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Mechanisms for *recF*-Dependent and *recB*-Dependent Pathways of Postreplication Repair in UV-Irradiated *Escherichia coli uvrB*

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The molecular mechanisms for the *recF*-dependent and *recB*-dependent pathways of postreplication repair were studied by sedimentation analysis of DNA from UV-irradiated *Escherichia coli* cells. When the ability to repair DNA daughter strand gaps was compared, *uvrB recF* cells showed a gross deficiency, whereas *uvrB recB* cells showed only a small deficiency. Nevertheless, the *uvrB recF* cells were able to perform some limited repair of daughter strand gaps compared with a "repairless" *uvrB recA* strain. The introduction of a *recB* mutation into the *uvrB recF* strain greatly increased its UV radiation sensitivity, yet decreased only slightly its ability to repair daughter strand gaps. Kinetic studies of DNA repair with alkaline and neutral sucrose gradients indicated that the accumulation of unrepaired daughter strand gaps led to the formation of low-molecular-weight DNA duplexes (i.e., DNA double-strand breaks were formed). The *uvrB recF* cells were able to regenerate high-molecular-weight DNA from these low-molecular-weight DNA duplexes, whereas the *uvrB recF recB* and *uvrB recA* cells were not. A model for the *recB*-dependent pathway of postreplication repair is presented.

The major "dark" repair system operating in excision repair-deficient cells of *Escherichia coli* K-12 after UV irradiation is postreplication repair (7). The current model for this repair process suggests that gaps are formed in newly synthesized DNA as the replication complex proceeds past UV radiation-induced lesions (e.g., pyrimidine dimers), and that these daughter strand gaps (DSGs) are subsequently filled by a recombination process (6, 15, 16). Based on cell survival studies, two independent pathways for postreplication repair have been proposed (14): one is dependent on the *recF* gene (14), and the other is dependent on the *recB*, *uvrD*, and *lexA* genes (22). In the present work, the molecular mechanisms for these two pathways of postreplication repair were studied by sedimentation analysis, on alkaline and neutral sucrose gradients, of parental DNA and DNA newly synthesized after UV irradiation. Our data confirm that the *recF*-dependent pathway of postreplication repair functions in the filling of DSGs (5, 10, 13) and establish that the *recB*-dependent pathway functions by a mechanism other than the classical repair of DSGs. A model for the *recB*-dependent pathway of postreplication repair is discussed.

MATERIALS AND METHODS

Bacterial strains and media. The bacterial strains used are listed in Table 1. Supplemented minimal medium (SMM) and DTM buffer have been described previously (23).

Irradiation. The source (254 nm) and measurement of fluence rate for UV irradiation have been described (22). For survival determination, cultures were grown exponentially at 37°C in SMM (containing thymine at 2 μg/ml) until they reached an optical density at 650 nm (OD_{650}) of 0.1 to 0.2 (Zeiss PMQ II Spectrophotometer). The cultures were diluted with SMM to an OD_{650} of 0.1 (ca. 10^8 cells per ml) and UV irradiated while in the medium. Survival determination was performed as previously described (22).

Alkaline and neutral sucrose gradient sedimentation experiments. The composition of the alkaline and neutral sucrose gradients (5 to 20%, wt/vol) has been described (18, 25). The gradients were centrifuged at 20°C in a SW50.1 rotor. The sedimentation conditions used were 16 h at 10 krpm for alkaline gradients and 40 h at 3.4 krpm for neutral gradients (12). The gradients were fractionated and processed for assaying radioactivity as described previously (23).

In experiments in which only the extent of repair of DSGs was measured, cells were grown and irradiated as described above. After irradiation, the cell suspensions were incubated at 37°C for 10 min in a shaking water bath before they were pulse-labeled for 5 min with [*methyl*-^{3}H]thymidine (41 Ci/mmol; Amersham Corp.) at 20 μCi/ml. The pulse-labeled cells were harvested on HA membrane filters (0.45 μm pore size; Millipore Corp.), washed with DTM buffer, and suspended in SMM containing thymine at 2 μg/ml. After 2 h of incubation at 37°C, 0.2-ml samples were added to equal volumes of ice-cold lysozyme solution (Worthington Biochemical Corp.) (400 μg/ml in 0.07 M

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

Stanford Radiology no.	Relevant genotype	Source (original designation) or derivation
SR305	Δ(*uvrB-chlA*) *recF143*	D. A. Youngs (DY243)
SR596	Δ(*uvrB-chlA*)	D. A. Youngs (DY241)
SR611	Δ(*uvrB-chlA*) *recB21*	D. A. Youngs (DY265)
SR839	Δ(*uvrB-chlA*) *recA56*	M. Tang
SR840	Δ(*uvrB-chlA*) *recF143 recB21*	M. Tang
SR1160	Δ(*uvrB-chlA*) *recB21*	Strain SR611, Tmp^{rb}

[a] These strains are derivatives of strain KH21, obtained from R. B. Helling. They are F$^-$ and λ$^-$ and carry *leuB19 thyA deo(C2?) lacZ53 malB45 rha-5 rpsL151*, except for strain SR611, which is *thyA*$^+$. Strain SR839 also carries *srlA*::Tn*10* (tetracycline resistance). Genotype symbols are those used by Bachmann and Low (1).

[b] The strain was selected for trimethoprim resistance to obtain a *thyA* mutant.

Tris–0.017 M EDTA, pH 7.6). After 15 min on ice, 0.2 ml of the spheroplast suspension was layered on an alkaline sucrose gradient.

In experiments in which the kinetics of DNA repair were followed, the cells were grown for at least four generations in SMM containing thymine at 2 μg/ml and [2-^{14}C]thymine (54.5 Ci/mol; New England Nuclear Corp.) at 2 μCi/ml. When the culture reached an OD_{650} of 0.2, the cells were harvested by filtration, washed with DTM, and suspended at an OD_{650} of 0.08 in SMM containing thymine at 2 μg/ml. The resuspended cells were incubated at 37°C for 20 to 30 min before being UV irradiated in the medium. After irradiation, the cells were pulse-labeled with [*methyl*-^{3}H]thymidine as described above. After different times of postirradiation incubation at 37°C, samples were treated with lysozyme as described above, and 0.2 ml of the suspensions was layered onto both alkaline and neutral sucrose gradients.

Molecular weight calculations. The number-average molecular weight, M_n, for single-stranded DNA was calculated relative to a ^{14}C-labeled bacteriophage T2 DNA marker as described previously (26). The only modification in the present work was that the calculation of the $1/\Sigma\,[f_i/(d_i)^{2.63}]$ value for the T2 DNA marker was done similarly to that for the *E. coli* DNA, i.e., all of the fractions in the major DNA peak were used in the calculation rather than just the one or two fractions that contained the most DNA (cf. reference 26). The present calculation assumes that the preparation of ^{14}C-labeled bacteriophage T2 is homogeneous and that the gradient profile obtained for its DNA represents what can be obtained experimentally for such a preparation, whereas the previous calculation (26) assumes that only the peak fraction(s) of the gradient profile for T2 DNA represents the intact DNA molecules.

The M_n values of [^{3}H]DNA after 120 min of repair incubation were used to calculate the average number of DNA single-strand breaks (i.e., the number of unrepaired DNA DSGs) per genome, as described by Town et al. (19, 20).

RESULTS

The effect of *recF*, *recB*, and *recA* mutations on the UV radiation sensitivity of *uvrB* cells and on their ability to repair DSGs was determined. The *uvrB recF* and *uvrB recB* strains had comparable UV radiation sensitivities (Fig. 1a), yet they differed tremendously in their ability to repair DNA DSGs (Fig. 1b). Compared with *uvrB* cells, the *uvrB recB* cells were only slightly deficient in the repair of DSGs. In contrast, the *uvrB recF* cells were grossly deficient in the repair of DSGs, in agreement with the results of other workers (5, 10, 13). We did not observe the complete repair of DSGs in our *uvrB recF* cells after UV radiation at 0.5 J/m^2, as reported by Kato (10) for a *uvrA recF* strain. Differences in the bacterial strains or experimental conditions used may be the basis for this discrepancy.

The introduction of a *recB* mutation into the *uvrB recF* strain greatly increased its UV radiation sensitivity (Fig. 1a), yet it caused only a small increase in its deficiency in the repair of DSGs (Fig. 1b). Compared with a "repairless" *uvrB recA* strain, the *uvrB recF* and *uvrB recF recB* cells were still able to perform some limited repair of DSGs.

The *recB* and *recF* mutations have similar effects on the survival of *uvrB* cells after UV irradiation, but only the *recF* mutation has a major inhibitory effect on the repair of DSGs. This suggests that there is a second postreplication repair mechanism in addition to the gap-filling process and that this repair system is dependent on the *recB* gene. To probe the underlying mechanism of this putative *recB*-dependent postreplication repair process, we asked the following questions: (i) what happens to unrepaired DSGs, and (ii) how can the cell generate lesion-free DNA molecules under such circumstances? One possible fate of unrepaired DSGs is that they may be converted to double-strand breaks by an endonucleolytic attack on the single-stranded parental DNA opposite the DSG (23). To test this possibility, we followed the kinetics of DNA repair after UV irradiation by sedimentation analysis, using alkaline and neutral sucrose gradients. For the *uvrB recA* strain, we confirmed the results of earlier work (17) showing that *recA* strains are deficient in the repair of DSGs (data not shown). When samples of the same cells were analyzed on neutral sucrose gradients, it was observed that with increasing times of incubation, an increasing fraction of [^{3}H]DNA sedimented at lower molecular weights, and after 3 h of postirradiation incubation most of the DNA synthesized after UV irradiation sedimented at low molecular

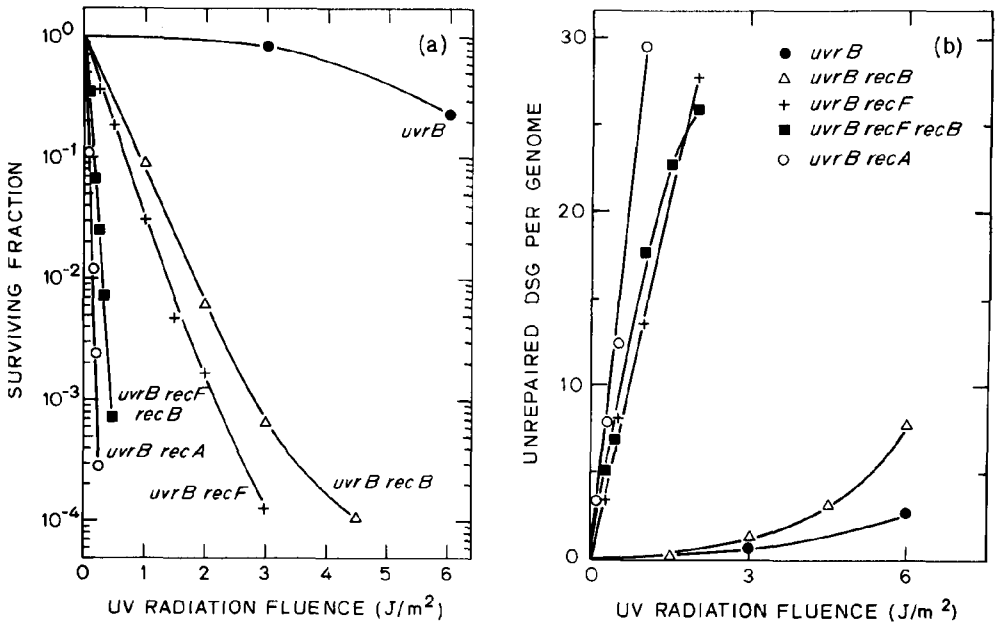

FIG. 1. Effects of *recB*, *recF*, and *recA* mutations on cell survival (a) and on the repair of DSGs (b) in UV-irradiated *uvrB* strains of *E. coli* K-12. Symbols for strains and the M_n values for the unirradiated controls (in daltons): ●, *uvrB* (1.18×10^8); △, *uvrB recB* (1.16×10^8); +, *uvrB recF* (1.4×10^8); ■, *uvrB recF recB* (1.39×10^8); and ○, *uvrB recA* (1.18×10^8).

weights (Fig. 2a). Similar but slower kinetics for the production of double-strand breaks were also evident in the neutral sucrose gradient sedimentation profiles of the parental [^{14}C]DNA (data not shown). The average molecular weight of the [^{3}H]DNA from irradiated cells after 3 h of repair incubation was ca. 5×10^8 daltons. This approximates the size of DNA that would be expected to be synthesized between the dimers (the calculations are based on 56 dimers per genome per J/m^2 [21]). These results suggest that if a DSG is not repaired it can lead to the formation of a DNA double-strand break. We observed no repair of these DNA double-strand breaks in the *uvrB recA* strain (Fig. 2a). The same conclusion was reached from similar studies on a *uvrB recF recB* strain (Fig. 2b).

Next, we asked whether DNA double-strand breaks were produced in UV-irradiated *uvrB recF* cells, and if so, could they be repaired? When assayed on alkaline sucrose gradients, the parental [^{14}C]DNA of UV-irradiated cells (0.25 J/m^2) accumulated single-strand breaks over the 2 h of repair incubation; this accumulation was even more pronounced after 0.5 or 1 J/m^2 of UV radiation (data not shown). When assayed on neutral sucrose gradients, the newly synthesized [^{3}H]DNA of UV-irradiated (0.25 J/m^2) *uvrB recF* cells showed the progressive accumulation of lower-molecular-weight DNA, indicating the formation of DNA double-strand breaks (Fig. 3a). However, unlike the results with *uvrB recA* and *uvrB recF recB* cells (Fig. 2), the [^{3}H]DNA of irradiated *uvrB recF* cells did not remain at the lower molecular weight. After incubation times longer than 1 h, a good fraction of the [^{3}H]DNA began to sediment at higher molecular weights (Fig. 3b). Similar kinetics for the production and repair of double-strand breaks were observed for irradiated parental [^{14}C]DNA (data not shown).

Because of the complex shape of the neutral gradient profiles, it is difficult to quantitate repair as it is done with alkaline gradient profiles (see above). We determined the kinetics of DNA double-strand break production and repair in UV-irradiated *uvrB recF*, *uvrB recF recB*, and *uvrB recA* cells by following over time the amount of DNA in a high-molecular-weight "window" on the DNA sedimentation profile. The window is shown in Fig. 3, and the kinetic data are plotted in Fig. 4. These data show that the addition of a *recB* mutation to a *uvrB recF* strain abolished its ability to repair DNA double-strand breaks.

DISCUSSION

The results of our molecular studies support those of our earlier genetic studies (22) and

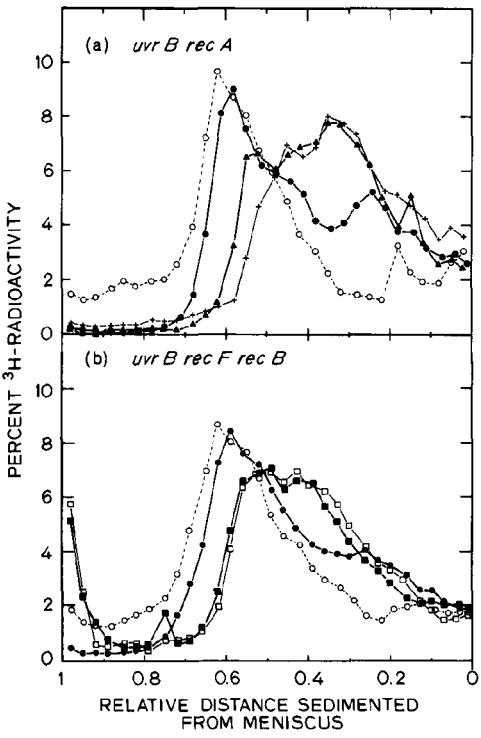

FIG. 2. Kinetics of the formation and repair of DNA double-strand breaks in UV-irradiated *uvrB recA* (a) and *uvrB recF recB* (b) cells of *E. coli* K-12. The [^{14}C]thymine-labeled cells were irradiated with 0.25 J/m^2 and after 10 min of postirradiation incubation at 37°C they were pulse-labeled with [^{3}H]thymidine and treated as described in the text. Only data for the ^{3}H-labeled daughter DNA from neutral sucrose gradients are shown here. Symbols: unirradiated control (○); UV-irradiated samples with no repair incubation (●); irradiated samples incubated for 1 (▲), 2 (□), 3 (+), or 4 (■) h. There was no significant degradation of [^{3}H]DNA (<7%) in UV-irradiated *uvrB recF recB* cells during 4 h of repair incubation, in contrast to irradiated *uvrB recA* cells, which degraded 45% of the [^{3}H]DNA after 3 h of repair incubation.

demonstrate that there is a difference between the *recF*-dependent and *recB*-dependent pathways of postreplication repair. Although both pathways are about equally important in terms of survival in *uvrB* cells (Fig. 1a), the repair of DSGs after 0 to 3 J/m^2 of UV radiation relies largely on the *recF*-dependent pathway (Fig. 1b), suggesting that the *recB*-dependent pathway is involved in other types of repair functions.

Kinetic studies of the fate of DNA newly synthesized after UV irradiation (Fig. 2 and 3) indicated that the accumulation of unrepaired DSGs was followed by the formation of lower-molecular-weight duplex DNA (i.e., double-strand breaks were formed) and that a *recB*-dependent repair function was able to regenerate high-molecular-weight duplex DNA (Fig. 4). The possibility that an artifact of plotting the data as percent ^{3}H radioactivity (Fig. 2 and 3) might contribute to the apparent increase of high-molecular-weight duplex DNA after long incubation times (Fig. 3) was ruled out, since <10% of the DNA was degraded over the 4-h repair period in UV-irradiated (0.25 J/m^2) *uvrB recF* cells (data not shown). A careful examination of the kinetics of regeneration of high-molecular-weight duplex DNA from four separate experiments indicated that this process did

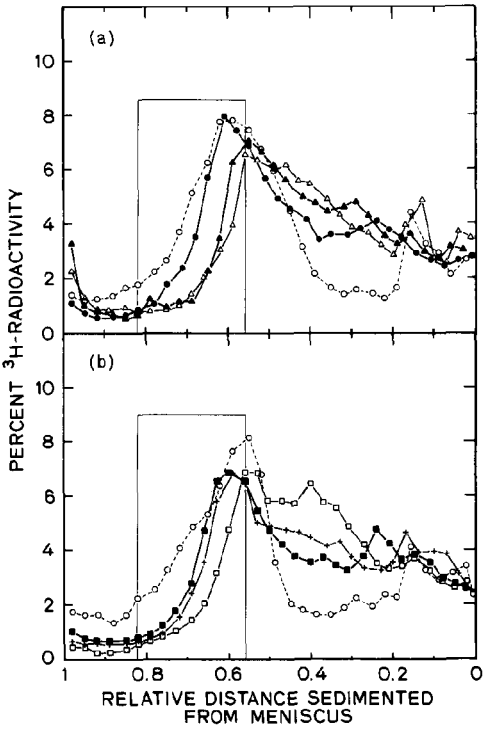

FIG. 3. Kinetics of the formation and repair of DNA double-strand breaks in UV-irradiated *uvrB recF* cells of *E. coli* K-12. Experimental conditions are described in the legend to Fig. 2. Symbols: unirradiated control (○); UV-irradiated (0.25 J/m^2 samples with no repair incubation (●) and with 0.5 (▲), 1 (△) 2 (□), 3 (+) or 4 (■) h of incubation. The data for early repair kinetics of [^{3}H]DNA synthesized after UV irradiation (a) and late repair kinetics (b) are from separate experiments. No significant degradation of [^{3}H]DNA (<10%) was observed for the irradiated cells during the 4 h of repair incubation. The rectangles outline DNA with sedimentation distances between 0.56 and 0.82, which was used to monitor the relative amount of high-molecular-weight double-stranded DNA after different times of repair incubation (see Fig. 4).

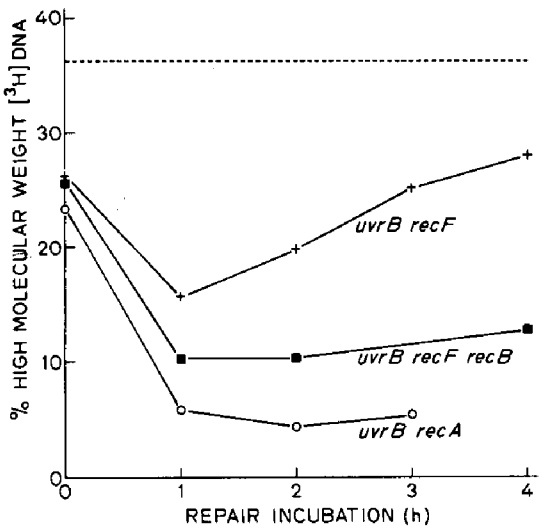

FIG. 4. Kinetics of DNA double-strand break production and repair in UV-irradiated *uvrB* cells of *E. coli* K-12. Experimental conditions are described in the legend to Fig. 2. Data are derived from sedimentation profiles of [^{3}H]DNA on neutral sucrose gradients. The percent high molecular weight [^{3}H]DNA refers to the DNA at a sedimentation distance between 0.56 and 0.82 (see rectangles in Fig. 3). This value was used to monitor the relative amount of high-molecular-weight double-stranded DNA. The data points for strain *uvrB recF* (+) are the averages of four separate experiments. The data points for strain *uvrB recA* (○) and *uvrB recF recB* (■) are from single representative experiments. The average value of unirradiated controls is shown as a dotted line to indicate the maximal repair possible. The decrease in high-molecular-weight DNA observed in the zero-time samples probably occurred during the incubation periods between the UV irradiation and the layering of the spheroplasts on the gradients (see the text).

not involve the simple rejoining of DNA double-strand breaks. Rather, it appeared to involve a redistribution of different sizes of duplex DNA molecules, i.e., an increase in the number of high-molecular-weight DNA molecules was accompanied by an increase in the number of lower-molecular-weight duplex DNA molecules (e.g., compare the 2- and 4-h incubation data in Fig. 3b). One mechanism that can explain this redistribution of molecular weights is a recombination process that involves double-strand exchanges between two DNA molecules. Figure 5 illustrates our model for this recombination process and how it can lead to the production of lesion-free DNA. We suggest that this recombination process is the mechanism for *recB*-dependent postreplication repair. We have termed this *recB*-dependent repair process sister duplex recombination (SDR) to distinguish it from gap-filling repair (GFR).

Based on these observations, we propose that there are two alternate mechanisms for postreplication repair: GFR and SDR. Normally, a DSG formed in UV-irradiated cells is repaired rapidly by the GFR process. Kinetic studies indicate that the filling of DSGs is essentially complete within 60 min of repair incubation at 37°C (17; unpublished data). The direct repair of DSGs by the GFR process provides one means of creating dimer-free DNA by diluting out the dimers through successive rounds of replication and repair (4). However, if a DSG is converted to a DNA double-strand break so that it is no longer repairable by the GFR process, the cell can utilize the alternate SDR mechanism to segregate out the DNA lesions. This SDR process is relatively slow (Fig. 3 and 4), and it requires proper recombinations at homologous regions of two DNA duplexes to produce lesion-free DNA (Fig. 5).

It has been reported that DNA double-strand breaks produced by ionizing radiation can be repaired (12). Our laboratory has recently confirmed this observation and has shown that the

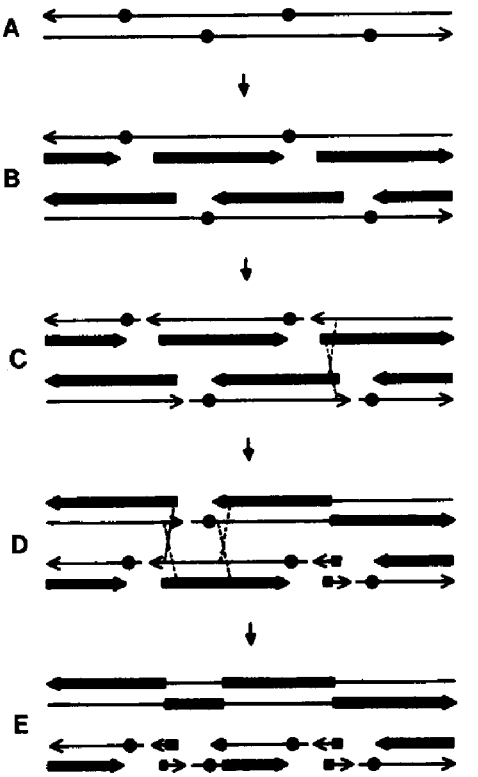

FIG. 5. Model for the recombination repair events of the *recB*-dependent pathway of postreplication repair in *E. coli*. When DNA containing lesions (A), such as pyrimidine dimers (●), is replicated, the newly synthesized DNA (thick lines) is discontinuous opposite each dimer (B). When not repaired, these DSGs can be converted to double-strand breaks (shown here by a random break at the 5′ end of the dimer in the irradiated parental DNA) (C), which initiate reciprocal recombination events at regions of homology between two duplex DNA molecules (C and D). The continuation of this type of recombination can generate intact, lesion-free DNA by segregating out DNA lesions (E).

repair of DNA double-strand breaks requires a functional *recB* gene, but not a functional *recF* gene (Sargentini and Smith, unpublished data). This is consistent with our proposal that the *recB*-dependent pathway of postreplication repair is for the repair of DNA double-strand breaks.

Earlier studies on postreplication repair have focused on GFR. At present, this filling of DSGs is thought to occur by recombination processes (6, 15, 16), although other models are possible (2, 3). In this regard, it is worthy to note that both *uvrB recF* and *uvrB recF recB* cells are able to perform a limited amount of GFR (Fig. 1b). If the *recF143* mutation is not leaky, these results suggest the existence of a *recF*-independent GFR process. Since both *uvrB recF* and *uvrB recF recB* cells were shown to be normal in UV radiation mutagenesis (11), this postulated *recF*-independent GFR process may be mutagenic.

In the cells of higher organisms, the induction of sister chromatid exchanges has been used as a sensitive indicator of DNA damage in proliferating cells, and it has been postulated that sister chromatid exchanges may result from DNA repair processes (8, 9, 24). The SDR process described in this work for *E. coli* cells would produce DNA having the characteristics of sister chromatid exchanges (Fig. 5).

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