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A minor pathway of postreplication repair in *Escherichia coli* is independent of the *recB, recC* and *recF* genes

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Summary

After ultraviolet (UV) irradiation, an *Escherichia coli* K12 *uvrB5 recB21 recF143* strain (SR1203) was able to perform a limited amount of postreplication repair when incubated in minimal growth medium (MM), but not if incubated in a rich growth medium. Similarly, this strain showed a higher survival after UV irradiation if plated on MM versus rich growth medium (i.e., it showed minimal medium recovery (MMR)). In fact, its survival after UV irradiation on rich growth medium was similar to that of a *uvrB5 recA56* strain, which does not show MMR or postreplication repair. The results obtained with a *uvrB5 recF332*::Tn3 *ΔrecBC* strain and a *uvrB5 recF332*::Tn3 *recB21 recC22* strain were similar to those obtained for strain SR1203, suggesting that the *recB21* and *recF143* alleles are not leaky in strain SR1203. The treatment of UV-irradiated *uvrB5 recB21 recF143* and *uvrB5 recF332*::Tn3 *ΔrecBC* cells with rifampicin for 2 h had no effect on survival or the repair of DNA daughter-strand gaps. Therefore, a pathway of postreplication repair has been demonstrated that is constitutive in nature, is inhibited by postirradiation incubation in rich growth medium, and does not require the *recB, recC* and *recF* gene products, which control the major pathways of postreplication repair.

Ultraviolet (UV)-irradiated *Escherichia coli* K12 *uvrA, uvrB* and *uvrC* cells show higher survival if plated on minimal growth medium rather than on rich growth medium [e.g., yeast extract–nutrient broth (Barfknecht and Smith, 1977; Ganesan and Smith, 1968a; Sharma et al., 1982), or minimal growth medium plus casamino acids (Sharma et al., 1982)], and a greater ability to perform postreplication repair when incubated after UV irradiation in minimal medium versus rich growth medium (Sharma et al., 1982, 1983). This higher survival on minimal medium plates has been referred to as "minimal medium recovery" (MMR) (Ganesan and Smith, 1968b; Smith, 1971). MMR is blocked by additional *recA* or *lexA* mutations, and is partially blocked by *recB, uvrD3* or *recF* mutations, but additional *polA1* or *polA5* mutations have no effect (Sharma et al., 1982).

A new mutation, *mmrA1*, does not alter the UV radiation survival and repair capacity of *uvrA* cells when incubated in minimal medium, but prevents the reduction in survival and repair capacity observed when *uvrA* cells are incubated in rich medium (Sharma et al., 1983). Since the survival and repair capacity of *uvrA* cells in minimal medium appears to be the baseline rather than that observed in rich medium, it may be more appropriate to call this phenomenon "rich medium death" rather than "minimal medium recovery".

There are two major pathways of postrepli-

cation repair (both require a functional *recA* gene) (Rothman et al., 1975; Smith and Meun, 1970; Wang and Smith, 1981); one is dependent on the *recF* gene (repair of DNA daughter-strand gaps), and the other is dependent on the *recB* gene (sister-duplex recombination) (Wang and Smith, 1981, 1983). The suggestion of a third pathway of postreplication repair that is independent of both the *recB* and *recF* genes comes from the observations that there is more repair of DNA daughter-strand gaps in UV-irradiated *ΔuvrB recB21 recF143* cells compared to *ΔuvrB recA56* cells (Wang and Smith, 1983), and more repair in *recB21 recC22 recF143* cells compared to *recA56* cells (Rothman et al., 1975). However, this conclusion of a third pathway would only be valid if the *recB21* and *recF143* alleles are not leaky. Therefore, we have used deletion and insertion mutations of the *recB recC* and *recF* genes, respectively, to test this conclusion. These strains still exhibit postreplication repair. In addition, the present study shows that a UV-irradiated *uvrB recB recC recF* strain exhibits MMR, and the small amount of repair of DNA daughter-strand gaps that remains in this strain is inhibited by rich growth medium, but not by rifampicin. This suggests that a part of the MMR phenomenon is the inhibition, by rich growth medium, of a constitutive pathway of postreplication repair that is independent of the *recB* and *recF* pathways of postreplication repair.

Materials and methods

Bacterial strains, media and cultures

The strains of *E. coli* K12 used in this study are listed in Table 1. Strain AC113, deleted for *recB* and *recC* genes, was provided by A.M. Chaudhury and its construction and properties have been described (Chaudhury and Smith, 1984). The *recF332*::Tn3 strain was obtained from A.J. Clark and the position of insertion of Tn3 mutation has been described (Blanar et al., 1984). Transduction was carried out by the method described by Miller (1972). All strains were checked for bacteriophage P1 lysogeny.

The salts buffer (DTM) contained 40 mM K_2HPO_4, 15 mM KH_2PO_4, 0.41 mM $MgSO_4$, 7.6 mM $(NH_4)_2SO_4$, 1.4 mM sodium citrate, 34 μM $CaCl_2$ and 0.9 μM $FeSO_4$ (Kaplan et al., 1962). The minimal growth medium (MM) was DTM containing 0.4% glucose, 10 μg/ml thymine (if required), 0.5 μg/ml thiamine hydrochloride, and each required amino acid at 1 mM. Minimal plating medium was MM solidified with 1.6% (wt/vol) Difco Noble agar. The rich plating media were CAA plates [Difco Casamino Acids (CAA, vitamin assay grade) added to the minimal plating medium at a final concentration of 2 mg/ml], and YENB plates (Difco yeast extract, 0.75%, and nutrient agar, 2.3%). The rich liquid medium was MM supplemented with CAA at 2 mg/ml (CAA medium). Phosphate buffer (PB) has been described (Wang and Smith, 1981).

Logarithmic-phase cultures were obtained by diluting (100-fold) a fresh overnight culture into MM, and growing by shaking at 37°C to an optical density at 650 nm (OD_{650}) of 0.4 ($\sim 3 \times 10^8$ cells/ml; Zeiss PMQ II spectrophotometer). Cells were harvested by centrifugation ($5000 \times g$ for 8 min), washed with PB, and resuspended in PB at an OD_{650} of 0.1 for survival determination (or in DTM at an OD_{650} of 0.2 for repair determination).

UV irradiation

The UV irradiation (254 nm) conditions have been described (Sharma and Smith, 1983). For survival determination, irradiated and control cell suspensions were diluted in PB and plated on MM, CAA and YENB plates. Incubation was for 1–3 days at 37°C. All experiments were done under yellow lights to prevent photoreactivation.

Measurement of DNA repair

The measurement of the repair of DNA daughter-strand gaps was similar to that previously reported (Sharma and Smith, 1983) except that after pulse labeling, the cultures (control and UV-irradiated) were divided in half, collected separately by filtration and washed with DTM. One sample of each culture was resuspended in MM and one in CAA medium. After shaking for 120 min at 37°C, the cells were converted to spheroplasts and layered on top of each gradient (5–20% sucrose in 0.1 N NaOH, 4.8 ml capped with 0.1 ml of 0.5 N NaOH). The gradients were centrifuged at 20°C at 10000 rpm for 16 h (13000 rpm for 16 h for strain SR772). The methods for processing

TABLE 1
LIST OF STRAINS OF *E. coli* K12 [a]

Strain number	Relevant genotype	Other characteristics	Source, derivation, reference
SR250	*uvrB5*	*thyA36 deo(C2?) leuB19 lacZ53 rha-5 rpsL151*	Youngs and Smith (1976a)
SR256	*uvrB5 recA56*	*deo(C2?) leuB19 metE70 lacZ53 rha-5 rpsL151*	Youngs and Smith (1976a)
SR772	*uvrB5 recA56*	Same as SR256, except also *thyA*	SR256, Spontaneous Tmp^r selection
SR782	*recB21 recC22*	*argE3 his-4 leuB6 proA2 thr-1 ara-14 galK2 lacY1 mtl-1 xyl-5 thi-1 tsx-33 rpsL31 supE44*	A.J. Clark (Strain JC3878)
SR792	+	*argA21 cysC43 lysA22 malA1 mtl-2 xyl-7 thi-1? rpsL104 supE44* λ^r	R. Tuveson (Strain AT713)
SR1203	*uvrB5 recB21 recF143*	*thyA deo(C2?) leuB19 lacZ53 rha-5 rpsL151*	T.V. Wang
SR1367	*recF332*::Tn3	*argE3 his-4 leuB6 proA2 thr-1 tnaA*::Tn10 *thi-1 ara-14 galK2 lacY1 mtl-1 xyl-5 tsx-33 rpsL31 supE44 HK19*^{r} ØX174^s S13^s	A.J. Clark (Strain JC10990)
SR1369	Δ(*thyA recB recC argA*)		Chaudhury and Smith (1984) (Strain AC113)
SR1400	*uvrB5*	*deo(C2?) leuB19 lysA22 metE70 lacZ53 rha-5 rpsL151*	P1vira·SR792×SR250; Thy^+ selection
SR1401	*uvrB5 recF332*::Tn3	Same as SR1400, except also *tnaA*::Tn10 Ap^r	P1vira·SR1367×SR1400; Ap^r selection
SR1403	*uvrB5 recF332*::Tn3 Δ(*thyA recB recC argA*)	*deo(C2?) leuB19 metE70 tnaA*::Tn10 *lacZ53 rha-5 rpsL151*	P1vira·SR1369×SR1401; Lys^+ selection
SR1408	*uvrB5*	*deo(C2?) argA21 leuB19 lysA22 metE70 lacZ53 rha-5 rpsL151*	P1vira·SR792×SR250; Thy^+ selection
SR1409	*uvrB5 recF332*::Tn3	Same as SR1408, except also *tnaA*::Tn10 Ap^r	P1vira·SR1367×SR1408; Ap^r selection
SR1410	*uvrB5 recF332*::Tn3	Same as SR1409, except also *thyA*	SR1409, spontaneous Tmp^r selection
SR1412	*uvrB5 recF332*::Tn3 *recB21 recC22*	*deo(C2?) leuB19 lysA22 metE70 tnaA*::Tn10 *lacZ53 rha-5 rpsL151*	P1vira·SR782×SR1410; Thy^+ Arg^+ selection
SR1414	*uvrB5 recF332*::Tn3 *recB21 recC22*	Same as SR1412, except also *thyA*	SR1412, spontaneous Tmp^r selection

[a] Genotype symbols are those used by Bachmann (1983). All strains are F^- and λ^-. Thy^+, Arg^+ and Lys^+ means that cells no longer require thymine, arginine and lysine, respectively. Ap^r and Tmp^r means that cells became resistant to ampicillin and trimethoprim, respectively.

the gradients (Hamelin et al., 1976) and calculating the data (Youngs and Smith, 1976a, b) have been described.

Results

The effect of various plating media on the UV-radiation sensitivity of logarithmic phase, MM-grown *uvrB5 recB21 recF143* and *uvrB5*

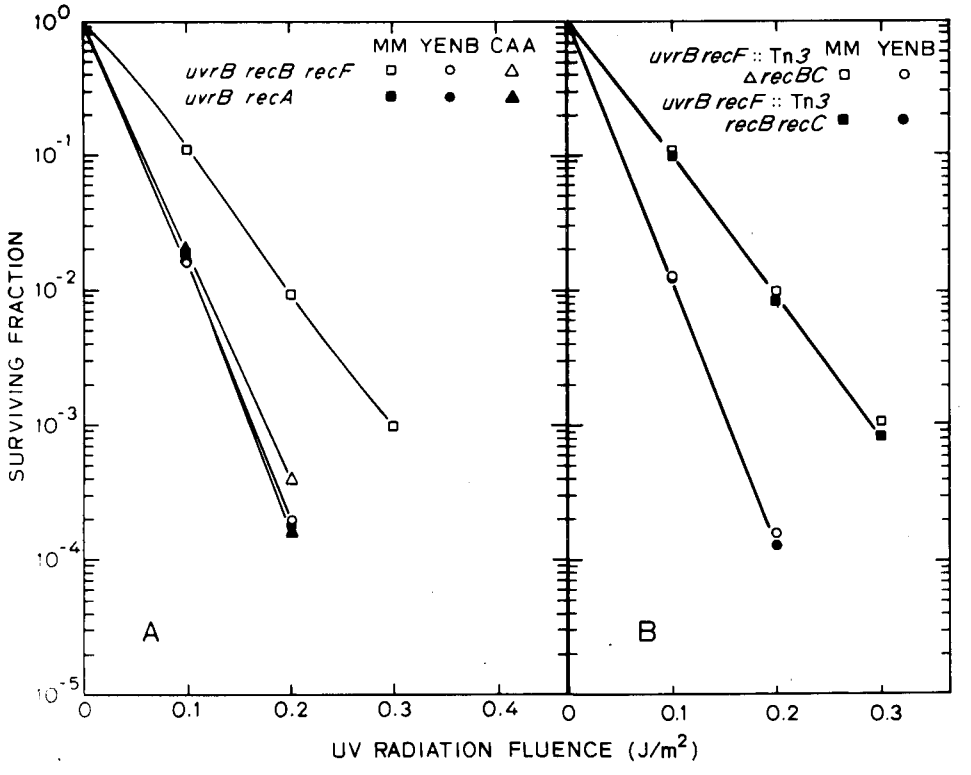

Fig. 1. Effect of various plating media on the survival of UV-irradiated *E. coli* K12 cells. (A) Cells grown in MM to logarithmic phase were UV irradiated (254 nm) in PB, diluted in PB and plated on MM (□, ■), YENB (○, ●) and CAA (△, ▲). Strains: *uvrB5 recB21 recF143* (open symbols), *uvrB5 recA56* (closed symbols). (B) Cells grown in MM were UV-irradiated in PB, diluted in PB and plated on MM (□, ■) and YENB (○, ●). Strain SR1403 (*uvrB5 recF332*::Tn3 Δ*recBC*) (open symbols); strain SR1414 (*uvrB5 recF332*::Tn3 *recB21 recC22*) (closed symbols). Results are the average of 2 Expts.

recA56 cells was investigated (Fig. 1A). The *uvrB5 recB21 recF143* cells showed MMR, and the F_{10} (UV-radiation fluence to yield 10% survival) for YENB- and CAA-plated cells was about 2.0-fold less than it was for MM-plated cells. In confirmation of earlier studies (Ganesan and Smith, 1970; Sharma et al., 1982), *uvrB5 recA56* cells did not show MMR (Fig. 1A); i.e., after UV irradiation the survival of MM-grown cells was the same on MM and rich medium plates (YENB or CAA). The *uvrB5 recB21 recF143* cells were more UV radiation-resistant than the *uvrB5 recA56* cells when plated on MM, however, both strains showed similar UV radiation survival on rich growth media (Fig. 1A).

Rich growth medium (YENB) has been shown to partially inhibit the repair of DNA daughter-strand gaps in *uvrA* and *uvrB* strains of *E. coli* K12 (Sharma et al., 1982, 1983). This observation, and our survival data shown in Fig. 1A, suggest that rich growth medium may inhibit the repair of DNA daughter-strand gaps in UV-irradiated *uvrB5 recB21 recF143* cells, We tested this hypothesis, and as a control, we used *uvrB5 recA56* cells, which show no postreplication repair (Smith and Meun, 1970) and no MMR (Fig. 1A). The *uvrB5 recB21 recF143* cells could repair some of their DNA daughter-strand gaps in MM, but not in rich growth medium (Fig. 2A). The number of unrepaired DNA daughter-strand gaps observed for *uvrB5 recB21 recF143* cells incubated in rich growth medium was about the same as that observed for *uvrB5 recA56* cells, which show no effect of rich growth medium on the repair of DNA daughter-strand gaps (Fig. 2A), or on survival (Fig. 1A).

These data suggest that there is a minor pathway for the repair of DNA daughter-strand gaps that is independent of the *recB* and *recF* genes. However, this conclusion is only valid if the *recB21*

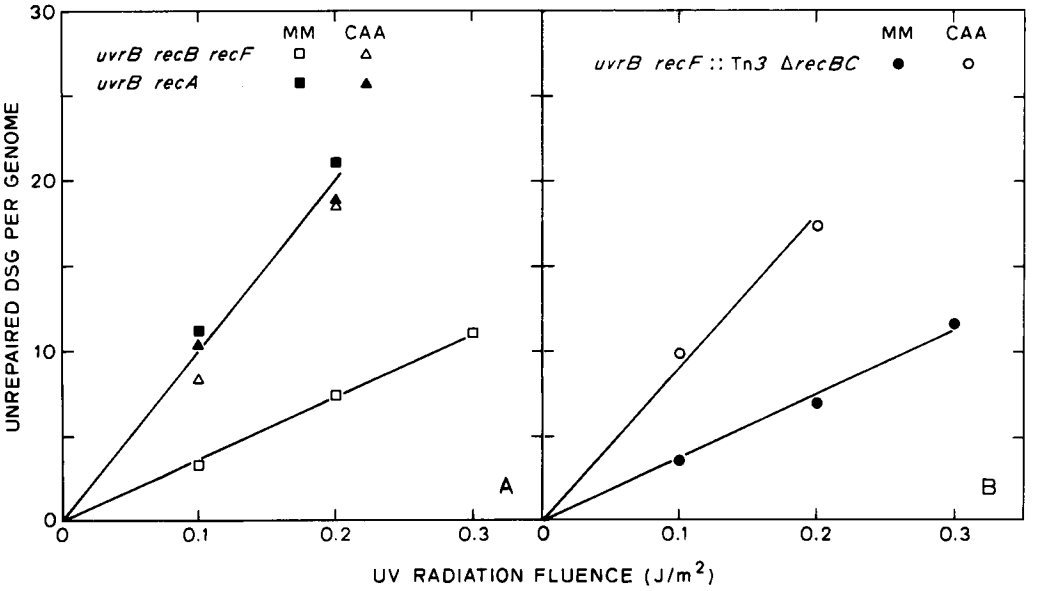

Fig. 2. Effect of postirradiation culture medium on the repair of UV radiation-induced DNA daughter-strand gaps in *E. coli*. UV-irradiated cells were pulse-labeled in MM with [^{3}H]thymidine for 10 min, and shaken in MM (□, ■, ●) or CAA medium (△, ▲, ○) for 120 min at 37°C before being assayed for the number of unrepaired DNA daughter-strand gaps (which is related to the number-average molecular weight, M_n). The M_n values of [^{3}H]DNA after repair incubation (120 min) were used to calculate the average number of unrepaired DNA daughter-strand gaps per genome, which is plotted as a function of UV radiation fluence. (A) strain SR1203 (*uvrB5 recB21 recF143*) (open symbols), strain SR772 (*uvrB5 recA56*) (closed symbols); (B) strain SR1403 (*uvrB5 recF332*::Tn3 Δ*recBC*). Results are the average of data from 2 Expts.

and *recF143* alleles are not leaky. To resolve this problem we constructed a *uvrB5 recF332*::Tn3 Δ*recBC* strain (strain SR1403; for details, see Table 1). Since this strain has a Tn3 insertion in the beginning of the *recF* protein-coding sequence (Blanar et al., 1984) and is deleted for the *recB* and *recC* genes (Chaudhury and Smith, 1984; Dykstra et al., 1984), it should show a null phenotype for these genes. Strain SR1403 shows MMR (Fig. 1B), and its UV radiation sensitivity is about the same as that observed for strain SR1203 (Fig. 1A). Both strains are similar in their ability to carry out the repair of DNA daughter-strand gaps (Fig. 2A,B). We have also constructed a *uvrB5 recF332*::Tn3 *recB21 recC22* mutant (strain SR1414; for details, see Table 1). This strain also shows MMR, and its UV radiation sensitivity (Fig. 1B) is about the same as observed for strains SR1203 (Fig. 1A) and SR1403 (Fig. 1B). These results with the deletion and insertion mutations of the *recB recC* and *recF* genes, respectively, support the conclusion that there is a minor pathway for the repair of DNA daughter-strand gaps that is independent of the *recB recC* and *recF* genes.

In order to determine whether the *recB recC recF*-independent pathway of postreplication repair is inducible or constitutive in nature, we treated UV-irradiated *uvrB5 recB21 recF143* and *uvrB5 recF332*::Tn3 Δ*recBC* cells with rifampicin (RIF) at 25, 50 and 100 μg/ml for 2 h before plating them on MM and rich medium. Under these conditions we observed no increase in OD_{650} of the culture, and no change in colony-forming units per ml of unirradiated cells plated on MM (data not shown). This treatment with RIF had no effect on MMR or UV-radiation sensitivity of these two strains (Fig. 3A,B). Similar results were obtained with chloramphenicol (50 and 100 μg/ml) (data not shown). A much lower concentration of RIF (4 μg/ml) is known to inhibit RecA protein induction (Moreau et al., 1980; Satta et al., 1979; Satta and Pardee, 1978). Therefore, these results suggest that the residual *recB recC recF*-independent postreplication repair does not require induced RecA protein and is constitutive in nature.

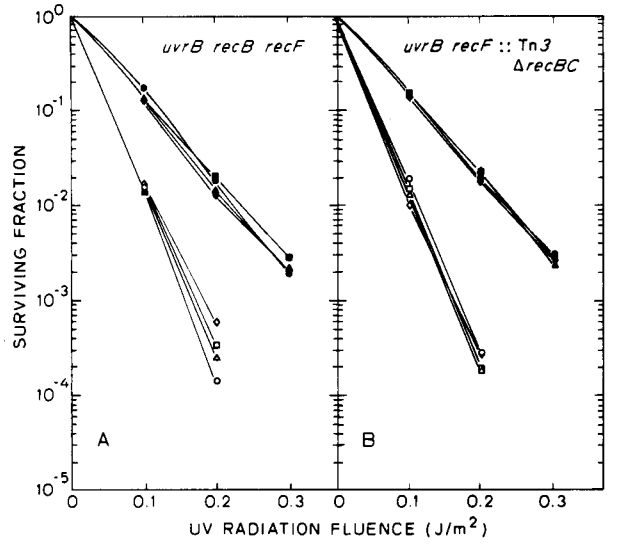

Fig. 3. Effect of rifampicin (RIF) on the survival of UV-irradiated *E. coli* K12 cells. Cells were grown to logarithmic phase in MM, were UV-irradiated in DTM, and were plated on MM (●) and on YENB (○). For RIF treatment, samples of these cells in DTM were immediately diluted 2-fold with MM containing twice the normal concentration of nutritional requirements and RIF, and shaken for 2 h at 37°C before being plated on MM (closed symbols) and on YENB (open symbols). RIF (final concentration) 25 (▲, △); 50 (■, □); or 100 μg/ml (◆, ◇). (A) strain SR1203 (*uvrB5 recB21 recF143*); (B) strain SR1403 (*uvrB5 recF332*::Tn3 Δ*recBC*). All the results in this figure are the average of data from 2 Expts.

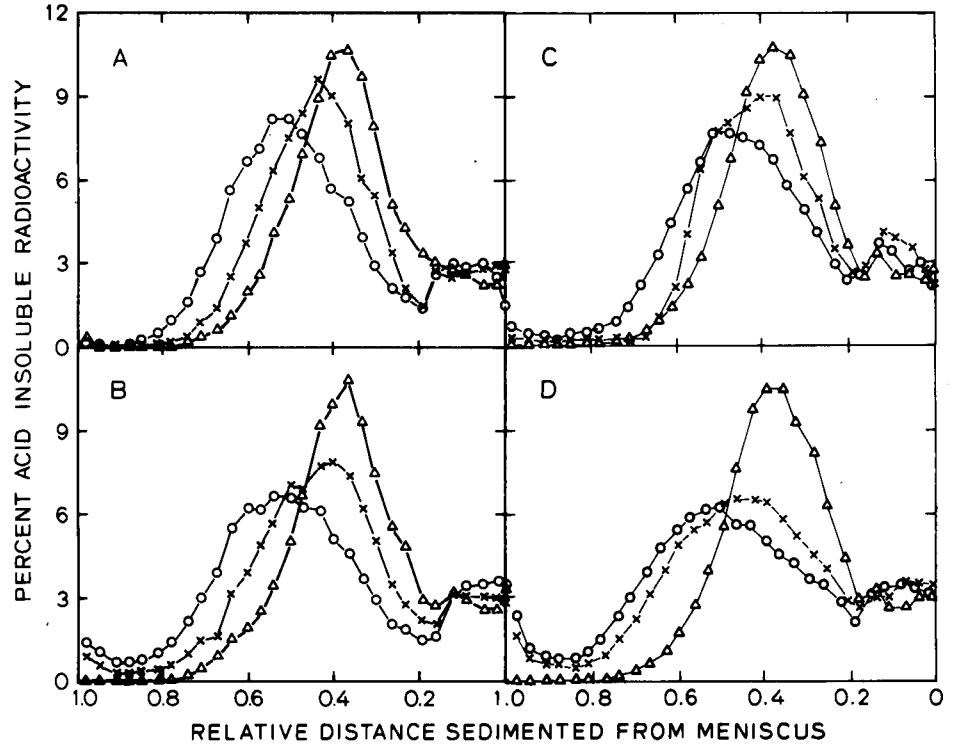

Fig. 4. Effect of rifampicin (RIF) on the repair of DNA daughter-strand gaps in UV-irradiated *E. coli* K12 cells. Cells were grown to logarithmic phase in MM, UV irradiated (0.3 J/m²) in DTM, and pulse-labeled with [³H]thymidine in MM. Immediately after pulse-labeling, cells were resuspended in MM (A, C) or MM + RIF (final concentration, 25 μg/ml for strain SR1203 and 100 μg/ml for strain SR1403) (B, D) medium and shaken at 37°C. Unirradiated cells were shaken for 120 min at 37°C (○). Irradiated cells were shaken for 0 (△), or 120 (×) min before being analyzed. (A, B) strain SR1203 (*uvrB5 recB21 recF143*); (C, D) strain SR1403 (*uvrB5 recF332*::Tn3 Δ*recBC*). The data are from a representative experiment for each strain.

In support of this conclusion, the presence of RIF during repair incubation in MM also had no effect on the amount of DNA daughter-strand gap repair observed in UV-irradiated *uvrB5 recB21 recF143* and *uvrB5 recF332*::Tn3 *ΔrecBC* cells (Fig. 4).

Discussion

Our results confirm the earlier suggestion (Rothman et al., 1975; Wang and Smith, 1983) that there may be a pathway for the repair of DNA daughter-strand gaps that is independent of the *recB* and *recF* genes. This repair system contributes to the higher survival of UV-irradiated *uvrB recB recC recF* cells when they are plated on minimal medium, but not if they are plated on rich growth medium. We have demonstrated minimal medium recovery in *uvrB recB recC recF* cells, and have correlated this phenomenon with the inhibition, by rich growth medium, of the repair of DNA daughter-strand gaps in UV-irradiated cells. We found no effect of rifampicin treatment either on UV-radiation survival or the repair of DNA daughter-strand gaps in *uvrB recB recC recF* cells, suggesting that *recB recC recF*-independent postreplication repair is constitutive in nature, even though it requires a functional *recA* gene. Our experiments using insertion and deletion mutations in the *recF* and *recB recC* genes, respectively, appear to eliminate the possibility that the residual postreplication repair observed in *uvrB recB recF* strains is due to leaky *recB* or *recF* mutations.

It has been reported that T4 endonuclease V-sensitive sites are transferred to the daughter-strands during postreplication repair in a *uvrB recB recF* strain (Wang and Smith, 1984). In this regard at least, the *recF*-independent pathway for the repair of DNA daughter-strand gaps is similar to the *recF*-dependent pathway (Wang and Smith, 1984). It may be that in the absence of a functional *recF* gene product, some other gene product may substitute for it, but does so very poorly. This would be analogous to the situation where DNA polymerase III can substitute, although poorly, for DNA polymerase I in the growth medium-independent repair of X-ray-induced DNA single-strand breaks (Hamelin et al., 1976).

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