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Different Effects of *recJ* and *recN* Mutations on the Postreplication Repair of UV-Damaged DNA in *Escherichia coli* K-12

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Two mutations known to affect recombination in a *recB recC sbcBC* strain, *recJ284*::Tn*10* and *recN262*, were examined for their effects on the postreplication repair of UV-damaged DNA. The *recJ* mutation did not affect the UV radiation sensitivity of *uvrB* and *uvrB recF* cells, but it increased the sensitivity of *uvrB recN* (~3-fold) and *uvrB recB* (~8-fold) cells. On the other hand, the *recN* mutation did not affect the UV sensitivity of *uvrB recB* cells, but it increased the sensitivity of *uvrB* (~1.5-fold) and *uvrB recF* (~4-fold) cells. DNA repair studies indicated that the *recN* mutation produced a partial deficiency in the postreplication repair of DNA double-strand breaks that arise from unrepaired daughter strand gaps, while the *recJ* mutation produced a deficiency in the repair of daughter strand gaps in *uvrB recB* cells (but not in *uvrB* cells) and a deficiency in the repair of both daughter strand gaps and double-strand breaks in *uvrA recB recC sbcBC* cells. Together, these results indicate that the *recJ* and *recN* genes are involved in different aspects of postreplication repair.

Conjugational recombination in wild-type *Escherichia coli* K-12 depends on the products of a number of genes, e.g., *recA* (3), *recB* (9), and *recC* (5). While mutations in *recA* can produce a complete deficiency in recombination, mutations in either *recB* or *recC* do not. The recombinational deficiency of *recB*(*C*) mutants is suppressible by a mutation in *sbcB* (12), which codes for DNA exonuclease I, an enzyme that degrades single-stranded DNA from the 3′ terminus (13). Recently, it has been shown that the commonly used *recB recC sbcB* strains carry an additional mutation in *sbcC* (15) that is necessary for full suppression of the *recBC* mutant phenotype. In *recB* (*recC*) *sbcBC* cells, an additional mutation in a *recF* (8, 24), *lexA* (17, 18), *recJ* (19), *recN* (22), *recQ* (21), *recO* (11), or *ruv* (14, 29) gene decreases recombination proficiency, and these genes are thought to affect *recBC*-independent recombination. Horii and Clark (8) have called this the RecF pathway of recombination. In wild-type cells, *recBC*-dependent recombination accounts for about 99% of the recombinants scored, while the RecF pathway of recombination accounts for the remainder (8).

Several lines of evidence indicate that recombination plays a key role in postreplication repair. Two processes of postreplication repair have been observed in excision-deficient cells of *E. coli* K-12 following UV irradiation (25, 35): (i) the repair of DNA daughter strand gaps that are thought to arise when replication skips past a noncoding lesion in DNA (e.g., pyrimidine dimer) and reinitiates downstream from the lesion (25), and (ii) the repair of DNA double-strand breaks that are thought to arise at unrepaired daughter strand gaps (35). While both processes are dependent on a functional *recA* gene (30), the repair of daughter strand gaps has an additional requirement for a functional *recF* gene (7, 10, 23, 35), and the repair of double-strand breaks has an additional requirement for a functional *recB* gene (35, 37). However, in the *uvr recB recC sbcBC* background, in which the *recBC* deficiency in recombination and repair is suppressed by the *sbcBC* mutations, the repair of double-strand breaks becomes dependent on *recF* (36). It has been postulated that DNA double-strand breaks that have blunt or nearly blunt ends are repaired by the *recBC*-dependent process, while double-strand breaks that contain long single-stranded tails are repaired by the *recF*-dependent process (36).

Studies on recombination processes following conjugation (4, 17) and on the postreplication repair of UV-damaged DNA (35, 36) have pointed to the same general conclusion: that those recombination and repair processes that rely on *recF* may utilize DNA containing a single-stranded region to promote recombination, while those requiring the *recBC* function may utilize linear duplex DNA with blunt or nearly blunt ends (31). However, little else is known about the actual molecular specifics involved in either case. Genetic and biochemical studies of postreplication repair in excision-deficient cells have identified a number of additional genes (e.g., *uvrD*, *lexA* [33], *radB* [26], and *polA* [28]) that affect the *recBC*-dependent process of postreplication repair, yet none has been identified that affects *recF*-dependent processes. The recent identification of a number of new genes thought to affect the RecF pathway of recombination (see above) prompted us to examine the role of these genes in postreplication repair. In this work, we report that the *recJ* gene is involved in *recF*-dependent repair processes, while the *recN* gene is involved largely in *recBC*-dependent repair processes.

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 (20). The identification of *recN*, *recF*, and *recB* mutants among transductants was done by screening their sensitivity to UV radiation. In many strains, a *recJ* mutation had no phenotypic effect. In such cases, the presence of the *recJ* mutation was confirmed by backcrossing into a *uvrA recB recC sbcB* (strain SR1419) recipient and testing for UV sensitivity. Supplemented minimal medium (SMM) and DTM buffer have been described (34). Selection media were 0.75% yeast extract (Difco) and 2.3% nutrient agar (Difco) containing tetracycline at 15 μg/ml or ampicillin at 40 μg/ml.

UV irradiation. The source (254 nm) and measurement of fluence rate for UV irradiation have been described (33). For survival studies, cultures were grown in SMM and irradiated

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TABLE 1. *E. coli* strains used[a]

Strain	Genotype	Derivation, source, or reference[b]
KH21 [Δ(*uvrB-chlA*) background][c]		
SR596	*rec*$^+$	35
SR1510	*recN262 tyrA16*::Tn*10*	SR596 × P1 SR1474, select Tc^r
SR1511	*recF143 tyrA16*::Tn*10*	SR305 × P1 SR1474, select Tc^r
SR1512	*recF143 recN262 tyrA16*::Tn*10*	SR305 × P1 SR1474, select Tc^r
SR1518	*recB21 tyrA16*::Tn*10 thyA*$^+$	SR1509 × P1 SR257, select Thy$^+$
SR1520	*recB21 recN262 tyrA16*::Tn*10 thyA*$^+$	SR1510 × P1 SR257, select Thy$^+$
SR1522	*recB21 recF143 tyrA16*::Tn*10 thyA*$^+$	SR1511 × P1 SR257, select Thy$^+$
SR1675	*recJ284*::Tn*10*	SR596 × P1 SR1660, select Tc^r
SR1676	*recF143 recJ284*::Tn*10*	SR305 × P1 SR1660, select Tc^r
SR1682	*recB21 recJ284*::Tn*10 thyA*$^+$	SR1675 × P1 SR1159, select Thy$^+$
SR1776	*recJ284*::Tn*10 recN262*	SR1774 × P1 SR1681, select Tc^r
AB1157 (*uvrA6 recB21 recC22 sbcB15* background)[d]		
SR1424	*recF332*::Tn*3*	SR1419 × P1 SR1367, select Ap^r
SR1507	*tyrA16*::Tn*10*	SR1419 × P1 SR1474, select Tc^r
SR1508	*recN262 tyrA16*::Tn*10*	SR1419 × P1 SR1474, select Tc^r
SR1677	*recJ284*::Tn*10*	SR1419 × P1 SR1660, select Tc^r
Strains used only for strain construction		
SR248	F$^-$ *leuB19 metE70 thyA36 bioA2 deo*(*C2*?) *lacZ53 malB45 rha-5 rpsL151* IN(*rrnD-rrnE*) λ$^-$	33; R. B. Hellig (KH21)
SR257	F$^-$ *uvrB5 recB21 leuB19 metE70 deo*(*C2*?) *lacZ53 rha-53 rpsL151* IN (*rrnD-rrnE*) λ$^-$	33; D. A. Youngs (DY157)
SR305	F$^-$ Δ(*uvrB-chlA*) *recF143 leuB19 thyA36 deo*(*C2*?) *lacZ53 malB45 rha-5 rpsL151* IN(*rrnD-rrnE*) λ$^-$	35; D. A. Youngs (DY243)
SR1159[d]	*recB21*	N. J. Sargentini
SR1367[d]	*recF332*::Tn*3 tnaA*::Tn*10* HK19^r φX174^s S13^s	A. J. Clark (JC10990)
SR1419[d]	*uvrA6 recB21 recC22 sbcB15*	36
SR1474[d]	*uvrB5 recN262 tyrA16*::Tn*10*	S. M. Picksley (SP264)
SR1509	F$^-$ Δ(*uvrB-chlA*) *tyrA16*::Tn*10 leuB19 thyA36 deo*(*C2*?) *lacZ53 malB45 rha-5 rpsL151* IN(*rrnD-rrnE*) λ$^-$	SR596 × P1 SR1474, select Tc^r
SR1660[d]	F$^-$ *recB21 recC22 sbcA23 recJ284*::Tn*10 tsx*$^+$ *supE*$^+$	A. J. Clark (JC12105)
SR1681	F$^-$ Δ(*uvrB-chlA*) *recJ284*::Tn*10 leuB19 deo* (*C2*?) *lacZ53 malB45 rha-5 rpsL151* IN(*rrnD-rrnE*) λ$^-$	SR1675 × P1 SR1159, select Thy$^+$
SR1774	F$^-$ Δ(*uvrB-chlA*) *recN262 leuB19 thyA36 deo*(*C2*?) *lacZ53 malB45 rha-5 rpsL151* IN(*rrnD-rrnE*) λ$^-$	SR1510 × P1 SR248, select Tyr$^+$

[a] Genotype symbols are those used by Bachmann (2). Strain numbers are those of the Stanford Therapeutic Radiology Department.

[b] Tc^r, Tetracycline resistance; Ap^r, ampicillin resistance. The P1 strain used is a reisolate of P1 *vir* that was obtained from A. J. Clark. Alternative strain designations are shown in parentheses.

[c] These strains are F$^-$ and λ$^-$ and carry *leuB19 thyA36 deo*(*C2*?) *lacZ53 malB45 rha-5 rpsL151* IN(*rrnD-rrnE*) unless otherwise specified.

[d] These strains are F$^-$ and λ$^-$ and carry *argE3 hisG4 leuB6* Δ(*gpt-proA*)*62 thr-1 thi-1 ara-14 galK2 lacY1 mtl-1 xyl-5 tsx-33 rfbD1 mgl-51 kdgK51 rpsL31 supE44 rac* unless otherwise specified. These strains are derivatives of JC7623 and, according to Lloyd and Buckman (15), they are *sbcC*.

with UV light as described previously (35). Survivors were determined by assaying CFU on SMM agar.

Recombination frequencies. The ability of cells to perform genetic recombination was assayed by using conjugational crosses with an Hfr donor as described previously (33).

DNA repair studies. The procedures for determining the ability of cells to perform the postreplication repair of DNA daughter strand gaps and double-strand breaks after UV irradiation have been described (38).

RESULTS

Effect of *recJ* and *recN* mutations on genetic recombination and UV sensitivity in *uvrA recB recC sbcBC* cells. To study the effect of the *recJ* and *recN* mutations on postreplication repair, we used excision repair-deficient cells to avoid complications that might arise as a result of the excision repair process. In the *uvrA recB recC sbcBC* background, in which only the RecF pathway of recombination is in operation, the *recN* mutation caused a slight increase of sensitivity to UV radiation (2.2-fold at 10% survival [D_{10}]). The *recJ* mutation had a much greater effect; the sensitivity increased 40-fold at D_{10} (Fig. 1A). In fact, the effect of a *recJ* mutation was about twice that of a *recF* mutation in this background (Fig. 1A). Similar to their effects on UV radiation sensitization, the *recN*, *recF*, and *recJ* mutations reduced the conjugational recombination proficiency of *uvrA recB recC sbcB* cells to about 12, 0.6, and 0.001%, respectively (data not shown). In general, these results are comparable to those obtained with *uvr*$^+$ cells (8, 11, 18, 22), except that the *recN* mutation had a much smaller effect on genetic recombination in our background than that reported by Lloyd et al. (22).

Effect of *recJ* and *recN* mutations on the UV sensitivity of *uvrB*, *uvrB recB*, and *uvrB recF* cells. To gain further insights about the roles of the *recJ* and *recN* genes in postreplication repair, we examined the effects of the *recJ* and *recN* mutations on a set of strains (*uvrB*, *uvrB recF*, and *uvrB recB*) whose postreplication repair proficiencies have been well characterized (35). In the *uvrB* background, the *recJ* mutation had no effect on UV sensitivity, while the *recN* mutation produced a small increase in sensitivity (1.5-fold at

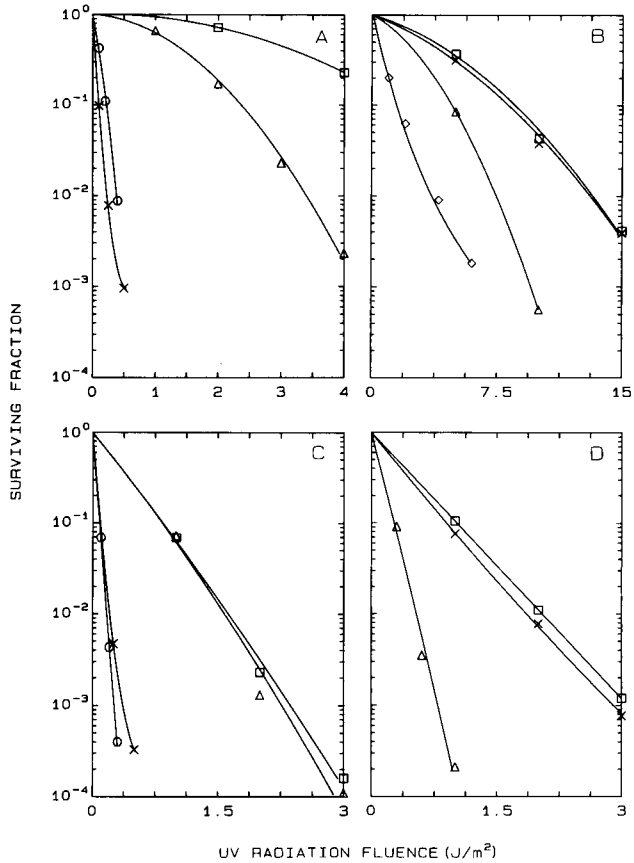

FIG. 1. Effect of *recJ* and *recN* mutations on the UV sensitivity of (A) *uvrA recB recC sbcBC*, (B) *uvrB*, (C) *uvrB recB*, and (D) *uvrB recF* parental strains. CFU were assayed on SMM. Symbols: □, parental strain; Δ, *recN*; ×, *recJ*; ◇, *recJ recN*; ○, *recF*. The data are averages from two experiments.

D_{10}) (Fig. 1B). A strain carrying both the *recJ* and *recN* mutations was considerably more sensitive than the sum of the sensitivities of each singly-mutant strain, indicating that the *recJ* and the *recN* mutations interact synergistically (33). In the *uvrB recB* background, the *recN* mutation had no effect on UV sensitivity, but the *recJ* mutation increased sensitivity eightfold at D_{10} (Fig. 1C). In contrast, the *recJ* mutation had very little effect on the sensitivity of a *uvrB recF* strain, but the *recN* mutation increased the sensitivity fourfold at D_{10} (Fig. 1D). The synergistic action of *recN* and *recF* mutations had been observed by Picksley et al. (22) in *uvr*$^+$ cells. However, the effect of the *recN* mutation was far less than that of the *recB* mutation in *uvrB* and *uvrB recF* strains. Together, these survival studies suggest that the *recJ* and *recN* genes affect different aspects of postreplication repair, with *recJ* affecting *recF*-dependent processes and *recN* affecting *recB*-dependent processes.

Effect of *recJ* and *recN* mutations on DNA repair. To confirm the conclusion that we derived from survival studies, we examined the effect of the *recJ* and *recN* mutations on the repair of UV radiation-induced DNA daughter strand gaps and double-strand breaks. In the *uvrB* background, a single *recJ* or *recN* mutation had essentially no effect on the repair of daughter strand gaps, but the combination of both mutations caused a slight deficiency (Fig. 2A). A *recF* mutation, on the other hand, produced a large deficiency in the repair of daughter strand gaps (Fig. 2A), as reported

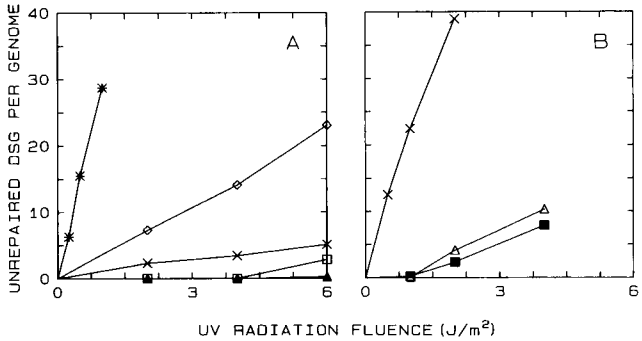

FIG. 2. Effect of *recJ* and *recN* mutations on the repair of DNA daughter strand gaps (DSG) in UV-irradiated *uvrB* (A) and *uvrB recB* (B) cells. Cells were UV irradiated, pulse-labeled with [^{3}H]thymidine at 37°C for 5 min, and incubated in nonradioactive SMM for 2 h. The cells were then converted to spheroplasts, lysed, and sedimented on alkaline sucrose gradients to determine the number of UV-induced DNA single-strand breaks in the nascent DNA (i.e., unrepaired daughter strand gaps), as described (38). (A) Symbols: □, *uvrB* (SR596); ▲, *uvrB recN* (SR1510); ×, *uvrB recJ* (SR1675); ◇, *uvrB recJ recN* (SR1776); *, *uvrB recF* (SR1511). (B) Symbols: ■, *uvrB recB* (SR1518); Δ, *uvrB recB recN* (SR1520); ×, *uvrB recB recJ* (SR1682). The data are averages from two experiments, except for data for *uvrB*, *uvrB recB*, and *uvrB recF* cells, which are from a single experiment and are in agreement with those reported previously (35).

previously (7, 10, 23, 35). In the *uvrB recB* background, the *recJ* mutation produced a major deficiency in the repair of daughter strand gaps, but the *recN* mutation had no effect (Fig. 2B).

We next examined the effect of the *recN* and *recJ* mutations on the repair of double-strand breaks that arise from unrepaired daughter strand gaps in UV-irradiated cells of a *uvrB recF* strain (35). The *recN* mutation produced a partial deficiency in the repair of double-strand breaks, in contrast to the complete deficiency produced by the *recB* mutation (Fig. 3). The partial deficiency in the repair of double-strand breaks in *recN* mutants is in agreement with that observed after ionizing radiation treatment (22, 27). The *recJ* mutation altered the kinetics of both the formation and the repair of double-strand breaks (Fig. 3). The production of double-strand breaks in UV-irradiated *uvrB recF recJ* cells was slower than that in *uvrB recF* cells, and the repair of these double-strand breaks was evident only after 3 h of postirradiation incubation (Fig. 3). Although we could not be certain whether the extent of the repair of double-strand breaks in *uvrB recF recJ* cells would eventually reach the same level as that in *uvrB recF* cells, the fact that the *recJ* mutation had little effect on the UV sensitivity of *uvrB recF* cells (Fig. 1D) suggests that the *recJ* mutation is unlikely to inhibit the repair of DNA double-strand breaks.

In the *uvrA recB recC sbcBC* background, the repair of both daughter strand gaps and double-strand breaks is dependent on a functional *recF* gene (36). We found that *recJ* is also needed for the repair of both types of lesions in this strain background, whereas *recN* is needed only for the repair of double-strand breaks (data not shown).

DISCUSSION

The *recJ* and *recN* mutations were originally isolated on the basis that they reduced the recombination proficiency of *recB recC sbcBC* cells (4, 9, 22). Since recombination is an important part of DNA repair, it seemed likely that these

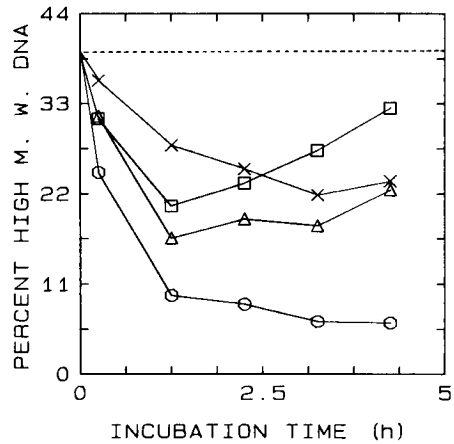

FIG. 3. Effect of the *recN* mutation on the postreplicational formation and repair of DNA double-strand breaks in UV-irradiated *uvrB recF* cells. Cells were UV irradiated (0.5 J/m^2), pulse-labeled with [^{3}H]thymidine at 37°C for 5 min, and incubated in nonradioactive SMM. At the specified times, the cells were converted to spheroplasts, lysed, and sedimented on neutral sucrose gradients to follow the formation and repair of DNA double-strand breaks, as described previously (38). The ability to repair DNA double-strand breaks was monitored by the ability of cells to reform high-molecular-weight (high M.W.) DNA after a 1.25-h incubation (38). The broken line indicates the values obtained from unirradiated control cells and reflects the maximum repair possible. Symbols: □, *uvrB recF* (SR1511); Δ, *uvrB recF recN* (SR1512); ×, *uvrB recF recJ* (SR1676); ○, *uvrB recF recB* (SR1522). The data are from a representative experiment.

mutations would also affect some aspects of postreplication repair. In this work, we present evidence that the *recJ* and *recN* mutations affect different processes of postreplication repair, based on the following observations. First, the *uvrB recN recJ* strain was more sensitive to UV radiation than either the *uvrB recN* or the *uvrB recJ* strain (Fig. 1B). Second, the *recJ* mutation but not the *recN* mutation increased the UV sensitivity of *uvrB recB* cells (Fig. 1C). In contrast, the *recN* mutation but not the *recJ* mutation increased the UV sensitivity of *uvrB recF* cells (Fig. 1D). These survival data indicate that the *recJ* mutation affects *recF*-dependent repair processes and the *recN* mutation affects *recB*-dependent repair processes. Third, DNA repair studies indicated that the *recN* mutation produced a deficiency in the repair of double-strand breaks but did not affect the repair of daughter strand gaps (Fig. 2 and 3). The involvement of the *recN* gene in the repair of double-strand breaks is consistent with previously published reports of *recN* mutants (22, 27). On the other hand, the *recJ* mutation produced a deficiency in the repair of daughter strand gaps in *uvrB recB* (Fig. 2) and *uvrA recB recC sbcBC* (data not shown) cells. This differential effect of the *recJ* and *recN* mutations in DNA repair is consistent with the conclusions drawn from the survival analyses discussed above.

A different effect of the *recJ* and *recN* mutations on conjugational recombination has been observed in crosses between *lacZ* mutants by monitoring the synthesis of the *lacZ*$^+$ product, β-galactosidase (16). While a mutation at *recB* and any combination of mutations at *recJ*, *recF*, or *recO* reduced the production of β-galactosidase by 10- to 25-fold, a *recN* mutation had no effect either alone or in combination with the other mutations. Similarly, mutations in the *recJ*, *recF*, and *recO* genes decreased the frequency of plasmid recombination, whereas the *recN*, *lexA3*, and *ruv* mutations had no effect (11). Therefore, of the several mutations that are known to reduce the recombination frequency of *recB recC sbcBC* cells, *recJ*, *recF*, and *recO* appear to have more similarities than the others. It is likely that the different effects of these mutations on recombination could also be reflected in DNA repair, as demonstrated in this study on the *recJ* and *recN* mutations.

The effect of a *recN* mutation on recombination and on postreplication repair was very similar to that of a *radB* mutation, which has been mapped near *recN* (26). Recent genetic and molecular analyses indicate that the *recN* and *radB* mutations affect the same allele (27a). The *recN* gene encodes a 62-kilodalton (kDa) protein that is induced to high intracellular levels after treatment with agents that induce SOS responses (6). At present, the biochemical function of the RecN protein remains unknown.

It is interesting that while all available data indicate that the *recJ* gene is involved in *recF*-dependent recombination and repair, a single *recJ* mutation did not increase UV sensitivity or inhibit the repair of daughter strand gaps in *uvrB* cells (Fig. 1 and 2). This is in sharp contrast to the effect of a *recF* mutation (35). Furthermore, while a *recJ* mutation and a *recF* mutation produced a comparable amount of UV sensitization in *uvrB recB* and *uvrA recB recC sbcBC* cells (Fig. 1A and C), a *recJ* mutation produced much less radiation sensitization than did a *recF* mutation in *uvrB recN* cells (compare Fig. 1B and D). Therefore, it appears that a *recJ* mutation produces the most radiation sensitization in *recB* mutants, whether they are *sbcB* or *sbcB*$^+$. The possibility that the *recJ* mutation results in the formation of more double-strand breaks, thereby accounting for its selective sensitization of *recB* mutants, was excluded, since we could not detect the formation of DNA double-strand breaks in UV-irradiated (3 J/m^2) *uvrB recJ* cells (unpublished data). Our DNA repair studies indicate that the selective sensitization to UV radiation of a *recB* strain by a *recJ* mutation is correlated with an inhibition of the repair of daughter strand gaps (Fig. 2B). This raises the very interesting possibility that the repair of daughter strand gaps requires either a functional RecJ protein or a functional RecBCD enzyme, i.e., the RecJ protein may possess an enzymatic activity that is common to one of the several activities possessed by the RecBCD enzyme, and such an activity is crucial for certain steps in the repair of daughter strand gaps. The RecBCD enzyme has been studied extensively and is known to possess ATP-dependent exonuclease, endonuclease, and helicase activities (1, 32). Although the *recJ* gene has been cloned and its product identified as a 53-kDa protein (19), its biochemical function remains unknown. Identification of the enzymatic activity associated with the RecJ protein should test the validity of our hypothesis and should also lead to a better understanding of the putative RecFJO system (16) that may provide an alternative to the RecBCD enzyme for the initiation of recombination.

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