, [*methyl*-$^3$H]thymidine (41 Ci/mmol; Amersham) was added to 1-ml samples at 20 μCi/ml either immediately after UV irradiation or after different times of post-irradiation incubation at 30°C. After 15 min of incubation with [$^3$H]thymidine, the cells were harvested by filtration, washed three times with DTM, and suspended in 0.7 ml of SMM containing thymine at 2 μg/ml. A 0.2-ml sample of cell suspension, with or without further incubation, was treated with lysozyme and layered on a neutral sucrose gradient as described above.

## RESULTS

**Effects of the *ssb* mutations on the UV radiation sensitivity of *uvrB* cells.** The *uvrB* strain was further sensitized to UV radiation by the presence of *ssb* mutations (Fig. 1A). The *uvrB ssb-113* strain was substantially more UV radiation sensitive than the *uvrB ssb-1* strain (Fig. 1A), in agreement with the data of Whittier and Chase (29) on similar strains. It should be noted that the UV radiation sensitivity of the *uvrB ssb-113* strain approached that of the *uvrB recA56* strain (Fig. 1B), which is believed to be deficient in all known dark repair processes for UV radiation-damaged DNA.

**Effects of the *ssb* mutations on DNA synthesis and degradation in *uvrB* cells.** Figure 2 illustrates the effect of UV radiation on the net thymine

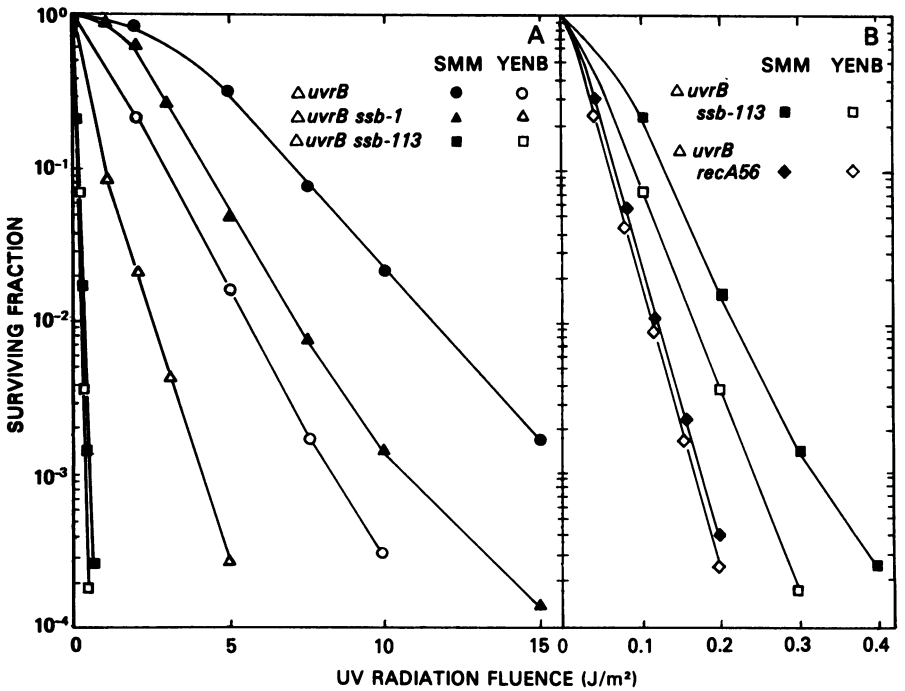


FIG. 1. Effect of the *ssb-1* and *ssb-113* mutations on the UV radiation survival of Δ*uvrB* cells of *E. coli* K-12 on YENB agar (△, ○, □, ◇) and SMM agar (▲, ●, ■, ◆). All points are the averages of at least two experiments. A, Symbols: Δ*uvrB* (SR1006) (○, ●); Δ*uvrB ssb-1* (SR1007) (△, ▲); and Δ*uvrB ssb-113* (SR979) (□, ■). The survival curve of strain SR978 (Δ*uvrB*), a cotransductant for strain SR979, is identical to strain SR1006 (data not shown). B, Symbols: Δ*uvrB ssb-113* (SR979) (□, ■); Δ*uvrB recA56* (SR839) (◇, ◆).

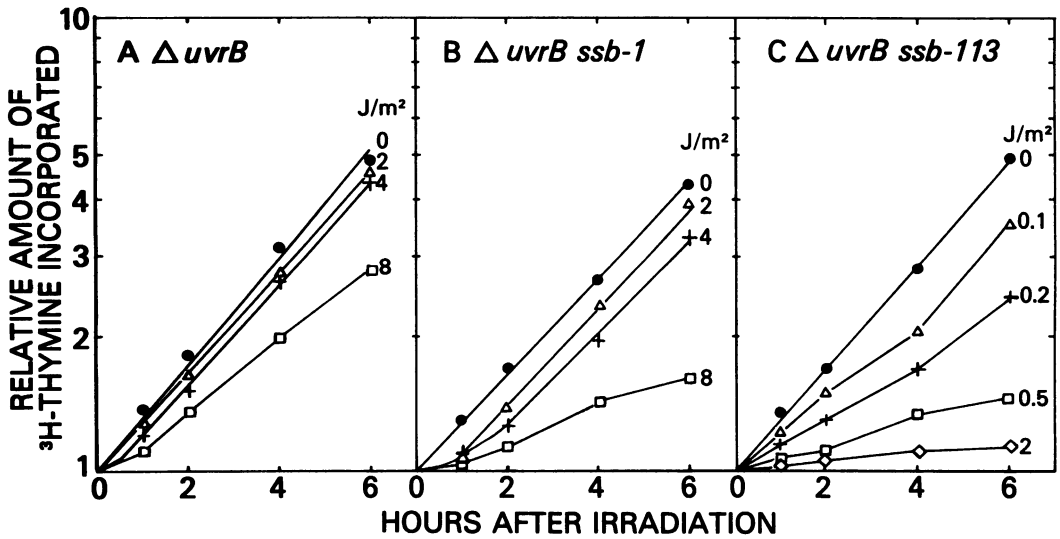


FIG. 2. Effect of UV radiation on net DNA synthesis in Δ*uvrB*, Δ*uvrB ssb-1*, and Δ*uvrB ssb-113* cells. Cultures were grown at 30°C and treated as described in the text. A, Δ*uvrB* (SR978); B, Δ*uvrB ssb-1* (SR1007); C, Δ*uvrB ssb-113* (SR979). The thymine incorporation data for strain SR978 are indistinguishable from those for strain SR1006 (data not shown).

TABLE 2. DNA degradation in UV-irradiated Δ*uvrB* cells of *E. coli* K-12

| Strain | UV radiation fluence (J/m²) | Relative amt of [³H]thymine in acid-insoluble fraction of DNA | | | |
|---|---|---|---|---|---|
| | | 0 h | 1 h | 2 h | 4 h |
| Δ*uvrB* (SR978) | 0 | 1 | —[a] | 0.96 (0.98)[b] | 0.95 (0.99) |
| | 4 | 1 | 0.92 (0.98) | 0.98 (0.97) | 0.96 (0.92) |
| | 8 | 1 | 0.99 (0.91) | 1.12 (0.94) | 1.07 (0.84) |
| Δ*uvrB ssb-1* (SR1007) | 0 | 1 | 0.99 | 0.93 | 0.98 |
| | 4 | 1 | 0.95 | 1.01 | 0.92 |
| | 8 | 1 | 0.79 | 0.98 | 0.93 |
| Δ*uvrB ssb-113* (SR979) | 0 | 1 | 0.97 | 0.98 | 0.95 |
| | 0.2 | 1 | 1 | 0.98 | 0.92 |
| | 0.4 | 1 | 0.93 | 0.89 | 0.83 |
| | 2 | 1 | — | 0.94 | 0.87 |

[a] —, Not determined.
[b] The data within parentheses are for Δ*uvrB* strain SR1006.

incorporation of cells incubated with radioactive thymine for at least four generations before irradiation as well as during and after irradiation. Under these conditions, the content of acid-insoluble radioactivity is a direct measure of the DNA content of the cells (4, 23, 24). The *uvrB* cells showed only a slight, transient effect in the apparent rate of thymine incorporation after 2 or 4 J of UV radiation per m² (Fig. 2A). After 8 J of UV radiation per m² the *uvrB* cells showed a somewhat reduced rate of thymine incorporation. In contrast, the *uvrB ssb-113* cells showed a marked and persistent decrease in the apparent rate of thymine incorporation after 2 J of UV radiation per m² (Fig. 2C). Even after fluences as low as 0.1 or 0.2 J of UV radiation per m², a noticeable reduction in the rate of thymine incorporation was evident. The incorporation of thymine by *uvrB ssb-1* cells was partially inhibited by all UV radiation fluences employed (compare Fig. 2B with 2A).

Since the apparent rate of thymine incorporation reflected both DNA synthesis and degradation, we examined the effects of the *ssb* mutation on UV radiation-induced DNA degradation in *uvrB* cells. Very little, if any, UV radiation-induced DNA degradation was observed in *uvrB*, *uvrB ssb-1*, and *uvrB ssb-113* cells over a 4-h period of postirradiation incubation at 30°C (Table 2). Therefore, the apparent rate of thymine incorporation shown in Fig. 2 reflects mostly DNA synthetic activity. It follows that the *ssb-113* mutation greatly inhibits the ability of *uvrB* cells to synthesize DNA in the presence of UV radiation damage, whereas the *ssb-1* mutation has a much less inhibitory effect on DNA synthesis after UV irradiation.

**DNA DSB.** We tested whether DSB were produced in the DNA of UV-irradiated *uvrB* and *uvrB ssb-113* cells. After 0.5 J of UV radiation per m², there were no detectable DNA DSB produced in *uvrB* cells that were lysed either immediately after irradiation or after 4 h of post-irradiation incubation at 30°C (Fig. 3A). On the other hand, the production of DNA DSB was evident from the DNA sedimentation profile for UV-irradiated *uvrB ssb-113* cells that had been incubated for 4 h after irradiation (Fig. 3B). The production of DNA DSB was also observed for *uvrB ssb-113* cells that had been incubated after exposure to 0.2, 1, or 2 J of UV radiation per m² (data not shown).

If the production of DNA DSB in UV-irradiated *uvrB ssb-113* cells is the result of endonucleolytic attack on the single-stranded parental DNA opposite daughter-strand gaps, one should have a much more sensitive assay for the production of DSB by this mechanism if one looks only at the DNA synthesized after UV irradiation (Fig. 4). For unirradiated cells, the sedimentation profiles of pulse-labeled [³H]DNA were indistinguishable from the profiles of parental [¹⁴C]DNA in the *uvrB*, *uvrB ssb-1*, and *uvrB ssb-113* cultures (Fig. 4A, D, and G). However, in cultures that were pulse-labeled immediately after 2 J of UV radiation per m² (Fig. 4B, E, and H), a larger fraction of [³H]DNA sedimented at lower molecular weights than did the [¹⁴C]DNA, with the *uvrB ssb-113* cells showing the most dramatic difference and the *uvrB* cells showing the least difference. Similar results were also obtained for cultures that were pulse-labeled after 60 min of post-irradiation incubation (Fig. 4C, F, and I).

## DISCUSSION

Since the *ssb* gene appears to play a significant role in the survival of UV irradiated excision repair-deficient cells (Fig. 1), the SSB protein must function either directly or indirectly in postreplication repair. The lesions on which

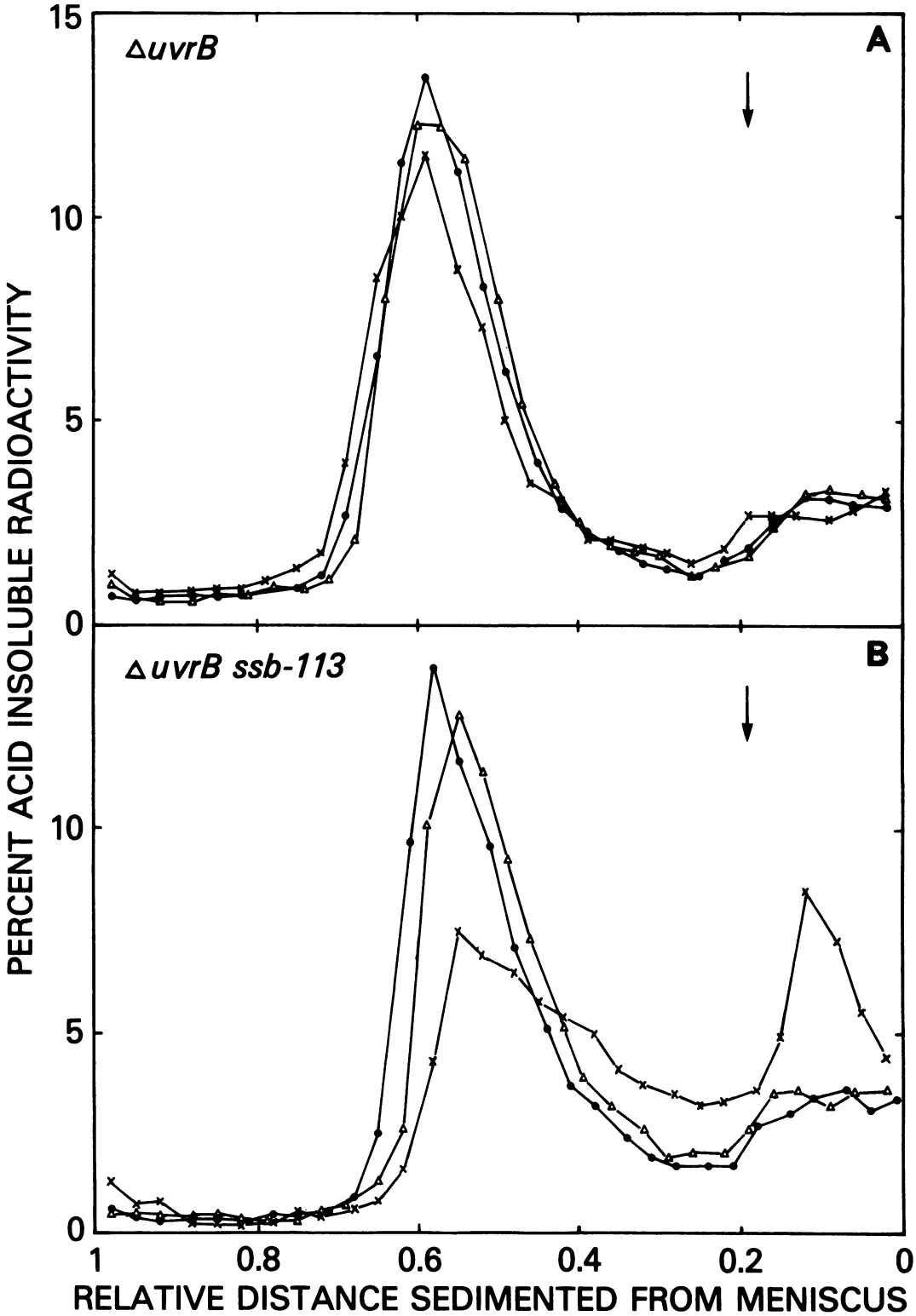


FIG. 3. Neutral sucrose-gradient sedimentation profiles of [$^3$H]thymine-prelabeled DNA from UV-irradiated cells grown at 30°C. A, Δ*uvrB* (SR978); B, Δ*uvrB ssb-113* (SR979). Symbols: ●, unirradiated control; △, 0.5 J/m$^2$, 0-h post-irradiation incubation; ×, 0.5 J/m$^2$, 4-h post-irradiation incubation. Centrifugation was at 3,570 rpm for 40 h. The arrow indicates the position of a bacteriophage T2 DNA marker.

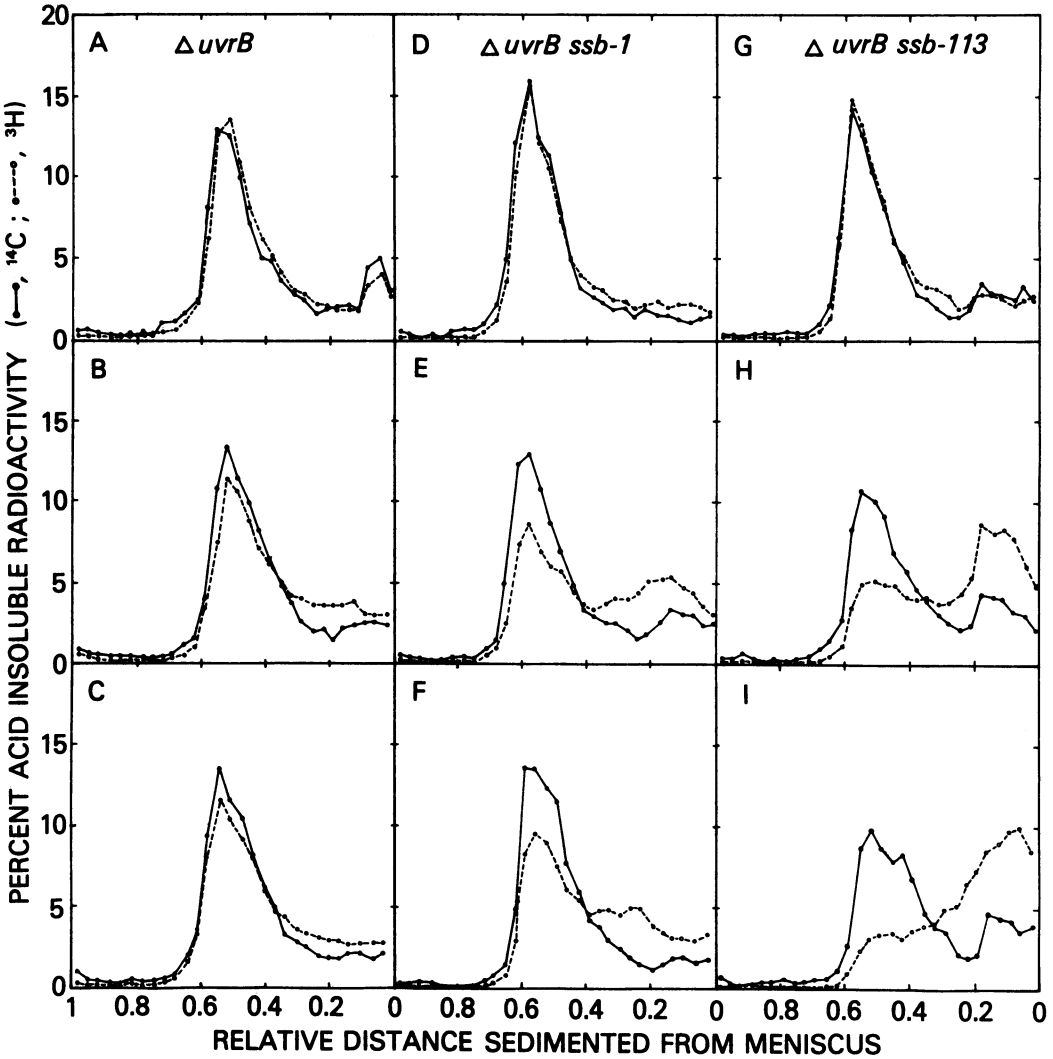


FIG. 4. Neutral sucrose-gradient sedimentation profiles of DNA from [$^{14}$C]thymine-prelabeled cells that were pulse-labeled with [$^{3}$H]thymidine after UV irradiation. Cultures were prelabeled with [$^{14}$C]thymine for at least 4 generations at 30°C. Unirradiated cultures (A, D, and G) and cultures irradiated with 2 J/m$^2$ were pulse-labeled with [$^{3}$H]thymidine for 15 min either immediately after UV irradiation (B, E, and H) or after 60 min of postirradiation incubation (C, F, and I). Experimental details are described in the text. Panels from left to right are cultures of Δ*uvrB* (SR978), Δ*uvrB ssb-1* (SR1007), and Δ*uvrB ssb-113* (SR979), respectively. The precise centrifugation speeds used were 3,300 rpm for SR978, 3,660 rpm for SR1007, and 3,570 rpm for SR979. Symbols: ●, $^{14}$C radioactivity; ○, $^{3}$H radioactivity.

postreplication repair is presumed to act are daughter-strand gaps that arise in newly synthesized DNA after UV irradiation (18, 19). These daughter-strand gaps are opposite single-stranded parental DNA, which may be susceptible to attack by DNA endonucleases. Our data on the production of DNA DSB in UV-irradiated *uvrB ssb* cells (Fig. 3 and 4) suggest that one function of the SSB protein in DNA repair is to protect this single-stranded DNA from nuclease attack so that other repair enzymes can proceed to complete the filling of gaps by recombinational processes. Alternately, the SSB protein may also participate in the repair of these gaps.

Our data on the effect of UV radiation on DNA synthesis show that the *ssb* mutations inhibit the ability of UV-irradiated *uvrB* cells to synthesize DNA (Fig. 2), suggesting that the SSB protein plays a role in the replication of damaged DNA. However, it is not known whether this inhibition of DNA synthesis is due to a poorer efficiency of replicating damaged

DNA in the presence of mutant SSB protein or is due to an alteration in the structure of DNA by the formation of double-strand breaks at unprotected single-strand sites such that the overall rate of DNA synthesis is reduced.

The DSB formed in *uvrB ssb-113* cells in DNA synthesized immediately after UV irradiation were not repaired during further incubation (data not shown), suggesting that either these DSB are nonrepairable under our growth conditions, or that wild-type SSB protein is required directly or indirectly for the repair of these DSB. Krasin and Hutchinson (12, 13) reported that, under certain conditions, DSB produced by ionizing radiation could be repaired; it required the presence of duplicate genomes, the function of the *recA* gene, and the synthesis of proteins that can be induced by UV irradiation. Several recent studies have indicated that the SSB protein is involved in the induction of DNA damage-inducible functions (2, 14, 26, 29). Therefore, the SSB protein may have both a regulatory and mechanistic role in DNA repair.

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## LITERATURE CITED

1. **Bachmann, B. J., and K. B. Low.** 1980. Linkage map of *Escherichia coli* K-12, edition 6. Microbiol. Rev. **44**:1–56.
2. **Baluch, J., J. W. Chase, and R. Sussman.** 1980. Synthesis of *recA* protein and induction of bacteriophage lambda in single-strand deoxyribonucleic acid-binding protein mutants of *Escherichia coli*. J. Bacteriol. **144**:489–498.
3. **Bonura, T., and K. C. Smith.** 1975. Enzymatic production of deoxyribonucleic acid double-strand breaks after ultraviolet irradiation of *Escherichia coli* K-12. J. Bacteriol. **121**:511–517.
4. **Doudney, C. O.** 1971. Deoxyribonucleic acid replication in UV-damaged bacteria revisited. Mutat. Res. **12**:121–128.
5. **Ganesan, A. K., and K. C. Smith.** 1968. Dark recovery processes in *Escherichia coli* irradiated with ultraviolet light. I. Effect of *rec*$^-$ mutations on liquid holding recovery. J. Bacteriol. **96**:365–373.
6. **Glassberg, J., R. R. Meyer, and A. Kornberg.** 1979. Mutant single-strand binding protein of *Escherichia coli*: genetic and physiological characterization. J. Bacteriol. **140**:14–19.
7. **Greenberg, J., L. J. Berends, J. Donch, and M. H. L. Green.** 1974. *exrB*: a *malB*-linked gene in *Escherichia coli* B involved in sensitivity to radiation and filament formation. Genet. Res. **23**:175–184.
8. **Greenberg, J., L. Berends, J. Donch, and B. Johnson.** 1975. Reversion studies with *exrB* in *Escherichia coli*. Genet. Res. **25**:109–117.
9. **Howard-Flanders, P.** 1968. DNA repair. Annu. Rev. Biochem. **37**:175–200.
10. **Howard-Flanders, P., R. P. Boyce, and L. Theriot.** 1966. Three loci in *Escherichia coli* that control the excision of pyrimidine dimers and certain other mutagen products from DNA. Genetics **53**:1119–1136.
11. **Johnson, B. F.** 1977. Genetic mapping of the *lexC-113* mutation. Mol. Gen. Genet. **157**:91–97.
12. **Krasin, F., and F. Hutchinson.** 1977. Repair of DNA double-strand breaks in *Escherichia coli*, which requires *recA* function and the presence of a duplicate genome. J. Mol. Biol. **116**:81–98.
13. **Krasin, F., and F. Hutchinson.** 1981. Repair of DNA double-strand breaks in *Escherichia coli* cells requires synthesis of proteins that can be induced by UV light. Proc. Natl. Acad. Sci. U.S.A. **78**:3450–3453.
14. **Lieberman, H. B., and E. M. Witkin.** 1981. Variable expression of the *ssb-1* allele in different strains of *Escherichia coli* K-12 and B: differential suppression of its effects on DNA replication, DNA repair and ultraviolet mutagenesis. Mol. Gen. Genet. **183**:348–355.
15. **Meyer, R. R., J. Glassberg, and A. Kornberg.** 1979. An *Escherichia coli* mutant defective in single-strand binding protein is defective in DNA replication. Proc. Natl. Acad. Sci. U.S.A. **76**:1702–1705.
16. **Meyer, R. R., J. Glassberg, J. V. Scott, and A. Kornberg.** 1980. A temperature-sensitive single-strand DNA-binding protein from *Escherichia coli*. J. Biol. Chem. **255**:2897–2901.
17. **Miller, J. H.** 1972. Experiments in molecular genetics. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.
18. **Rupp, W. D., and P. Howard-Flanders.** 1968. Discontinuities in the DNA synthesized in an excision-defective strain of *Escherichia coli* following ultraviolet irradiation. J. Mol. Biol. **31**:291–304.
19. **Rupp, W. D., C. E. Wilde III, D. L. Reno, and P. Howard-Flanders.** 1971. Exchanges between DNA strands in ultraviolet-irradiated *Escherichia coli*. J. Mol. Biol. **61**:25–44.
20. **Scott, J. F., S. Eisenberg, L. L. Bertsch, and A. Kornberg.** 1977. A mechanism of duplex DNA replication revealed by enzymatic studies of phage φX174. Catalytic strand separation in advance of replication. Proc. Natl. Acad. Sci. U.S.A. **74**:193–197.
21. **Seeberg, E.** 1978. Reconstitution of an *Escherichia coli* repair endonuclease activity from the separated *uvrA*$^+$ and *uvrB*$^+$/*uvrC*$^+$ gene products. Proc. Natl. Acad. Sci. U.S.A. **75**:2569–2573.
22. **Shimada, K., H. Ogawa, and J. Tomizawa.** 1968. Studies on radiation-sensitive mutants of *E. coli*. II. Breakage and repair of ultraviolet irradiated intracellular DNA of phage lambda. Mol. Gen. Genet. **101**:245–256.
23. **Smith, K. C.** 1969. DNA synthesis in sensitive and resistant mutants of *Escherichia coli* B after ultraviolet irradiation. Mutat. Res. **8**:481–495.
24. **Smith, K. C., and M. E. O'Leary.** 1968. The pitfalls of measuring DNA synthesis kinetics as exemplified in ultraviolet radiation studies. Biochim. Biophys. Acta **169**:430–438.
25. **Tang, M.-s., and K. C. Smith.** 1981. The effects of *lexA101*, *recB21*, *recF143* and *uvrD3* mutations on liquid-holding recovery in ultraviolet-irradiated *Escherichia coli* K12 *recA56*. Mutat. Res. **80**:15–25.
26. **Vales, L. D., J. W. Chase, and J. B. Murphy.** 1980. Effect of *ssbA1* and *lexC113* mutations on lambda prophage induction, bacteriophage growth, and cell survival. J. Bacteriol. **143**:887–896.
27. **Wang, T.-c. V., and K. C. Smith.** 1981. Effect of *recB21*, *uvrD3*, *lexA101* and *recF143* mutations on ultraviolet radiation sensitivity and genetic recombination in Δ*uvrB* strains of *Escherichia coli*. Mol. Gen. Genet. **183**:37–44.
28. **Weiner, J. H., L. L. Bertsch, and A. Kornberg.** 1975. The deoxyribonucleic acid unwinding protein of *Escherichia coli*. J. Biol. Chem. **250**:1972–1980.
29. **Whittier, R. F., and J. W. Chase.** 1981. DNA repair in *E. coli* strains deficient in single-strand DNA binding protein. Mol. Gen. Genet. **183**:341–347.
30. **Wickner, S., and J. Hurwitz.** 1974. Conversion of φX174 viral DNA to double-stranded form by purified *Escherichia coli* proteins. Proc. Natl. Acad. Sci. U.S.A. **71**:4120–4124.
31. **Youngs, D. A., and K. C. Smith.** 1976. Genetic control of multiple pathways of post-replication repair in *uvrB* strains of *Escherichia coli* K-12. J. Bacteriol. **125**:102–110.