JOURNAL OF BACTERIOLOGY, May 1970, p. 404-410
Vol. 102, No. 2

Dark-Recovery Processes in *Escherichia coli* Irradiated with Ultraviolet Light

III. Effect of *rec* Mutations on Recovery of Excision-Deficient Mutants of *Escherichia coli* K-12

ANN K. GANESAN AND KENDRIC C. SMITH
Department of Radiology, Stanford University School of Medicine, Stanford, California 94305

Received for publication 29 December 1969

Mutants of *Escherichia coli* K-12 unable to excise pyrimidine dimers from their deoxyribonucleic acid (DNA) because of a *uvr* mutation show a higher survival when plated on a minimal salts medium after exposure to ultraviolet radiation than when plated on a complex medium such as nutrient agar containing yeast extract. This response has been called minimal medium recovery (MMR). Recovery of *uvr* mutants can take place in liquid as well as on solid medium, but not in buffer or under conditions of amino acid starvation that do not permit cell growth and normal DNA replication. MMR can thus be distinguished from the recovery of recombination-deficient (*rec*$^-$ *uvr*$^+$) derivatives of K-12 which can occur under conditions where growth is not possible. Because MMR is characteristic of excision-defective mutants, it evidently reflects a type of repair independent of excision. We have obtained genetic evidence that MMR is determined by the *rec* genes, which also control recombination in K-12. Cells carrying a *uvr* mutation together with *recA13*, *recA56*, *recB21*, or *recC22* failed to show MMR and were more sensitive to ultraviolet radiation than either their *rec*$^+$ *uvr*$^-$ or *rec*$^-$ *uvr*$^+$ parents. The *rec*$^+$ *uvr*$^-$ derivatives obtained from *recA uvr*$^-$ strains by transduction or by reversion regained the capacity for MMR. Our results indicate that inactivation of any one of the three genes, *recA*, *recB*, or *recC*, prevents cells from showing MMR.

One means by which *Escherichia coli* can recover from the effects of ultraviolet (UV) radiation is to excise pyrimidine dimers produced in its deoxyribonucleic acid (DNA) by irradiation, replacing them with normal DNA synthesized by using the undamaged complementary region of the opposite strand as template (*see* 9, 11, 20, 21 for reviews). The *uvrA*, *uvrB*, and *uvrC* mutants of *E. coli* K-12 are deficient in their capacity for excision. They are thus unable to repair DNA by this mechanism and are more sensitive to UV radiation than corresponding *uvr*$^+$ strains (1, 13). However, such mutants appear to be able to recover from UV damage by some other process. When irradiated and plated on minimal medium, they show a higher survival than when plated on a complex medium, such as nutrient agar containing yeast extract. This response, called minimal medium recovery (MMR), indicates that the cells can recover on minimal medium and that their recovery is inhibited by complex medium (6, 7).

The recombination-deficient (*rec*) mutants of K-12 examined, except *recC22*, showed a similar response (6). In contrast to the *uvr* (*rec*$^+$) mutants, however, the *rec* (*uvr*$^+$) mutants could also recover in buffer or in minimal medium lacking amino acids needed for cell growth. The difference in the conditions required suggested that recovery of the *uvr* mutants reflected a different mechanism of repair than the one underlying the recovery of *rec* mutants (7). In addition, genetic evidence indicated that the recovery of *rec* mutants was inactivated by *uvr* mutations (8). Thus, the recovery shown by the *uvr* mutants must involve a system different from the one responsible for the recovery of *rec* mutants.

The *rec uvr* recombinants which had been tested showed little or no recovery on minimal medium, indicating that the *rec* genes might control the recovery of excision-deficient cells on minimal medium (7). However, we felt that more data relating to this possibility was needed. With the exception of one strain, all of the *rec uvr* derivatives examined had been constructed from the same Hfr *recA* parent. Matings had been

made with several different *thy* F⁻ strains. In each case, Thy⁺ recombinants were selected (8). Thus, it was possible that the lack of recovery in the *recA uvr* isolates from these crosses might have resulted from a mutation in a gene linked to *thy* but different than *recA*. In addition, no recombinants carrying a *recB* or *recC* mutation together with a *uvr* marker had been tested, so that the effect of these genes on recovery was unknown. The experiments described in this paper were designed to provide more information regarding the role of the *rec* genes in the recovery of excision-deficient mutants.

MATERIALS AND METHODS

Most of the procedures have been described previously (6–8).

Strains. The derivatives of *E. coli* used are described in Table 1.

Media. Complex medium included YENB (0.75% Difco yeast extract, 2.3% Difco nutrient agar) and YENB liquid (0.75% Difco yeast extract, 0.8% Difco nutrient broth).

The phosphate-buffered minimal media used have been described previously (6).

L-Amino acids were incorporated to a final concentration of 10^{-3} M: thymine, 10 μg/ml; thiamine, 0.5 μg/ml; and dihydrostreptomycin sulfate, 200 μg/ml.

Irradiation. An unfiltered 25-w General Electric Germicidal lamp was used at a distance of 54 cm from the surface of a platform shaker. Two perforated grills were used to adjust the dose rate to 820 ergs per mm² per min (for doses above 100 ergs/mm²) and 26 ergs per mm² per min (for doses below 100 ergs/mm²) as measured by the photodecomposition of potassium ferrioxalate (10, 17).

Reversion. Selection based on UV resistance was used to obtain Rec⁺ revertants. Single-colony isolates were grown overnight in Penassay broth (Difco Antibiotic Medium 3). Samples of 0.1 ml were spread on YENB agar, incubated for 30 min, and then irradiated with 12 ergs/mm². After 2 days of incubation, colonies were picked and tested for UV sensitivity, recombination, and host-cell reactivation, as previously described (6).

RESULTS

Preliminary experiments. Experiments were undertaken to determine what conditions were suitable for measuring recovery in excision-deficient (*uvr*) derivatives of K-12, and to compare the responses of *uvr* and recombination-deficient (*rec*) mutants under these conditions.

The *uvr* (*rec*⁺) mutants, in contrast to *rec*

TABLE 1. *Escherichia coli K-12 derivatives used*[a]

Designation	Mating type	Relevant genotype	Other markers	Reference
W3110	F⁻		λˢ	
AB1884	F⁻	*uvrC34*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	13
AB1885	F⁻	*uvrB5*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	13
AB1886	F⁻	*uvrA6*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	13
AB2480	?	*recA13 uvrA6*	λˢ *pro thi lac gal* str^r $T6^r$	16
AB2487	F⁻	*recA13*	λˢ *thr leu arg his pro thy ara lac gal mtl xyl* str^r $T6^r$ *thi*	15
AB2498	F⁻	*uvrC34*	λˢ *thr leu arg his pro thy ara lac gal mtl xyl* str^r $T6^r$ *thi*	13
AB2499	F⁻	*uvrB5*	λˢ *thr leu arg his pro thy ara lac gal mtl xyl* str^r $T6^r$ *thi*	13
AB2500	F⁻	*uvrA6*	λˢ *thr leu arg his pro thy ara lac gal mtl xyl* str^r $T6^r$ *thi*	13
JC1569	F⁻	*recA1*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	2
JC2918	F⁻		λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	
JC2926	F⁻	*recA13*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	
JC5088	Hfr	*recA56*	λˢ *thr ilv thi* spm^r	2
JC5489	F⁻	*recC22*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	
JC5495	F⁻	*recA13 recB21*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	24
JC5743	F⁻	*recB21*	λˢ *thr leu arg his pro ara lac gal mtl xyl* str^r $T6^r$ *thi*	
SR58	F⁻	*recA56 uvrB5*	λˢ *thr leu arg his pro (ara lac gal mtl xyl)* str^r $T6^r$ *thi*	
SR80	F⁻	*recC22 uvr B5*	λˢ *thr leu arg his pro (ara lac gal mtl xyl)* str^r $T6^r$ *thi*	
SR87	F⁻	*recB21 uvrB5*	λˢ *thr leu arg his pro (ara lac gal mtl xyl)* str^r $T6^r$ *thi*	

[a] Abbreviations (3, 4, 24): The symbols *arg, his, ilv, leu, pro, try, thi, thr, thy* denote requirements for arginine, histidine, isoleucine and valine, leucine, proline, tryptophan, thiamine, threonine, and thymine, respectively; *ara, gal, lac, mtl,* and *xyl* denote the inability to utilize arabinose, galactose, lactose, mannitol, and xylose, respectively; *T6*, λ, *spm,* and *str* denote response to the phages T6 and λ, and to the antibiotics, spectinomycin and streptomycin (^{r} indicates resistance, ^{s}, sensitivity); *rec* denotes genes affecting genetic recombination and UV sensitivity; *uvr* designates genes affecting host-cell reactivation and UV sensitivity. Markers in parentheses have not been tested, but are inferred from the characteristics of the parent strains.

(*uvr*$^+$) mutants, did not recover in minimal medium lacking amino acids required for cell growth. The irradiated *uvr* cells retained the capacity for recovery under these conditions, but recovery did not take place until they were transferred to minimal growth medium (Fig. 1). When incubated in YENB liquid after irradiation, *uvr* cells did not recover and gradually lost the capacity to recover on minimal medium (Fig. 2). In this respect, their behavior resembled that of *rec* cells recovering in buffer (7). Addition of complex medium to the buffer inhibited the recovery of *rec* cells, and the inhibition became progressively less reversible as the time of exposure to yeast extract increased (7).

Starvation for amino acids before irradiation consistently improved the recovery of *rec* mutants on minimal medium, including that of the *recC22* mutant which had shown very little recovery under other conditions (Table 2). Results with *uvr* strains were more variable (Table 3). The *uvrC* derivatives showed better recovery when starved for amino acids. The *uvrA* derivatives and the *uvrB* mutant, AB1885, showed an increase in the ratio of survivors on minimal medium to survivors on YENB agar after amino acid starvation, but this appeared to be due primarily to decreased survival on YENB agar, rather than to increased survival on minimal medium. The *uvrB* derivative, AB2499, showed slightly better recovery if not starved for amino acids before irradiation. Because of the variability among *uvr* strains, cultures were usually tested for recovery both with and without starvation for amino acids.

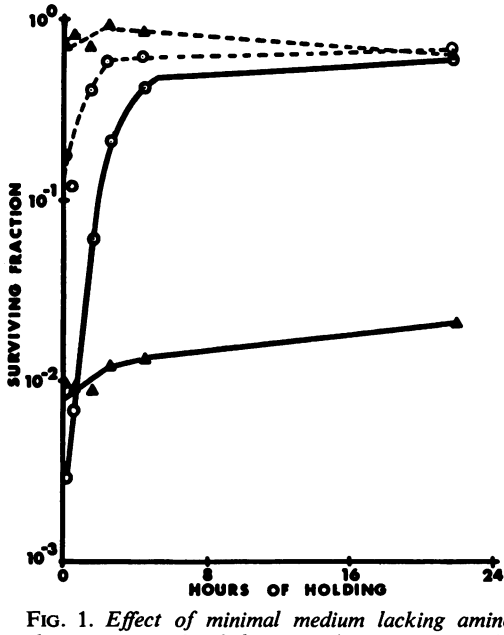

FIG. 1. *Effect of minimal medium lacking amino acids on recovery. Symbols: △, rec$^+$ uvrB5 (AB2499); ⊙, recA13 uvr$^+$ (AB2487). Cells were incubated in minimal medium without amino acids for 2 hr before UV irradiation, and held in this medium at 37 C for various times after irradiation before plating on YENB agar (solid lines) and minimal medium agar (broken lines). AB2487 received 60 ergs/mm^2; AB2499 received 97 ergs/mm^2 at 254 nm.*

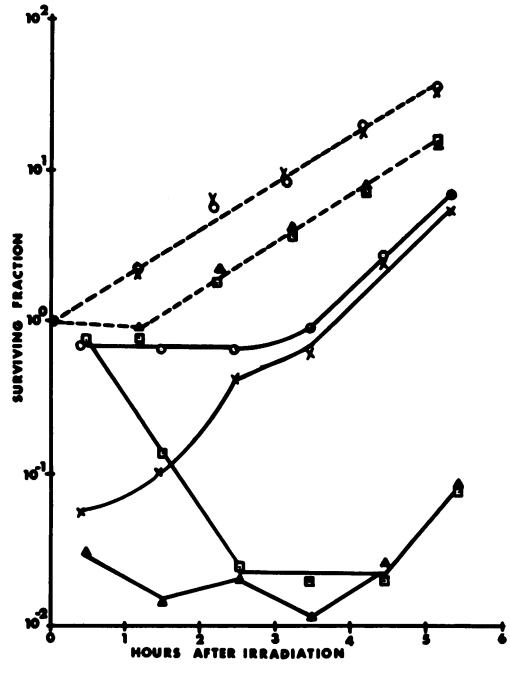

FIG. 2. *Effect of liquid media on recovery of AB2499 (rec$^+$ uvrB5). Cultures growing exponentially in minimal medium were irradiated (72 ergs/mm^2), and then incubated in liquid minimal medium or YENB liquid. At intervals, samples were plated on minimal medium or YENB agar. Solid lines indicate irradiated cultures, broken lines indicate unirradiated controls. Symbols: ⊙, incubated in minimal medium, plated on minimal medium agar; ×, incubated in minimal medium, plated on YENB agar; △, incubated in YENB liquid, plated on YENB agar; ⊡, incubated in YENB liquid, plated on minimal medium agar. The increase in colony-forming units among the irradiated cells after 2.5 hr in minimal medium, and after 3.5 hr in YENB liquid probably reflects growth.*

Experiments with recA derivatives. Two strains, AB2480 (*recA13 uvrA6*) and SR58 (*recA56 uvrB5*), were chosen to study the effect of *recA* mutations on MMR. Neither showed detectable amounts of recovery (6, 7). If the *recA* mutations in these strains were responsible for their failure to show MMR, restoration of the Rec$^+$ character should also restore their ability to recover on minimal medium. We therefore obtained Rec$^+$

TABLE 2. *Effect of amino acid starvation before UV irradiation on survival of rec derivatives of Escherichia coli K-12*[a]

Strain	Preirradiation treatment	UV dose (ergs/mm²)	Surviving fraction		Ratio B/A
			YENB agar (A)	Minimal medium agar (B)	
JC2918 (*rec*⁺)	+AA	1,640	5.0×10^{-3}	4.8×10^{-4}	<1
	−AA		3.1×10^{-3}	1.9×10^{-4}	<1
JC1569 (*recA1*)	+AA	65	2.0×10^{-4}	1.5×10^{-3}	8
	−AA		2.5×10^{-3}	1.7×10^{-1}	68
JC2926 (*recA13*)	+AA	52	5.2×10^{-3}	9.2×10^{-3}	2
	−AA		7.6×10^{-3}	3.9×10^{-1}	51
JC5088 (*recA56*)	+AA	65	4.1×10^{-3}	7.8×10^{-3}	2
	−AA		1.5×10^{-3}	7.6×10^{-2}	51
JC5743 (*recB21*)	+AA	820	8.3×10^{-4}	1.2×10^{-3}	1
	−AA		1.1×10^{-3}	2.5×10^{-2}	23
JC5489 (*recC22*)	+AA	820	4.8×10^{-4}	7.1×10^{-4}	1
	−AA		1.2×10^{-3}	2.4×10^{-2}	20
JC5495 (*recA13 recB21*)	+AA	52	1.1×10^{-3}	5.2×10^{-3}	5
	−AA		6.2×10^{-4}	2.2×10^{-1}	355

[a] Cells were irradiated during exponential growth in minimal medium (+AA) or after 2 hr of incubation in minimal medium lacking amino acids (−AA). Immediately after irradiation, they were plated on YENB agar and on minimal medium agar.

TABLE 3. *Effect of amino acid starvation before UV irradiation on survival of uvr derivatives of Escherichia coli K-12*[a]

Strain	Preirradiation treatment	UV dose (ergs/mm²)	Surviving fraction		Ratio B/A
			YENB agar (A)	Minimal medium agar (B)	
AB1886 (*uvrA6*)	+AA	137	3.6×10^{-4}	6.1×10^{-3}	17
	−AA		1.3×10^{-4}	1.7×10^{-2}	131
AB2500 (*uvrA6*)	+AA	205	1.1×10^{-4}	6.7×10^{-3}	61
	−AA		4.0×10^{-5}	9.9×10^{-3}	248
AB1885 (*uvrB5*)	+AA	205	3.3×10^{-4}	5.3×10^{-4}	2
	−AA		5.8×10^{-5}	1.2×10^{-3}	21
AB2499 (*uvrB5*)	+AA	205	2.1×10^{-3}	2.4×10^{-1}	114
	−AA		2.1×10^{-3}	1.3×10^{-1}	62
AB1884 (*uvrC34*)	+AA	205	5.2×10^{-4}	4.1×10^{-3}	8
	−AA		6.0×10^{-4}	1.1×10^{-1}	183
AB2498 (*uvrC34*)	+AA	273	1.2×10^{-4}	5.7×10^{-4}	5
	−AA		4.6×10^{-4}	1.4×10^{-2}	30

[a] The procedure used was the same as described in Table 2.

derivatives by transduction and reversion and examined them for MMR. Since *rec*$^+$ *uvr*$^-$ cells are more resistant to UV than are *rec*$^-$ *uvr*$^-$ cells, clones were selected by exposure to UV. Survivors were tested for recombination, to verify that they were Rec$^+$, and for host-cell reactivation (Hcr). The loss of the capacity to reactivate irradiated phage is characteristic of *uvr* cells (13) and was used to indicate the presence of a *uvr* mutation. Isolates which behaved as Rec$^+$ Hcr$^-$ were then tested for recovery on minimal medium.

From AB2480, thirteen Rec$^+$ Hcr$^-$ clones were obtained, eight by transduction with phage P1*kc* grown on W3110 (*rec*$^+$ *uvr*$^+$), and five by reversion of independent single-colony isolates. All 13 of the clones showed MMR. From SR58, seven Rec$^+$ Hcr$^-$ clones were obtained, all by reversion of independent single-colony isolates. All of them recovered on minimal medium. In each case the restoration of the capacity for recombination and the increase in resistance to UV associated with it were accompanied by the acquisition of the ability to recover on minimal medium. From these results, we conclude that a functional *recA* gene is necessary for MMR in excision-deficient strains.

Experiments with recB and recC derivatives. To determine whether active *recB* and *recC* genes were also needed for MMR, we constructed strains carrying *uvrB5* together with *recB21* or *recC22*. Lysates of a clear plaque-forming mutant of P1*kc*, generously provided by N. Franklin, were grown on JC5743 (*recB21 uvr*$^+$ *thy*$^+$) and JC5489 (*recC22 uvr*$^+$ *thy*$^+$). These were used to transduce AB2499 (*rec*$^+$ *uvrB5 thy*$^-$). Thy$^+$ transductants were selected and tested for UV sensitivity, recombination, and host-cell reactivation.

Like the *recA uvr* recombinants previously described (8, 12, 16), the *recB21 uvrB5* and *recC22 uvrB5* transductants were more sensitive to UV than any of their parents (Fig. 3, 4). However, they were less sensitive than *recA uvrB5* recombinants (8), as might be expected from the fact that the *recB* and *recC* mutants are less sensitive to UV than are *recA* mutants (2, 5, 15, 24).

Twenty-two *recB21 uvrB5* and 14 *recC22 uvrB5* transductants were tested for MMR. None showed significant amounts of recovery (Fig. 3, 4). Starvation for required amino acids prior to irradiation did not affect the amount of recovery observed (Table 4).

From these data we conclude that functional *recB* and *recC* genes are required for MMR in excision-deficient cells.

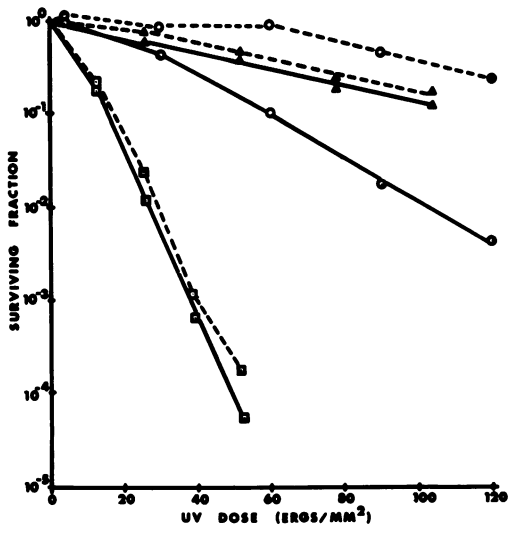

FIG. 3. *Survival of SR87 (recB21 uvrB5) compared to its parents, AB2499 (rec*$^+$ *uvrB5) and JC5743 (recB21 uvr*$^+$*). Cultures growing exponentially in minimal medium were irradiated and plated on YENB agar (solid lines) or minimal medium agar (broken lines). Symbols: ⊙, AB2499, △, JC5743, ⊡, SR87.*

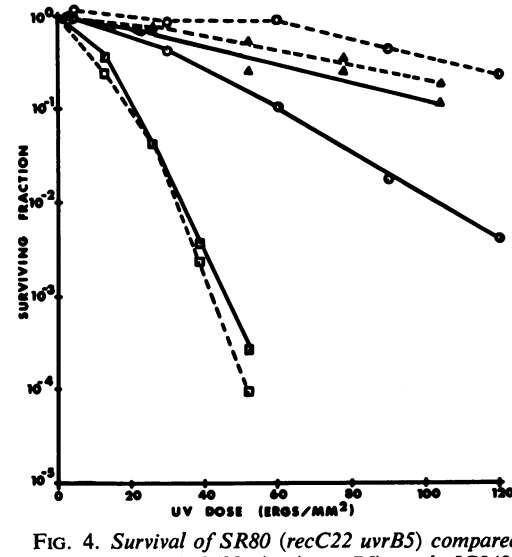

FIG. 4. *Survival of SR80 (recC22 uvrB5) compared to its parents, AB2499 (rec*$^+$ *uvrB5) and JC5489 (recC22 uvr*$^+$*). Conditions were the same as those described in Fig. 3. Symbols: ⊙, AB2499 (same data as in Fig. 3); △, JC5489; ⊡, SR80.*

DISCUSSION

In several respects, the recovery of the *uvrA*, *uvrB*, and *uvrC* mutants of K-12 resembles that of the recombination-deficient derivatives. In both, recovery is observed as a higher survival

TABLE 4. *Effect of amino acid starvation before UV irradiation on survival of rec uvr derivatives of Escherichia coli K-12*[a]

Strain	Preirradiation treatment	UV dose (ergs/mm²)	Surviving fraction		Ratio B/A
			YENB agar (A)	Minimal medium agar (B)	
SR58 (*recA56 uvrB5*)	+AA	3	1.1×10^{-3}	1.8×10^{-3}	2
	−AA		9.1×10^{-4}	9.2×10^{-4}	1
SR87 (*recB21 uvrB5*)	+AA	33	1.4×10^{-3}	2.4×10^{-3}	2
	−AA		4.6×10^{-4}	9.8×10^{-4}	2
SR80 (*recC22 uvrB5*)	+AA	33	2.9×10^{-3}	1.1×10^{-3}	<1
	−AA		1.2×10^{-3}	3.8×10^{-4}	<1

[a] The procedure used was the same as described in Table 2.

of irradiated cells on minimal than on complex medium, indicating that recovery is inhibited by complex medium (6). In both, inhibition by complex medium appears to be incomplete, since the survival on complex medium of cells carrying either a *uvr* or a *rec* mutation alone is higher than that of cells carrying the two together. In both, the inhibition of recovery by complex medium is at least partially irreversible, and the capacity of the irradiated cells to recover when transferred to minimal medium decreases as the time of exposure to complex medium increases. In both, the difference between survival on minimal medium and survival on complex medium is generally enhanced by starvation for amino acids before irradiation.

In spite of their similarities, the recovery of *uvr* mutants can be distinguished physiologically and genetically from that of the *rec* mutants. The *uvr* cells cannot recover during starvation for amino acids after irradiation, whereas *rec* cells can (Fig. 1). This difference implies that distinct metabolic processes underlie recovery in the two types of mutants. Previous studies showed that the introduction of a *uvrA*, *uvrB*, or *uvrC* mutation into a *rec* strain increased its sensitivity to UV and eliminated or reduced the minimal medium effect (7, 12). Similarly, results of the experiments reported in this paper indicate that the introduction of a *recA*, *recB*, or *recC* mutation into a strain carrying a *uvr* marker increased its sensitivity to UV and reduced its recovery on minimal medium. From these results it appears that the recovery of *rec* derivatives depends upon the activity of the *uvr* genes and, conversely, the recovery of *uvr* derivatives depends upon the activity of the *rec* genes. Because *uvr* mutations impair the cells' capacity to excise pyrimidine dimers (13), the recovery observed in *rec* (*uvr*$^+$) mutants may reflect excision-dependent repair (8).

The biochemical activity of the *rec* genes is largely unknown, and thus the nature of the process underlying recovery of the *uvr* (*rec*$^+$) mutants cannot be clearly inferred. According to the present model for this process, however, DNA synthesized after irradiation contains a discontinuity opposite each of the dimers induced in the irradiated template. The discontinuities are thought to be repaired, and a viable genome reconstructed by some mechanism involving exchanges between sister DNA duplexes (14, 19). Analyses using alkaline sucrose density gradients indicate that *recA* mutants are defective in the repair of discontinuities present in DNA synthesized after UV irradiation (Smith and Ganesan, 1969, Biophys. J., A20; Smith and Meun, *submitted for publication*). Similar defects have not been detected in *recB* or *recC* mutants. However, *recB* and *recC* mutants are more resistant to UV than are *recA* mutants (2, 5, 15, 24). Similarly, recombinants containing a *recB* or *recC* mutation together with a *uvr* marker are more resistant to UV than are equivalent *recA uvr* isolates, and thus seem capable of repairing some of the lesions induced by UV. The *recB* and *recC* mutants also retain some capacity for recombination, as indicated by the integration into *rec* recipients of genetic markers from Hfr donors (18) and by chromosome mobilization in F′ *rec* strains (23). The failure to detect a deficiency in the repair of discontinuities in DNA synthesized after UV irradiation in *recB* and *recC* mutants may be due to their residual capacity for repair. Alternatively, these mutations may affect some subsequent step in repair. In either case, the activities of the *recB* and *recC* genes, as well as of *recA*, appear to be necessary

for recovery of excision-deficient cells of *E. coli* K-12 after UV irradiation.

ACKNOWLEDGMENTS

We are grateful for the technical assistance of John Miller. We thank R. P. Boyce, A. J. Clark, J. Foulds, and S. Linn for supplying bacterial mutants.

This investigation was supported by Public Health Service research grant CA-02896, research program project grant CA-10372, and research career development award CA-03709 (to K.C.S.) from the National Cancer Institute.

LITERATURE CITED

1. Boyce, R. P., and P. Howard-Flanders. 1964. Release of ultraviolet light-induced thymine dimers from DNA in *E. coli* K-12. Proc. Nat. Acad. Sci. U.S.A. 51:293–300.
2. Clark, A. J. 1967. The beginning of a genetic analysis of recombination proficiency. J. Cell. Comp. Physiol. (Suppl. 1) 70:165–180.
3. Curtiss, R. III. 1968. UV-induced genetic recombination in a partially diploid strain of *Escherichia coli*. Genetics 58:9–54.
4. Demerec, M., E. A. Adelberg, A. J. Clark, and P. E. Hartman. 1966. A proposal for a uniform nomenclature in bacterial genetics. Genetics 54:61–76.
5. Emmerson, P. T. 1968. Recombination deficient mutants of *Escherichia coli* K-12 that map between *thyA* and *argA*. Genetics 60:19–30.
6. 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.
7. Ganesan, A. K., and K. C. Smith. 1968. Recovery of recombination deficient mutants of *Escherichia coli* K-12 from ultraviolet irradiation. Cold Spring Harbor Symp. Quant. Biol. 33:235–242.
8. Ganesan, A. K., and K. C. Smith. 1969. Dark recovery processes in *Escherichia coli* irradiated with ultraviolet light. II. Effect of *uvr* genes on liquid holding recovery. J. Bacteriol. 97:1129–1133.
9. Hanawalt, P. C., D. E. Pettijohn, E. C. Pauling, E. F. Brunk, D. W. Smith, L. C. Kanner, and J. L. Couch. 1968. Repair replication of DNA *in vivo*. Cold Spring Harbor Symp. Quant. Biol. 33:187–194.
10. Hatchard, C. G., and C. A. Parker. 1956. A new sensitive chemical actinometer. II. Potassium ferrioxalate as a standard chemical actinometer. Proc. Roy. Soc. (London) A235:518–536.
11. Howard-Flanders, P. 1968. DNA repair. Annu. Rev. Biochem. 37:175–200.
12. Howard-Flanders, P., and R. P. Boyce. 1966. DNA repair and genetic recombination: studies on mutants of *Escherichia coli* defective in these processes. Radiat. Res. Suppl. 6:156–184.
13. Howard-Flanders, P., R. P. Boyce, and L. Theriot. 1966. Three loci in *Escherichia coli* K-12 that control the excision of pyrimidine dimers and certain other mutagen products from DNA. Genetics 53:1119–1136.
14. Howard-Flanders, P., W. D. Rupp, B. M. Wilkins, and R. S. Cole. 1968. DNA replication and recombination after UV irradiation. Cold Spring Harbor Symp. Quant. Biol. 33:195–205.
15. Howard-Flanders, P., and L. Theriot. 1966. Mutants of *Escherichia coli* K-12 defective in DNA repair and in genetic recombination. Genetics 53:1137–1140.
16. Howard-Flanders, P., L. Theriot, and J. B. Stedeford. 1969. Some properties of excision-defective recombination-deficient mutants of *Escherichia coli* K-12. J. Bacteriol. 97:1134–1141.
17. Jagger, J. 1967. Introduction to research in ultraviolet photobiology. Prentice-Hall, Inc., N.J.
18. Low, B. 1968. Formation of merodiploids in matings with a class of *rec*$^-$ recipient strains of *Escherichia coli* K-12. Proc. Nat. Acad. Sci. U.S.A. 60:160–167.
19. 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. 31:291–305.
20. Setlow, R. B. 1967. Repair of DNA. p. 61–62. *In* V. V. Koenigsberger and L. Bosch (ed.), Regulation of nucleic acid and protein biosynthesis. Elsevier, Amsterdam.
21. Strauss, B. S. 1968. DNA repair mechanisms and their relation to mutation and recombination. Curr. Top. Microbiol. Immunol. 44:1–85.
22. Taylor, A. L., and C. H. Trotter. 1967. Revised linkage map of *Escherichia coli*. Bacteriol. Rev. 31:332–353.
23. Wilkins, B. M. 1969. Chromosome transfer from F-*lac*$^+$ strains of *Escherichia coli* K-12 mutant at *recA*, *recB*, or *recC*. J. Bacteriol. 98:599–604.
24. Willets, N. S., and A. J. Clark. 1969. Characteristics of some multiply recombination-deficient strains of *Escherichia coli*. J. Bacteriol. 100:231–239.