JOURNAL OF BACTERIOLOGY, Apr. 1987, p. 1731–1736
0021-9193/87/041731-06$02.00/0

Vol. 169, No. 4

Regulatory Role of *recF* in the SOS Response of *Escherichia coli*: Impaired Induction of SOS Genes by UV Irradiation and Nalidixic Acid in a *recF* Mutant

BRIGITTE THOMS AND WILFRIED WACKERNAGEL*

AG Genetik, Fachbereich Biologie, Universität Oldenburg, D-2900 Oldenburg, Federal Republic of Germany

Received 22 September 1986/Accepted 9 January 1987

We isolated a new *recF* mutant of *Escherichia coli* K-12 by insertion of transposon Tn*5* into the *recF* gene. This *recF400*::Tn*5* allele displayed the same phenotypic characteristics as the classic *recF143* mutation. By using Mu d(Ap *lac*) fusions, the induction of nine SOS genes, including *recA*, *uvrA*, *umuC*, *dinA*, *dinB*, *dinD*, *dinF*, *recN*, and *sulA*, by UV irradiation and nalidixic acid was examined. Induction of eight genes by the two agents was impaired by *recF400*::Tn*5* to different extents. The ninth fused SOS gene, *dinF*, was no longer inducible by UV when combined with *recF400*::Tn*5*. The generally impaired SOS response in *recF* strains did not result from weak induction of *recA* protein synthesis, since a *recA* operator-constitutive mutation did not alleviate the inhibitory effect of the *recF* mutation. The results suggest that *recF* plays a regulatory role in the SOS response. It is proposed that this role is to optimize the signal usage by *recA* protein to become a protease.

In *Escherichia coli*, a set of genes is coordinately expressed when the cells are exposed to agents that damage DNA or block replication (e.g., UV irradiation, mitomycin C, nalidixic acid). Several of these damage-inducible genes have been shown to improve cellular survival by providing increased repair capacity and by transiently retarding cell division (44). Accordingly, these genes have been termed SOS genes. Based on a huge body of genetic and biochemical data (28, 45), it is generally assumed that the SOS response is regulated by the *recA* and *lexA* proteins. The *lexA* protein is the common repressor of the SOS genes. After DNA has been damaged, a signal is generated which reversibly activates *recA* protein to become a specific protease cleaving *lexA* protein and thereby derepressing the SOS genes. The *lexA* and *recA* genes are themselves regulated by the *lexA* repressor.

The SOS-inducing signal has not yet been identified in vivo, but in vitro *recA* protein is activated by single-stranded polynucleotides and nucleoside triphosphates (15). Depending on the SOS-inducing agent, certain gene functions appear to be necessary for derepression of the *recA* gene, which has been most intensively studied as a typical SOS gene (6, 11, 21, 23, 33, 38). The *recBC* genes were shown to be required for induction of *recA* protein synthesis by nalidixic acid but not by UV irradiation, whereas a *recF* mutation impaired efficient induction by UV but not by nalidixic acid. This has led to the proposal that the *recF* and *recBC* genes function in different pathways for the generation of an SOS signal(s) that eventually activates the *recA* protease: *recBC* in a pathway active after nalidixic acid treatment and *recF* in the signal production from UV-damaged DNA (33). Support for this proposal came from the observation that in *recF* strains the UV induction of prophage λ was retarded and the cleavage of λ prophage repressor was impaired (2, 13, 36). The observed effects of *recF* and *recBC* mutations on SOS induction were initially confined to the induction and expression of the *recA* gene, and thus the signal pathway hypothesis was only tentatively applied to the whole SOS system (28, 33).

The *recF* gene has a key function in the *recF* pathway of genetic recombination (22), which is itself regulated in at least some of its components by the *recA-lexA* circuitry (1, 30). *recF* is also involved in DNA repair, which may proceed via recombination processes (19, 22, 37). Recently it was hypothesized that the *recF* gene might have a regulatory function in the *recF* pathway of recombination by determining the expression of *recF* pathway genes (41). To establish more definitely the influence of the *recF* gene on the induction of the SOS response, we isolated a transposon Tn*5* insertion in the *recF* gene and used this putative null allele to examine its effect on the induction of nine SOS genes by UV irradiation and nalidixic acid.

MATERIALS AND METHODS

Bacterial strains. The strains used are listed in Table 1. The fusion strains have the *lacZ* gene fused to various SOS genes by Mu d(Ap *lac*) (10). All transductions were done with P1 *kc*. Since the original GW strains carried the *recA441* mutation (25), which partially suppresses the phenotype of *recF* mutants (43), these strains were made *recA*$^+$. Alleles of *recA* were cotransduced by using *srlC300*::Tn*10* as a marker (16). The presence of the *recA*$^+$ allele in these transductants was monitored by the loss of thermoinducibility of *lacZ* and by decreased survival in a *recF* genetic background (43). The presence of the *recAo281* allele was verified by an increase in UV sensitivity (14). The *recF400*::Tn*5* allele was transduced by selecting for kanamycin resistance and screening for increased UV sensitivity. The *recB21* allele was cotransduced with *argA*$^+$ into strains made *argA*::Tn*10* by transduction (if necessary, strains were first transduced to *argE*$^+$). Strains made *recB21* were identified by increased UV sensitivity. The *recN259*::Mu d(Ap *lac*) fusion from strain SP194 (29) was transduced into the AB1157 genetic background (having a *pro-lac* deletion) after selection of a spontaneous temperature-resistant Mu d prophage in SP194 (9).

SOS induction. Strains were grown in M9-glucose minimal medium at 30°C supplemented with the required amino acids (50 μg/ml each) and thiamine (0.1 μg/ml). Log-phase cultures (10^8 cells per ml) were irradiated at room temperature with UV light (germicidal lamp; Osram HNS10, ozone free) under

* Corresponding author.

TABLE 1. Bacterial strains

Strain	Mu d(Ap *lac*) fusion	Designation and relevant genotype	Source or reference[a]
WA554		W3550; *recF400*::Tn*5*	This work
WA486		AB1157; Δ(*pro-lac*)XIII	P. Howard-Flanders
WA411		JC10241; *srl-300*::Tn*10 recA*$^+$	16
WA484		JC11846; *recAo281 srl-300*::Tn*10 recF143*	A. J. Clark
WA497		AFT228; *argA*::Tn*10*	A. Taylor
WA439		KL186; *recB21*	K. B. Low
WA642		JC10990; *recF332*::Tn*3*	A. J. Clark
WA460		JC9239; *recF143*	A. J. Clark
WA560	*dinA1*	GW1010; *recA441*	25
WA596	*dinA1*	*recA*$^+$	WA560 × WA411
WA568	*dinA1*	*recF400*::Tn*5*	WA596 × WA554
WA611	*dinA1*	*recB21*	WA596 ××× WA439[b]
WA612	*dinA1*	*recB21 recF400*::Tn*5*	WA611 × WA554
WA472	*dinB1*	GW1030; *recA441*	25
WA579	*dinB1*	*recA*$^+$	WA472 × WA411
WA580	*dinB1*	*recF400*::Tn*5*	WA579 × WA554
WA442	*dinD1*	GW1040; *recA441*	25
WA581	*dinD1*	*recA*$^+$	WA442 × WA411
WA582	*dinD1*	*recF400*::Tn*5*	WA581 × WA554
WA600	*dinD1*	*recAo281*	WA442 × WA484
WA601	*dinD1*	*recAo281 recF400*::Tn*5*	WA600 × WA554
WA561	*dinF1*	GW1070; *recA441*	25
WA590	*dinF1*	*recA*$^+$	WA561 × WA411
WA587	*dinF1*	*recF400*::Tn*5*	WA590 × WA554
WA551	*uvrA215*	GW1060; *recA441*	26
WA591	*uvrA215*	*recA*$^+$	WA551 × WA411
WA589	*uvrA215*	*recF400*::Tn*5*	WA591 × WA554
WA613	*uvrA215*	*recB21*	WA591 ××× WA439[b]
WA614	*uvrA215*	*recB21 recF400*::Tn*5*	WA613 × WA554
WA552	*umuC*	GW1104; *recA441*	4
WA597	*umuC*	*recA*$^+$	WA552 × WA411
WA588	*umuC*	*recF400*::Tn*5*	WA597 × WA554
WA574	*sulA*	GC4415; *recA*$^+$	R. d'Ari
WA578	*sulA*	*recF400*::Tn*5*	WA574 × WA554
WA571	*recA*	GC2241; (λp*recA*$^+$ *c*I857)	11
WA577	*recA*	*recF400*::Tn*5*	WA571 × WA554
WA647	*recA*	*recB21*	WA577 ×× WA439[b]
WA648	*recA*	*recB21 recF400*::Tn*5*	WA647 × WA554
WA533	*recN259*	SP194	29
WA645	*recN259*	AB1157; Δ(*pro-lac*)XIII	WA486 × WA533
WA646	*recN259*	*recF400*::Tn*5*	WA645 × WA554

[a] Transductional crosses are described by stating the strain number of the recipient first and the strain number of the cells on which P1 was grown (donor) second.

[b] Multiple-step transductions as described in Materials and Methods.

stirring and at a depth of less than 1 mm. Cells were induced with nalidixic acid by adding a small sample of a concentrated solution (1 mg/ml; Sigma Chemical Co., St. Louis, Mo.) to an M9 log-phase culture to give a final concentration of 10 μg/ml. After the inducing treatments, aeration at 30°C was continued.

Assay of β-galactosidase. Preparation of samples from a culture and determination of β-galactosidase activity were performed as described (24) except that the photometric measurements were done at 29°C in a microcomputer-assisted LKB Ultrospec 4050. This allowed determination of the initial reaction kinetics. Units of β-galactosidase were expressed relative to the optical density at 600 nm (OD_{600}) of a culture: $\Delta OD_{420} \times 1{,}000/\Delta t$ (min) per OD_{600}.

RESULTS

Isolation and characterization of *recF400*::Tn*5*. We chose to isolate an insertion of Tn*5* conferring kanamycin resistance (Km^r) in *recF* to easily combine this *recF* allele with the Mu d(Ap *lac*) fusions of SOS genes. Insertions of Tn*5* into the chromosome of strain W3550 *sup*$^+$ were obtained as described previously (17). About 80,000 colonies resistant to kanamycin were replica plated on complete medium contain-

ing nitrofurantoin (5 μg/ml). Of 15 clones sensitive to nitrofurantoin, 1 displayed a UV sensitivity similar to that of a *recF143* single mutant. The insertional mutation of this strain was localized by a series of transductions with Km^r as the marker (Fig. 1). The low cotransduction frequencies of Km^r with *ilv* and *pyrE* and the high cotransduction frequencies with *dnaA* and *tnaA* are consistent with a Tn*5* insertion in *recF* (3, 22). The Km^r and UV sensitivity were generally cotransduced close to 100%, indicating that translocation of Tn*5* in the course of transduction is a rare event. When the insertion was transferred into strain AB1157, the resulting mutant showed the same UV sensitivity as a *recF143* single mutant (data not shown) (22). The insertion was also transduced into JC7623 *recB21 recC22 sbcB15*, in which the *recF* pathway of recombination and repair is operative. The transductants (31 were tested) showed the extreme UV sensitivity and recombination deficiency in conjugation experiments of a *recB21 recC22 sbcB15 recF143* mutant (data not shown) (22). These results strongly suggested that the Tn*5* insertion was in fact in the *recF* gene, and the mutation was thus termed *recF400*::Tn*5*. This notion was confirmed by the results of a further transduction experiment. The *recF400*::Tn*5* allele was transferred into strain JC10990, having a Tn*3* insertion in the *recF* gene, making the cells ampicillin resistant (5). The 96 Km^r clones obtained had all lost their resistance to ampicillin. This must have resulted from replacement of the *recF332*::Tn*3* allele with the *recF400*::Tn*5* allele.

Induction of SOS operons in *recF* mutants. We used strains with *lacZ* genes fused to different SOS operons by Mu d(Ap *lac*) (10). The production of β-galactosidase in fusion strains after treatments that provoke the SOS response is a relatively precise measure of the derepression of SOS operons and generally correlates well with measurements of specific mRNA or protein synthesis if such data are available (23, 32, 38). For the purpose of this work, in which the relative effects of secondary mutations (*recF*, *recB*, *recAo*) on the induction of nine SOS operons were studied, *lacZ* fusions appeared to be ideally suited.

The UV dose for induction was adjusted to the sensitivity of the strains to give survival rates generally between 0.1 and 0.01, although the efficiency of induction was rather independent of the UV dose in the instances studied (two examples are given in Fig. 2). Nalidixic acid at a concentration of 10 μg/ml was sufficient to block replication and to induce the SOS response (41).

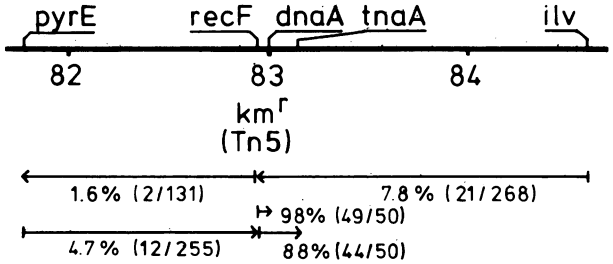

FIG. 1. Phage P1 cotransduction frequencies for mapping of *recF400*::Tn*5*. The ends of the arrows indicate the selected marker, and the heads indicate the cotransduced marker. Numbers in parentheses are the actual number of transductants analyzed. Strains with *pyrE* and *ilv* were auxotrophs; strains with *dnaA* had the temperature-sensitive allele *dnaA46*, eliminating colony formation at 42°C. The *tnaA* mutant had a *tnaA*::Tn*10* insertion; selection for this marker was by tetracycline resistance. The location of these genes is from Bachman (3).

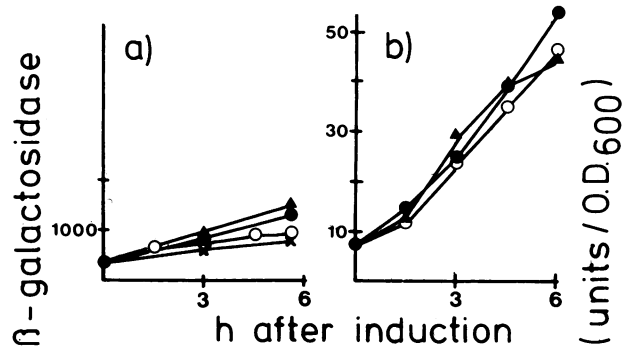

FIG. 2. Induction of β-galactosidase in fusion strains *recA*::Mu d(Ap *lac*)/*recA*$^+$ *recF400* (a) and *dinB1*::Mu d(Ap *lac*) *recF400* (b) by different UV doses. Symbols (UV doses [J/m^2]/cell survival [%]): (a) ▲, 9/10%; ●, 18/1%; ○, 36/0.03%; ×, 54/0.003%. (b) ▲, 9/10%; ●, 27/1%; ○, 54/0.02%.

The nine SOS operons were impaired in their inducibility by the *recF* mutation to different extents (Fig. 3). Several classes were tentatively distinguished. One class consisted of *dinA*, *dinB*, *sulA*, and *umuC*. Induction by UV irradiation or nalidixic acid was slightly reduced, i.e., the response was somewhat delayed or the final extent of derepression was lower. Another class included *dinD*, *recA*, and *uvrA*. Induction by both treatments was very clearly reduced, particularly *recA* induction in response to UV. This latter observation is in accord with published data on the synthesis of *recA* mRNA or protein (33, 38). The *recF* mutation reduced the response to UV irradiation and nalidixic acid of all seven SOS operons plus *recN* (see below). This would argue against the assumption that *recF* is involved in only one route of signal generation, namely the one active after UV damage has occurred (33).

Interestingly, the Mu d-fused *dinF* gene was no longer inducible by UV irradiation in a *recF* mutant, whereas induction by nalidixic acid was still observed. In *dinF*::Mu d/*dinF*$^+$ merodiploids, the UV induction of *dinF* was normal (Table 2), indicating that *dinF*$^+$ is dominant. The presence of F'134 did not affect the response of *dinB* and *uvrA* to UV irradiation and nalidixic acid. This specific response to SOS-inducing treatments is the first reported phenotype of *dinF*. Apparently, the gene is required for its own UV induction in a *recF* genetic background. The slightly higher induction of *dinF* by nalidixic acid in the *recF* strain is puzzling.

The ninth SOS gene studied, *recN*, was reported recently to be uninducible by UV irradiation in a *recF143* mutant (35). We transferred the *recN259*::Mu d(Ap *lac*) allele from strain SP194 into the AB1157 genetic background and observed a clearly reduced but significant induction by UV irradiation and nalidixic acid in the presence of *recF400*::Tn*5*. The UV dose in our experiment was 3 J/m^2 for the double mutant (corresponding to 1% survival), much less than the 20 J/m^2 used previously (35) for this very UV-sensitive strain. This puts *recN* into one group with *dinD*, *recA*, and *uvrA*.

With the exception of *umuC*, the level of β-galactosidase synthesis in uninduced cells was always higher in the presence of the *recF400*::Tn*5* allele. This finding extends the observation of increased *recA* protein synthesis in *recF* mutants (23, 38) to a wide array of SOS genes. The reason for this is not clear. One might speculate that the reduced repair capacity of *recF* mutants results in a slight SOS induction by persistence of spontaneous lesions.

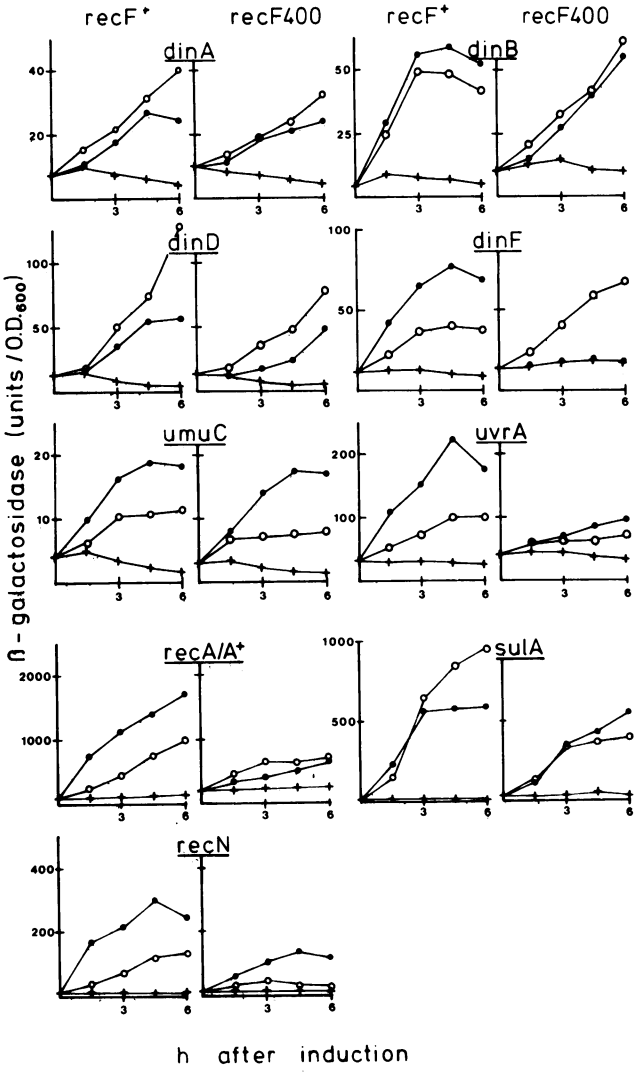

FIG. 3. Induction of β-galactosidase in Mu d(Ap *lac*) fusion strains and their *recF400* derivatives with UV (●) and nalidixic acid (○). Uninduced controls (+) were included. The UV doses (J/m²) were as follows (*recF*⁺/*recF400*): *dinA1*, 54/36; *dinB1*, 54/27; *dinD1*, 54/27; *dinF1*, 54/27; *umuC*, 36/18; *uvrA215*, 3/1.5; *recA/A*⁺, 54/18; *sulA*, 54/18; *recN259*, 54/3.

Reduced SOS response in *recF* strains does not result from low levels of *recA* protein synthesis. Since the induction of *recA* protein synthesis by UV irradiation was reduced in a *recF* mutant (Fig. 3) (11, 33), the low amount of *recA* protein could account for the reduced induction of other SOS genes. To test this possibility, an operator-constitutive *recAo281* mutation (20) was crossed into the *dinD* fusion strain to provide a high level of *recA* protein. The *recAo281* mutation did not alleviate the inhibitory effect of the *recF400*::Tn*5* mutation on induction of *dinD* by UV irradiation and nalidixic acid (Fig. 4). From this result it is concluded that the poor induction of *recA* protein synthesis in a *recF* mutant is not the cause of the limited SOS response. Rather, it appears that this limited response results from an absolutely lower level of *recA* protease than in wild-type cells, even when *recA* protein is overproduced. Our results are consistent with the recent finding that a *recAo* mutation does not affect the pattern of induction of several SOS genes (32). The data are also consonant with the observation that a *recAo* mutation does not suppress the block to UV mutagenesis of φX174 and the decrease in cellular UV resistance caused by *recF143* (14).

Effect of a *recB* mutation. Mutations in *recB* or *recC* abolish the induction of *recA* protein synthesis by nalidixic acid but not by UV irradiation (6, 21). A *recB21* mutation crossed into *dinA* and *uvrA* fusion strains abolished induction by nalidixic acid but left UV induction unaffected (Fig. 4). The additional presence of *recF400*::Tn*5* did not decrease the UV inducibility of both strains beyond the level observed in the respective *recB*⁺ *recF* strains (compare Fig. 3 and 4). This shows that the *recBC* enzyme is required for the induction by nalidixic acid of other SOS genes besides *recA* and confirms that the *recBC* enzyme does not contribute significantly to the SOS induction by UV (6, 33).

It has been reported that a *recF* mutation restores the inducibility of *recA* protein synthesis by nalidixic acid in a *recB* mutant (23). We cannot confirm this observation with our strains. The results in Fig. 4 are in agreement with the suggestion that the *recBC* enzyme is directly involved in the signal generation in nalidixic acid-treated cells (12) and that *recF*⁺ generally improves the SOS response.

DISCUSSION

We isolated a Tn*5* insertion mutation in the *recF* gene. This *recF400*::Tn*5* allele had the same phenotypic characteristics as the classic *recF143* mutation: (i) it made an otherwise wild-type cell moderately UV sensitive, (ii) it caused high UV sensitivity of *recB recC sbcB* cells, and (iii) it made such cells recombination deficient (22). This suggests that *recF* activity is greatly or totally abolished by both mutations.

The *recF400*::Tn*5* allele affected the damage inducibility of the *recA* gene and of the other eight SOS genes tested. Generally, we observed reduced induction by both agents used, UV light and nalidixic acid, although the extent of reduction was different for the various genes. UV light and nalidixic acid have been assumed to trigger the SOS re-

TABLE 2. β-Galactosidase expression in Mu d(Ap *lac*) fusion strains and partial diploids[a]

Strain	β-Galactosidase activity (U/OD$_{600}$) 4 h after induction		
	Uninduced	UV (J/m²)	Nalidixic acid
dinF1::Mu d(Ap *lac*)			
—[b]	10	68 (54)	39
F'134	8	58 (54)	38
recF400	14	15 (18)	54
F'134 *recF400*	8	58 (18)	29
dinB1::Mu d(Ap *lac*)			
—	5	57 (54)	50
F'134	8	63 (54)	54
recF400	10	37 (27)	40
F'134 *recF400*	5	31 (18)	27
uvrA215::Mu d(Ap *lac*)			
—	30	190 (3)	92
F'134	35	168 (54)	93
recF400	40	80 (1.5)	65
F'134 *recF400*	24	85 (18)	53

[a] Partial diploids carry F'134 (31), which covers the auxotrophic markers *argE thi thr leu* of the fusion strains and genes *dinF1*::Mu d(Ap *lac*) and *uvrA215*::Mu d(Ap *lac*), but not *dinB1*::Mu d(Ap *lac*).

[b] —, *recF*⁺, no F'134.

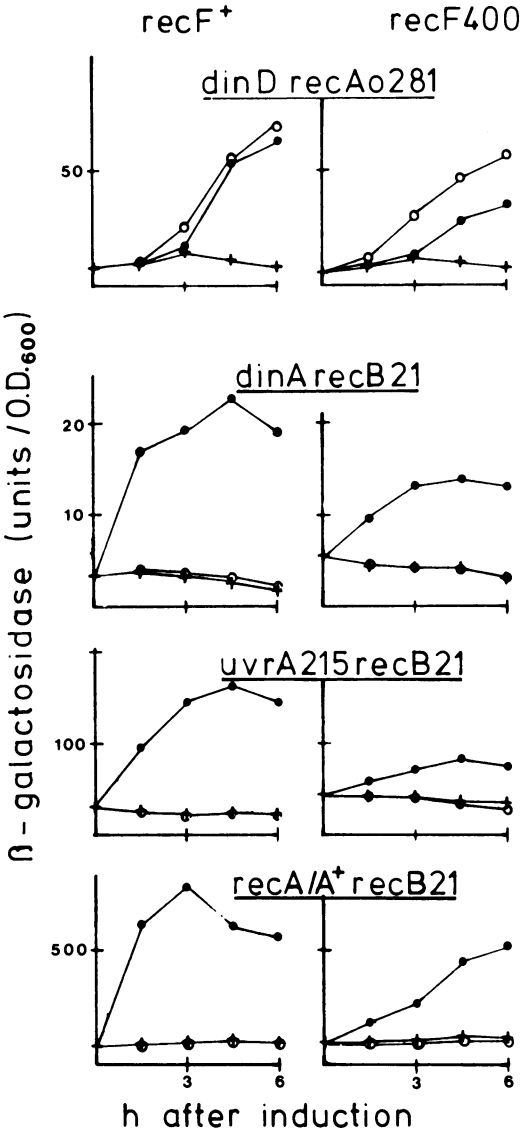

FIG. 4. Effect of a *recAo281* mutation and a *recB21* mutation on the induction of β-galactosidase in Mu d(Ap *lac*) fusion strains and their *recF400* derivatives. Induction was by UV (●), nalidixic acid (○), or uninduced (+). The UV doses (J/m²) were as follows (*recF*⁺/*recF400*): *dinD1 recAo281*, 54/18; *dinA1 recB21*, 18/1; *uvrA215 recB21*, 1.25/0.1; *recA/A*⁺ *recB21*, 9/1.

sponse via different pathways for the generation of the *recA*-activating signal, and *recF* was proposed to act specifically in the signal pathway after UV irradiation (33). Our data indicate that *recF* has a more general function in the signal metabolism. We assume that this function is confined to optimization of the induction process, since the *recF* mutation did not completely block SOS induction. We propose that the *recF* protein is required for maximum use of the SOS signal (produced in cells treated with UV, nalidixic acid, or mitomycin C; unpublished data) by *recA* protein to become a protease. It is conceivable that a physical interaction between *recF* protein and *recA* protein is necessary for optimal signal use by *recA* protein. Close interaction between *recA* and *recF* proteins was suggested by the recent findings that certain mutations in *recA* (*recA441* and *srfA* [42, 43]) partly suppress the need for *recF* for recovery from UV damage and genetic recombination.

Repair of UV damage and recombination during conjugation involve gene functions of the SOS regulon (e.g., *uvrA*, *uvrB*, *recN*, and *ruv* [18, 26, 29, 39]). Thus, the repair-deficient phenotype of *recF* mutants could be at least partly explained by inefficient induction of the SOS system. In support of this view we have shown (manuscript in preparation) that the *recF*-impaired induction of a damage-inducible gene in a *recBC sbcB* strain correlates with the reduced repair capacity of the cells. Clearly our experiments (Fig. 4) and the results of others (14, 32, 40) exclude the possibility that the limited induction of *recA* protein synthesis in *recF* mutants was the only cause of incomplete derepression of the other SOS genes. The requirement of *recF* protein for maximum *recA* protein activation is also consistent with the previous notion (40), based on studies of repair and recombination in strains overproducing *lexA* repressor, that the *recF* gene functions in reducing the level of *lexA* repressor in the cells.

The *recF* gene is essential for genetic recombination in *recBC sbcB* mutants (22), and this recombination depends on gene functions that are inducible by DNA damage (1, 30). The data presented here give no answer to the question of whether *recF* functions in the *recF* pathway of recombination solely by optimizing derepression of the required genes. This possibility was suggested by the defect in recombinational postreplicative repair that was observed (19) in *recF* as well as in *lexA3* mutants (the latter mutation makes the SOS system uninducible). However, our finding (manuscript in preparation) that derepression of the SOS system by a *lexA51* mutation (34) in a *recBC sbcB recF* strain does not restore recombination proficiency strongly argues for a separate role of *recF* in genetic recombination.

The different SOS genes studied here were impaired to various extents in their induction. Provided that the respective gene function is not itself involved in a step of the inducing process (the *recA*⁺ function was supplied in the *recA* fusion strains by an extra copy of the wild-type allele; *dinF* will not be considered) and provided that the assumption of *recF*-mediated maximum signal use by *recA* protein is correct, then the level of reduction of SOS induction in the *recF* mutants would reflect the *lexA* protein operator affinity of the individual operons. Operons more severely affected would require more extensive *recA* protein activation (and therefore lower *lexA* concentrations) for their derepression. Accordingly, *recA* and *dinD*, but possibly also *uvrA* and *recN*, would have strong operators and *dinA*, *dinB*, *sulA*, and *umuDC* less strong operators. Physical binding studies have shown that the *recA* operator binds *lexA* protein more strongly than the *uvrB*, *lexA*, and *sulA* operators (7, 8), and genetic studies indicated a weaker binding of *lexA* protein to operators of *dinA*, *dinB*, *umuDC*, and *uvrA* than to *dinD* (27). Thus, these data, although limited at present, show some convincing congruency. It must be taken into account, however, that derepression kinetics monitored by β-galactosidase synthesis are an indirect measure which encompasses perhaps unknown fine-tuning processes and the influence of promoter strength on the induced gene expression.

The absence of *dinF* induction by UV irradiation in a *recF* mutant demonstrates for the first time a phenotype of *dinF*. It suggests that *dinF* is either involved in the generation of the SOS-inducing signal after UV irradiation or has a function similar to *recF* but limited to UV induction. Further work on the physiological effects of a *dinF* mutation on SOS

induction is required, particularly in the view that *dinF* may be downstream of *lexA* in the same operon (27).

ACKNOWLEDGMENTS

We are grateful to R. d'Ari, B. Bachman, J. Clark, P. Howard-Flanders, R. Lloyd, B. Low, D. Mount, Andrew Taylor, and G. Walker for kindly supplying bacterial strains.

This work was supported by the Deutsche Forschungsgemeinschaft.

LITERATURE CITED

1. **Armengod, M.-E.** 1982. *recF*-dependent recombination as a SOS function. Biochemie **64**:629–632.
2. **Armengod, M.-E., and M. Blanco.** 1978. Influence of the *recF* mutation of *Escherichia coli* K12 on prophage λ induction. Mutat. Res. **52**:37–47.
3. **Bachman, B.** 1983. Linkage map of *Escherichia coli* K-12, edition 7. Microbiol. Rev. **47**:180–230.
4. **Bagg, A., C. J. Kenyon, and G. C. Walker.** 1981. Inducibility of a gene product required for UV and chemical mutagenesis in *Escherichia coli*. Proc. Natl. Acad. Sci. USA **78**:5749–5753.
5. **Blanar, M. A., S. J. Sandler, M.-E. Armengod, L. W. Ream, and A. J. Clark.** 1984. Molecular analysis of the *recF* gene of *Escherichia coli*. Proc. Natl. Acad. Sci. USA **81**:4622–4626.
6. **Bockrath, R. C., and P. C. Hanawalt.** 1980. Ultraviolet light induction of *recA* protein in a *recB uvrB* mutant of *Escherichia coli*. J. Bacteriol. **143**:1025–1028.
7. **Brent, R.** 1982. Regulation and autoregulation by *lexA* protein. Biochimie **64**:565–569.
8. **Brent, R., and M. Ptashne.** 1981. Mechanism of action of the *lexA* gene product. Proc. Natl. Acad. Sci. USA **78**:4204–4208.
9. **Bukhari, A. T.** 1975. Reversal of mutatorphage Mu integration. J. Mol. Biol. **96**:87–99.
10. **Casadaban, M. J., and S. N. Cohen.** 1979. Lactose genes fused to exogenous promotors in one step using a Mu-*lac* bacteriophage: in vivo probe for transcriptional control sequences. Proc. Natl. Acad. Sci. USA **76**:4530–4533.
11. **Casegarola, S., R. d'Ari, and O. Huisman.** 1982. Quantitative evaluation of *recA* gene expression in *Escherichia coli*. Mol. Gen. Genet. **185**:430–439.
12. **Chaudhury, A. M., and G. R. Smith.** 1985. Role of *Escherichia coli recBC* enzyme in SOS induction. Mol. Gen. Genet. **201**: 525–528.
13. **Clark, A. J., M. R. Volkert, and L. J. Margossian.** 1978. A role for *recF* in repair of UV damage to DNA. Cold Spring Harbor Symp. Quant. Biol. **43**:887–892.
14. **Clark, A. J., M. R. Volkert, L. J. Margossian, and H. Nagaishi.** 1982. Effects of a *recA* operator mutation on mutant phenotypes conferred by *lexA* and *recF* mutations. Mutat. Res. **106**:11–26.
15. **Craig, H. L., and J. W. Roberts.** 1981. Function of nucleoside triphosphate and polynucleotide in *Escherichia coli recA* protein-directed cleavage of phage lambda repressor. J. Biol. Chem. **256**:8039–8044.
16. **Csonka, L. N., and A. J. Clark.** 1980. Construction of an Hfr strain useful for transferring *recA* mutations between *Escherichia coli* strains. J. Bacteriol. **143**:529–530.
17. **deBruijn, F. J., and J. R. Lupski.** 1984. The use of transposon Tn5 mutagenesis in the rapid generation of correlated physical and genetic maps of DNA segments cloned into multicopy plasmids—a review. Gene **27**:131–149.
18. **Fogliano, M., and P. F. Schendel.** 1981. Evidence for the inducibility of the *uvrB* operon. Nature (London) **289**:196–198.
19. **Ganesan, A. K., and P. C. Seawell.** 1975. The effect of *lexA* and *recF* mutations on postreplication repair and DNA synthesis in *Escherichia coli* K-12. Mol. Gen. Genet. **141**:189–205.
20. **Ginsburg, H., S. H. Edmiston, J. Harper, and D. W. Mount.** 1982. Isolation and characterization of an operator-constitutive mutation in the *recA* gene of *E. coli* K-12. Mol. Gen. Genet. **187**:4–11.
21. **Gudas, L. J., and A. B. Pardee.** 1976. DNA synthesis inhibition and the induction of protein X in *Escherichia coli*. J. Mol. Biol. **101**:459–477.
22. **Horii, Z.-I., and A. J. Clark.** 1973. Genetic analysis of the *recF* pathway to genetic recombination in *Escherichia coli* K12: isolation and characterization of mutants. J. Mol. Biol. **80**:327–344.
23. **Karu, A. E., and E. D. Belk.** 1982. Induction of *E. coli recA* protein via *recBC* and alternate pathways: quantitation by enzyme-linked immunosorbent assay (ELISA). Mol. Gen. Genet. **185**:275–282.
24. **Kenyon, C. J., R. Brent, M. Ptashne, and G. C. Walker.** 1982. Regulation of damage-inducible genes in *Escherichia coli*. J. Mol. Biol. **160**:445–457.
25. **Kenyon, C. J., and G. C. Walker.** 1980. DNA-damaging agents stimulate gene expression at specific loci in *Escherichia coli*. Proc. Natl. Acad. Sci. USA **77**:2819–2823.
26. **Kenyon, C. J., and G. C. Walker.** 1981. Expression of the *E. coli uvrA* gene is inducible. Nature (London) **289**:808–810.
27. **Krueger, J. H., S. J. Elledge, and G. C. Walker.** 1983. Isolation and characterization of Tn*5* insertion mutations in the *lexA* gene of *Escherichia coli*. J. Bacteriol. **153**:1368–1378.
28. **Little, J. W., and D. W. Mount.** 1982. The SOS regulatory system of *Escherichia coli*. Cell **29**:11–22.
29. **Lloyd, R. G., S. M. Picksley, and C. Prescott.** 1983. Inducible expression of a gene specific to the *recF* pathway for recombination in *Escherichia coli* K12. Mol. Gen. Genet. **190**:162–167.
30. **Lovett, S. T., and A. J. Clark.** 1983. Genetic analysis of the *recF* pathway of recombination in *Escherichia coli* K-12. J. Bacteriol. **153**:1471–1478.
31. **Low, B. K.** 1972. *Escherichia coli* K-12 F-prime factors, old and new. Bacteriol. Rev. **36**:587–607.
32. **Markham, B. E., J. E. Harper, D. W. Mount, G. B. Sancar, A. Sancar, D. W. Rupp, C. J. Kenyon, and G. C. Walker.** 1984. Analysis of mRNA synthesis following induction of the *Escherichia coli* SOS system. J. Mol. Biol. **177**:237–248.
33. **McPartland, A., L. Green, and H. Echols.** 1980. Control of *recA* gene RNA in *E. coli*: regulatory and signal genes. Cell **20**:731–737.
34. **Mount, D. W.** 1977. A mutant of *Escherichia coli* showing constitutive expression of the lysogenic induction and error-prone DNA repair pathways. Proc. Natl. Acad. Sci. USA **74**: 300–304.
35. **Picksley, S. M., R. G. Lloyd, and C. Buckman.** 1984. Genetic analysis and regulation of inducible recombination in *Escherichia coli* K-12. Cold Spring Harbor Symp. Quant. Biol. **49**: 469–474.
36. **Roberts, J. W., and C. W. Roberts.** 1975. Proteolytic cleavage of bacteriophage lambda repressor in induction. Proc. Natl. Acad. Sci. USA **72**:147–151.
37. **Rothman, R. H., and A. J. Clark.** 1977. The dependence of postreplication repair on *uvrB* in a *recF* mutant of *Escherichia coli* K-12. Mol. Gen. Genet. **155**:279–286.
38. **Salles, B., and C. Paoletti.** 1983. Control of UV induction of *recA* protein. Proc. Natl. Acad. Sci. USA **80**:65–69.
39. **Shurvington, C. E., and R. G. Lloyd.** 1982. Damage to DNA induces expression of the *ruv* gene of *Escherichia coli*. Mol. Gen. Genet. **185**:352–355.
40. **Thomas, A., and R. G. Lloyd.** 1983. Control of *recA* dependent activities in *Escherichia coli*: a possible role for the *recF* product. J. Gen. Microbiol. **129**:681–686.
41. **Thoms, B., and W. Wackernagel.** 1984. Genetic control of damage-inducible restriction alleviation in *Escherichia coli* K-12: an SOS function not repressed by *lexA*. Mol. Gen. Genet. **197**:297–303.
42. **Volkert, M. R., and M. A. Hartke.** 1984. Suppression of *Escherichia coli recF* mutations by *recA*-linked *srfA* mutations. J. Bacteriol. **157**:498–506.
43. **Volkert, M. R., L. J. Margossian, and A. J. Clark.** 1984. Two-component suppression of *recF143* by *recA441* in *Escherichia coli* K-12. J. Bacteriol. **160**:702–705.
44. **Walker, G. C.** 1984. Mutagenesis and inducible responses to deoxyribonucleic acid damage in *Escherichia coli*. Microbiol. Rev. **48**:60–93.
45. **Walker, G. C.** 1985. Inducible DNA repair systems. Annu. Rev. Biochem. **54**:425–457.