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Mechanism of *sbcB*-suppression of the *recBC*-deficiency in postreplication repair in UV-irradiated *Escherichia coli* K-12

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Summary. The mechanism by which an *sbcB* mutation suppresses the deficiency in postreplication repair shown by *recB recC* mutants of *Escherichia coli* was studied. The presence of an *sbcB* mutation in *uvrA recB recC* cells increased their resistance to UV radiation. This enhanced resistance was not due to a suppression of the minor deficiency in the repair of DNA daughter-strand gaps or to an inhibition of the production of DNA double-strand breaks in UV-irradiated *uvrA recB recC* cells; rather, the presence of an *sbcB* mutation enabled *uvrA recB recC* cells to carry out the repair of DNA double-strand breaks. In the *uvrA recB recC sbcB* background, a mutation at *recF* produced a huge sensitization to UV radiation, and it rendered cells deficient in the repair of both DNA daughter-strand gaps and DNA double-strand breaks. Thus, an additional *sbcB* mutation in *uvrA recB recC* cells restored their ability to perform the repair of DNA double-strand breaks, but the further addition of a *recF* mutation blocked this repair capacity.

Introduction

Based on studies of various recombination-deficient mutants, at least two different pathways of chromosomal recombination have been described in *Escherichia coli* K-12, i.e., the *recBC*-pathway and the *recF*-pathway (Clark 1973; Clark et al. 1984). The *recBC*-pathway is the major recombination process during conjugation, and accounts for about 99% of the recombinants scored, while the *recF*-pathway accounts for only 1% of the recombinants (Horii and Clark 1973). Although knowledge of the events during recombination is very limited, studies on recombination following conjugation in *Escherichia coli* suggest that the *recF*-pathway may utilize DNA containing a single-stranded region to promote recombination, whereas the *recBC*-pathway may utilize the ends of linear duplex DNA to promote recombination (Mahajan and Datta 1979; Lloyd and Thomas 1983, 1984; Clark et al. 1984). In vitro evidence supporting the requirement for linear duplex DNA containing blunt or nearly blunt ends to initiate *recBC*-dependent recombination has recently been obtained (Taylor and Smith 1985).

Analogous to conjugational recombination, two major pathways for the postreplication repair of UV radiation-damaged DNA have been observed in excision-deficient cells of *E. coli* K-12, i.e., the *recBC*-pathway and the *recF*-pathway (Rothman et al. 1975; Wang and Smith 1981). These two pathways of postreplication repair are equally important in terms of cell survival, when assayed on minimal growth medium, and they are involved in the repair of different DNA lesions (Wang and Smith 1983). Several studies indicate that the *recF*-dependent pathway is largely responsible for the repair of DNA daughter-strand gaps, which are produced in nascent DNA when the replication complex proceeds past UV radiation-induced pyrimidine dimers (Ganesan and Seawell 1975; Rothman and Clark 1977; Kato 1977; Wang and Smith 1983). On the other hand, the *recBC*-dependent pathway plays little role in the repair of DNA daughter-strand gaps, but is responsible for the repair of DNA double-strand breaks that arise from unrepaired DNA daughter-strand gaps (Wang and Smith 1983, 1985). Therefore, it appears that, depending on the nature of the DNA lesions produced in the replicated DNA of UV-irradiated cells, these lesions may be repaired either by the *recF*-dependent pathway or the *recBC*-dependent pathway of postreplication repair.

The recombinational defect and the radiation sensitivity of *recB recC* mutants are known to be suppressed by a mutation in the *sbcB* gene, which codes for DNA exonuclease I (Exo I) (Kushner et al. 1971, 1972), an enzyme that degrades single-stranded DNA from the 3′ terminus (Lehman and Nussbaum 1964). It has been postulated that the inactivation of Exo I by an *sbcB* mutation may preserve a putative DNA intermediate for the *recF*-pathway of recombination in *recB recC* mutants (Horii and Clark 1973; Clark et al. 1984), thus accounting for the phenotypic suppression of the recombination-deficiency and radiation sensitivity of *recB recC* mutants.

Since *recF*-dependent and *recBC*-dependent repair processes are required in UV-irradiated cells for the postreplication repair of DNA daughter-strand gaps and DNA double-strand breaks, respectively, we considered the following possibilities for the effect of an *sbcB* mutation: (1) The $sbcB^{+}$ gene product is required for the conversion of a DNA daughter-strand gap to a DNA double-strand break; thus, an *sbcB* mutation would preserve the DNA daughter-strand gaps for repair by *recF*-dependent recombination. (2) The $sbcB^{+}$ gene product and the RecBC enzyme are involved in the degradation of single-stranded DNA at the free ends of a DNA double-strand break, and produce a substrate that is no longer suitable for *recF*-dependent recombination (i.e., linear duplex DNA with blunt ends).

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Table 1. *E. coli* K-12 strains used[a]

Stanford radiology No.	Genotype	Source or derivation
SR114	*uvrA6 argE3 his-4 leuB6 proA2 thr-1 ara-14 galK2 lacY1 mtl-1 xyl-5 thi-1 tsx-33 rpsL31 supE44*	S. Linn (AB1886)
SR1119	*deoC araD139 Δ(lac) U169 malE7*::Tn*5 f16B relA rpsL*	T. Silhavy (T5M7)
SR1274	*trpA540*	C. Yanofsky (W3110 *trpA540)*
SR1367	*recF332*::Tn*3 argE3 his-4 leuB6 proA2 thr-1 ara-14 galK2 lacY1 mtl-1 xyl-5 thi-1 tsx-33 rpsL31 supE44 tnaA*::Tn*10 HK19*^{r} *Φ*X174^s S13^s	A.J. Clark (JC10990)
SR1388	*recB21 recC22 sbcB15 argE3 his-4 leuB6 proA2 thr-1 ara-14 galK2 lacY1 mtl-1 xyl-5 thi-1 tsx-33 rpsL31 supE44*	A.J. Clark (JC7623)
SR1417	as SR1388, but *malE7*::Tn*5*	SR1388 × Plvira · SR1119, select *malE7*::Tn*5* (Kn^r)
SR1419	as SR1417, but *uvrA6* Mal$^+$ Kn^s	SR1417 × Plvira · SR114, select Mal$^+$ Kn^s
SR1424	as SR1419 but *recF332*::Tn*3*	SR1419 × Plvira · SR1367, select Ap^r
SR1454	as SR1419, but His$^+$	SR1419 × Plvira · SR1274, select His$^+$
SR1455	as SR1419, but *sbcB*$^+$ His$^+$	SR1419 × Plvira · SR1274, select His$^+$
SR1619	as SR1455, but *recF332*::Tn*3*	SR1455 × Plvira · SR1367, select Ap^r

[a] All strains are F$^-$ and λ$^-$. Except for SR1119 and SR1274, all strains are derivatives of AB1157. Genotype symbols are those used by Bachmann (1983)

However, in the absence of such degradation, a DNA double-strand break produced in *recB recC sbcB* mutants would be a substrate for *recF*-dependent recombination. Our results are consistent with the second postulate.

Materials and methods

Bacterial strains and media. The bacterial strains used are listed in Table 1. The transduction technique used in strain construction was similar to that described by Miller (1972). Supplemented minimal medium (SMM) and DTM buffer have been described (Wang and Smith 1982).

Irradiation. The source (254 nm) and measurement of fluence rate for UV irradiation have been described (Wang and Smith 1981). For survival studies, cultures were grown in SMM and UV irradiated as previously described (Wang and Smith 1983). Survivors were determined by assaying colony-forming units on SMM agar.

DNA-repair studies. Cells were grown exponentially at 37° C in SMM. The cultures were UV-irradiated and pulse-labeled with [methyl-^{3}H]-thymidine (64 Ci/mmol; Amersham Corp.) as described previously (Wang and Smith 1983). The fate of DNA newly synthesized after UV irradiation was followed during repair incubation by sedimentation analysis on both alkaline and neutral sucrose gradients, as previously described (Wang and Smith 1983). The recovery of radioactivity from both alkaline and neutral sucrose gradients was greater than 90% in all samples tested. Sedimentation data obtained from alkaline sucrose gradients were used to determine the number-average molecular weights, Mn, for single-stranded DNA, as previously described (Wang and Smith 1983). The Mn value of [^{3}H]DNA after 120 min of repair incubation were used to calculate the average number of UV radiation-induced DNA single-strand breaks per genome using the formula

$$[(\mathrm{Mn})c/(\mathrm{Mn})uv - 1] \cdot [2.8 \times 10^9/(\mathrm{Mn})c],$$

where (Mn)c and (Mn)uv are the number-average molecular weights of DNA from unirradiated control and UV-irradiated samples, respectively. It is assumed that the UV radiation-induced DNA single-strand breaks in the nascent DNA after 120 min of repair incubation were due to DNA daughter-strand gaps that were not repaired. Neutral sucrose gradients were used to study the formation and repair of DNA double-strand breaks.

Results

Survival studies

To study the mechanism by which an *sbcB* mutation suppresses the deficiency in postreplication repair in *recB recC* mutants, excision repair-deficient cells were used to avoid any complications that might arise as a result of the excision repair process. In the *uvrA* mutant background, the presence of an *sbcB15* mutation suppressed much of the UV-radiation sensitivity observed in *recB recC* mutants (Fig. 1). When the UV radiation fluences that inactivate 90% of the cell population were compared, the introduction of a *recF* mutation into *uvrA recB recC sbcB* cells produced an increase in UV-radiation sensitivity of about 24-fold. The *uvrA recB recC sbcB recF* cells were about twice as resistant as *uvrA recB recC recF* cells to UV radiation, but were 4- to 5-fold more sensitive than a *uvrA recB recC* strain (Fig. 1), or a *uvrA recF* strain (data not shown).

Repair of DNA daughter-strand gaps

The cells were UV-irradiated, pulse-labeled with ^{3}H-thymidine, and incubated in nonradioactive medium at 37° C for 2 h to allow the maximal repair of DNA daughter-strand gaps (Smith and Meun 1970; unpublished data). A representative profile of ^{3}H-DNA after 2 h of repair incubation

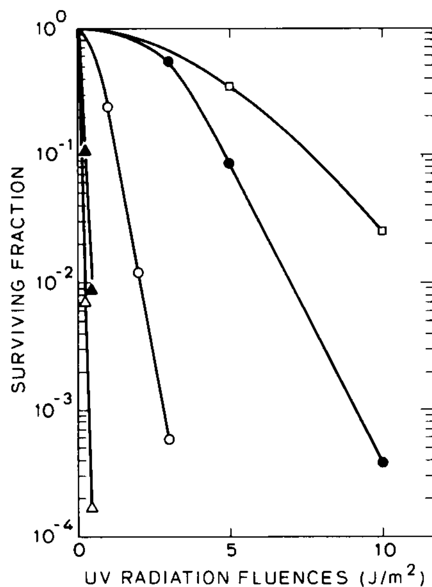

Fig. 1. Survival curves of UV-irradiated *uvrA* cells of *E. coli* K-12. All strains were grown and treated as described in Materials and methods. Colony-forming units were assayed on supplemented minimal medium. □, *uvrA* (SR114); ○, *uvrA recB recC* (SR1455); ●, *uvrA recB recC sbcB* (SR1454); ▲, *uvrA recB recC sbcB recF* (SR1424); and △, *uvrA recB recC recF* (SR1619). Data are the average of two experiments

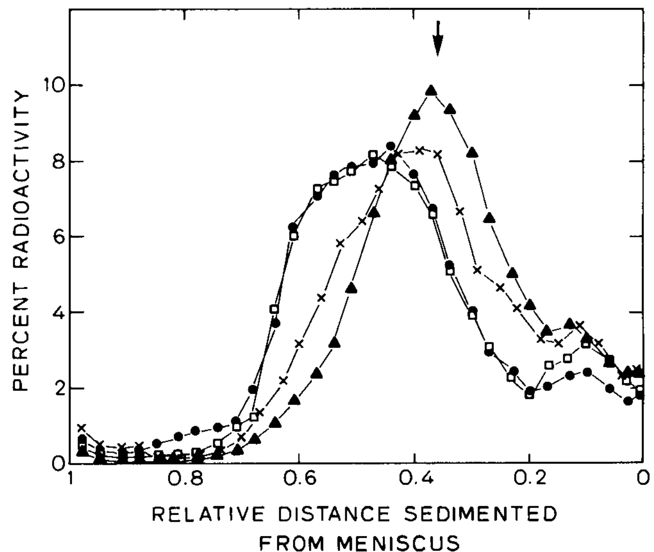

Fig. 2. Sedimentation on alkaline sucrose gradients of DNA synthesized after UV irradiation. Cells of strain SR1454 (*uvrA recB recC sbcB*) were UV irradiated, pulse-labeled with ^{3}H-thymidine at 37° C for 5 min, and incubated in nonradioactive supplemented minimal medium for 2 h. The cells were converted to spheroplasts and sedimented on alkaline sucrose gradients as described in Materials and methods. The *arrow* indicates the position of a bacteriophage T2 DNA marker. Unirradiated control (●); 3 J/m^2 (□); 6 J/m^2 (×); and 9 J/m^2 (▲)

and subsequent fractionation on alkaline sucrose gradients is shown in Fig. 2. The number average molecular weight of the DNA was determined from the major peak of the DNA profile (i.e., the top 5–6 fractions of the gradient were not included), and was used to calculate the average number of DNA single-strand breaks (i.e., the number of unrepaired DNA daughter-strand gaps) as described in Materials and methods.

The *recB recC* mutations produce a minor deficiency in the repair of DNA daughter-strand gaps in *uvrA* cells (Fig. 3), as was also observed for the effect of a *recB* mutation in the *uvrB* background (Youngs and Smith 1976; Wang and Smith 1983). This minor deficiency in the repair of DNA daughter-strand gaps is not suppressed by an *sbcB* mutation (Fig. 3). The introduction of a *recF* mutation into *uvrA recB recC sbcB* cells produced a large inhibition in the repair of DNA daughter-strand gaps (Fig. 3).

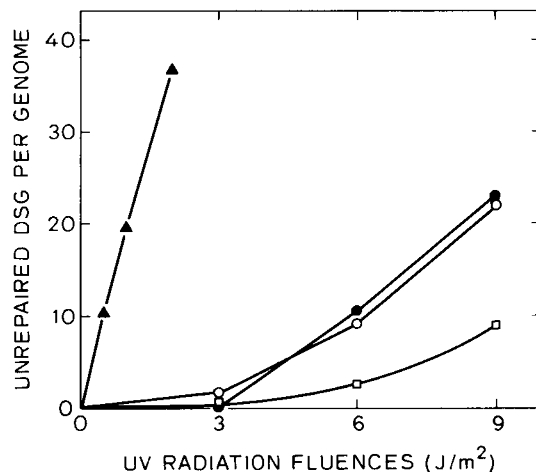

Fig. 3. Effect of *sbcB* and *recF* mutations on the repair of DNA daughter-strand gaps (DSG) in UV-irradiated *uvrA recB recC* cells of *E. coli* K-12. The experimental protocol was the same as that described in the legend to Fig. 2. The average number of UV radiation-induced DNA single-strand breaks in the nascent DNA (i.e., unrepaired DSG) was calculated as described in Materials and methods. *Symbols* for the strains are the same as shown in the legend to Fig. 1. Data for strains SR114 and SR1424 are from one experiment, the other data are the average of two experiments

Postreplicational formation and repair of DNA double-strand breaks

Since the *recB* mutants are deficient in the repair of DNA double-strand breaks (Wang and Smith 1983), an indirect suppression of this deficiency by an *sbcB* mutation might occur by inhibiting the postreplicational formation of DNA double-strand breaks, or by allowing the repair of DNA double-strand breaks. These two alternatives were tested by following the kinetics of the postreplicational formation and repair of DNA double-strand breaks in UV-irradiated cells (Fig. 4).

With the continued incubation of UV-irradiated (3 J/m^2) *uvrA recB recC* cells, the ^{3}H-DNA that was pulse-labeled after UV-irradiation was converted to molecules that sedimented slowly in neutral sucrose gradients, indicating the formation of DNA double-strand breaks, and these DNA double-strand breaks were not repaired (Fig. 4A). In contrast, when the ^{3}H-DNA synthesized in UV-irradiated (3 J/m^2) *uvrA recB recC sbcB* cells was followed during repair incubation, a smaller fraction of the ^{3}H-DNA sedi-

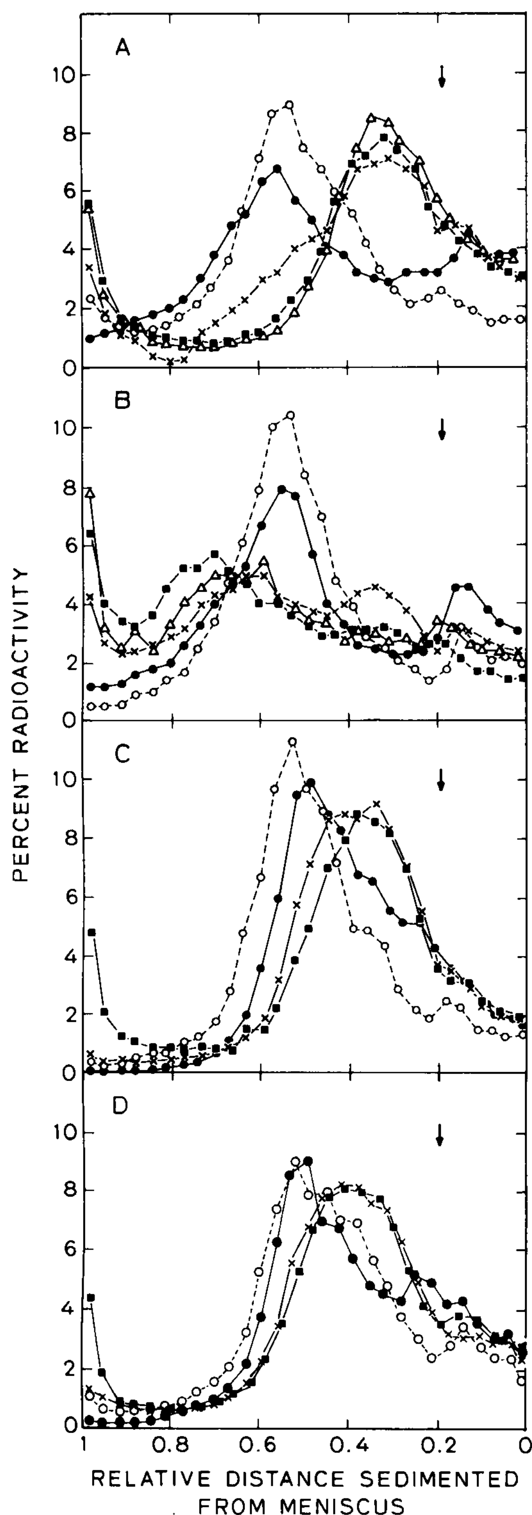

Fig. 4. Kinetics of the formation and repair of DNA double-strand breaks in UV-irradiated (**A**) *uvrA recB recC* (SR1455), (**B**) *uvrA recB recC sbcB* (SR1454), (**C**) *uvrA recB recC sbcB recF* (SR1424), and (**D**) *uvrA recB recC recF* (SR1619) cells. The cells were UV-irradiated and pulse-labeled with [^{3}H]thymidine as described in Materials and methods. The *uvrA recB recC recF* and *uvrA recB recC sbcB recF* cells were very deficient in the repair of DNA daughter-strand gaps (Fig. 3, and unpublished data) and were irradiated with 0.25 J/m^2, all other strains were irradiated with 3 J/m^2. The repair of the [^{3}H]DNA was followed during subsequent repair incubation by sedimentation on neutral sucrose gradients. The *arrows* indicate the positions of bacteriophage T2 DNA markers. Unirradiated control (o); UV-irradiated with no repair incubation (•); UV-irradiated and incubated for 2 h (×), 4 h (■) or 6 h (△)

mented slowly during the first two hours of incubation. After longer incubation times, the majority of the DNA sedimented at a fast rate (Fig. 4 B), indicating that the additional *sbcB* mutation facilitated the repair of these DNA double-strand breaks.

We also investigated the postreplicational formation and repair of DNA double-strand breaks in UV-irradiated *uvrA recB recC sbcB recF* (0.25 J/m^2) cells. As shown in Fig. 4C, the ^{3}H-DNA synthesized after UV-irradiation was converted to slowly-sedimenting DNA during repair incubation, and these DNA double-strand breaks were not repaired, indicating that an *sbcB* mutation does not prevent the production of DNA double-strand breaks, and that a *recF* mutation renders *uvrA recB recC sbcB* cells deficient in the repair of DNA double-strand breaks. These results were similar to those obtained with UV-irradiated (0.25 J/m^2) *uvrA recB recC recF* cells (Fig. 4D).

Discussion

Our survival studies (Fig. 1) indicate that the UV-radiation sensitivity of a *uvrA recB recC* strain can be suppressed by an *sbcB* mutation, and that a functional *recF* gene is needed for the expression of this enhanced resistance to UV radiation. These data are in general agreement with the results obtained with *uvr*$^+$ cells, and are consistent with the postulate that an *sbcB* mutation acts by shunting DNA intermediates primarily to a repair pathway that is dependent on functional *recF* gene products (Horii and Clark 1973). It should be noted that an *sbcB* mutation also suppresses the UV radiation sensitivity of *uvrA recB recC recF* cells by about 2-fold (Fig. 1). This enhanced resistance to UV radiation in *uvrA recB recC recF sbcB* cells may be related to an enhanced capacity to perform the *recBC recF*-independent repair of DNA daughter-strand gaps (Wang and Smith 1983, 1984; Sharma and Smith 1985).

Our DNA repair studies indicate that an *sbcB* mutation does not suppress the minor deficiency in the repair of DNA daughter-strand gaps in *uvrA recB recC* cells (Fig. 3), nor does it inhibit the formation of DNA double-strand breaks in UV-irradiated *uvrA recB recC recF* cells (Fig. 4C); rather, the presence of an *sbcB* mutation enables *uvrA recB recC* cells to repair DNA double-strand breaks (Fig. 4B). In contrast to the results for UV-irradiated *uvrA recB recC* cells, where DNA double-strand breaks are produced and remain unrepaired (Fig. 4A), the complex kinetics observed in UV-irradiated *uvrA recB recC sbcB* cells (Fig. 4B) may reflect the fact that both the formation and repair of DNA double-strand breaks occur simultaneously. We consistently observed that during the repair of DNA double-strand breaks in *uvrA recB recC sbcB* cells, there was a component of ^{3}H-DNA from UV-irradiated cells that had a faster sedimentation rate than did DNA from unirradiated cells. This component of ^{3}H-DNA reached its fastest sedimentation rate after about 4 h of incubation at 37° C, and then gradually returned towards that of the unirradiated control (Fig. 4B). The nature of this fast-sedimenting DNA is not known, but it may reflect a repair intermediate similar to

that observed during the repair of DNA double-strand breaks produced by ionizing radiation (Ulmer et al. 1979).

We offer two explanations, which are not mutually exclusive, for this effect of an *sbcB* mutation. One explanation is that the *sbcB* gene may have a regulatory function, and an *sbcB* mutation may derepress a set of genes whose products can perform the repair of DNA double-strand breaks in *recB recC* mutants. Since a *recF* mutation inhibits the repair of DNA double-strand breaks in the *recB recC sbcB* background (Fig. 4C), the *recF* gene may be among those genes that are derepressed by an *sbcB* mutation, or else the induced repair enzymes require the cooperation of functional *recF* gene products to perform the repair of DNA double-strand breaks. A second explanation is that Exo I is involved in the degradation of single-stranded tails at the free ends of a DNA double-strand break. We postulate that when a DNA daughter-strand gap, which is about 1000 nucleotides long (Iyer and Rupp 1971), is converted to a DNA double-strand break by a putative single-stranded DNA endonuclease, the resulting DNA double-strand break could contain long single-stranded tails. In *recBC*$^+$ *sbcB*$^+$ cells, these single-stranded tails may be degraded by the single-strand DNA exonuclease activities of Exo I and Exo V to produce blunt ends so that Exo V can unwind the duplex (Rosamond et al. 1979; Smith et al. 1984) and proceed to initiate recombination, leading to the repair of these double-strand breaks. In the absence of such degradation (i.e., in *recB recC sbcB* cells), these DNA double-strand breaks containing long single-strand tails are preserved as substrates for *recF*-dependent recombination. The utilization of DNA containing single-stranded regions in *recF*-dependent recombination has been postulated (see Clark et al. 1984; Lloyd and Thomas 1984).

The involvement of the *recF* gene in postreplication repair was previously thought to be in the repair of DNA daughter-strand gaps, and not in the repair of DNA double-strand breaks (Wang and Smith 1983). However, the present work indicates that the *recF* gene plays a role in the repair of DNA double-strand breaks as well as DNA daughter-strand gaps in *uvrA recBC sbcB* cells. Perhaps depending on the chemical nature of the DNA double-strand breaks, they may be repaired either by *recBC*-dependent recombination or by *recF*-dependent recombination. Although the *recF*-dependent repair of DNA double-strand breaks may be a minor pathway in *recBC*$^+$ *sbcB*$^+$ cells, it appears to be the major pathway in *recBC sbcB* cells.

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References

Bachmann BJ (1983) Linkage map of *Escherichia coli* K-12, edn 7. Microbiol Rev 47:180–230

Clark AJ (1973) Recombination deficient mutants of *E. coli* and other bacteria. Ann Rev Genet 7:67–86

Clark AJ, Sandler SJ, Willis KD, Chu CC, Blanar MA, Lovett ST (1984) Genes of the RecE and RecF pathways of conjugational recombination in *E. coli*. Cold Spring Harbor Symp Quant Biol 49:453–462

Ganesan AK, Seawell PC (1975) The effect of *lexA* and *recF* mutations on post-replication repair and DNA synthesis in *Escherichia coli* K-12. Mol Gen Genet 141:189–205

Horii ZI, Clark AJ (1973) Genetic analysis of the RecF pathway of genetic recombination in *Escherichia coli* K12: isolation and characterization of mutants. J Mol Biol 80:327–344

Iyer VN, Rupp WD (1971) Usefulness of benzoylated naphthoylated DEAE-cellulose to distinguish and fractionate double-stranded DNA bearing different extents of single-stranded regions. Biochim Biophys Acta 222:117–126

Kato T (1977) Effects of chloramphenicol and caffeine on postreplication repair in *uvrA*$^-$ *umuC*$^-$ and *uvrA*$^-$ *recF*$^-$ strains of *Escherichia coli* K-12. Mol Gen Genet 156:115–120

Kushner SR, Nagaishi H, Templin A, Clark AJ (1971) Genetic recombination in *Escherichia coli*: The role of Exonuclease I. Proc Natl Acad Sci USA 68:824–827

Kushner SR, Nagaishi H, Clark AJ (1972) Indirect suppression of *recB* and *recC* mutations by Exonuclease I deficiency. Proc Natl Acad Sci USA 69:1366–1370

Lehman KR, Nussbaum AL (1964) The deoxyribonucleases of *Escherichia coli*. On the specificity of exonuclease I (Phosphodiesterase). J Biol Chem 239:2628–2636

Lloyd RG, Thomas A (1983) On the nature of the RecBC and RecF pathways of conjugational recombination in *Escherichia coli*. Mol Gen Genet 190:156–161

Lloyd RG, Thomas A (1984) A molecular model for conjugational recombination in *Escherichia coli* K12. Mol Gen Genet 197:328–336

Mahajan SK, Datta AR (1979) Mechanisms of recombination by the RecBC and the RecF pathways following conjugation in *Escherichia coli* K12. Mol Gen Genet 169:67–78

Miller JH (1972) Experiments in molecular genetics. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York

Rosamond J, Telander KM, Linn S (1979) Modulation of the action of the *recBC* enzyme of *Escherichia coli* by Ca^{2+}. J Biol Chem 254:8646–8652

Rothman RH, Clark AJ (1977) The dependence of postreplication repair on *uvrB* in a *recF* mutant of *Escherichia coli* K-12. Mol Gen Genet 155:279–286

Rothman RH, Kato T, Clark AJ (1975) The beginning of an investigation of the role of *recF* in the pathways of metabolism of ultraviolet-irradiated DNA in *Escherichia coli*. In: Hanawalt PC, Setlow RB (eds) Molecular mechanisms for repair of DNA. Plenum Publishing Corporation, New York, pp 283–291

Sharma RC, Smith KC (1985) A minor pathway of postreplication repair in *Escherichia coli* is independent of the *recB*, *recC* and *recF* genes. Mutat Res 146:169–176

Smith KC, Meun DHC (1970) Repair of radiation-induced damage in *Escherichia coli*. 1. Effect of *rec* mutations on post-replication repair of damage due to ultraviolet radiation. J Mol Biol 51:459–472

Smith GR, Amundsen SK, Chaudhury AM, Cheng KC, Ponticelli AS, Roberts CM, Schultz DW, Taylor AF (1984) Roles of RecBC enzyme and Chi sites in homologous recombination. Cold Spring Harbor Symp Quant Biol 49:485–495

Taylor AF, Smith GR (1985) Substrate specificity of the DNA unwinding activity of the RecBC enzyme of *Escherichia coli*. J Mol Biol (in press)

Ulmer KM, Gomez RF, Sinskey AJ (1979) Ionizing radiation damage to the folded chromosome of *Escherichia coli* K-12: Repair of double-strand breaks in deoxyribonucleic acid. J Bacteriol 138:486–491

Wang TV, Smith KC (1981) Effect of *recB21*, *uvrD3*, *lexA101* and *recF143* mutations on ultraviolet radiation sensitivity and genetic recombination in *ΔuvrB* strains of *Escherichia coli*. Mol Gen Genet 183:37–44

Wang TV, Smith KC (1982) Effects of the *ssb-1* and *ssb-113* mutations on survival and DNA repair in UV-irradiated *ΔuvrB* strains of *Escherichia coli* K-12. J Bacteriol 151:186–192

Wang TV, Smith KC (1983) Mechanisms for the *recF*-dependent and *recB*-dependent pathways of postreplication repair in UV-irradiated *Escherichia coli uvrB*. J Bacteriol 156:119–124

Wang TV, Smith KC (1984) *recF*-dependent and *recF recB*-independent DNA gap-filling repair processes transfer dimer-containing parental strands to daughter strands in *Escherichia coli* K-12 *uvrB*. J Bacteriol 158:727–729

Wang TV, Smith KC (1985) Postreplicational formation and repair of DNA double-strand breaks in UV-irradiated *Escherichia coli uvrB* cells. Mutat Res (in press)

Youngs DA, Smith KC (1976) Genetic control of multiple pathways of post-replicational repair in *uvrB* strains of *Escherichia coli* K-12. J Bacteriol 125:102–110

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