JOURNAL OF BACTERIOLOGY, June 1978, p. 929-935
0021-9193/78/0134-0929$02.00/0

Vol. 134, No. 3
Printed in U.S.A.

Hyper-Recombination in *Escherichia coli* K-12 Mutants Constitutive for Protein X Synthesis

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Received for publication 7 February 1978

Genetic recombination was studied in *Escherichia coli* F⁻ strains in which synthesis of the *recA* gene product protein X is increased due to mutation in either *recA* (*tif-1*) or *lexA* (*spr*). When a single donor marker was selected, the recombination proficiency of these strains was not significantly altered in Hfr crosses. However, linkage of unselected, proximal Hfr markers was found to be much reduced among the progeny tested, and more of the progeny showed evidence of multiple exchanges between donor and recipient DNA. These effects were much more apparent when the recipient carried both *tif-1* and *spr* mutations, but in this case recombination proficiency was reduced compared with those strains carrying either mutation alone, particularly in crosses with Hfr Cavalli. A *lexA* mutation was found to suppress the effect of *tif-1* on the recombinant genotype.

The study of repair processes in *Escherichia coli* has revealed that damage to DNA initiates what is now frequently referred to as the "SOS" response (25). This involves the coordinate expression of a series of pleiotropic functions which include "error-prone" repair, inhibition of DNA degradation, filamentation, increased synthesis of protein X, and induction of prophage (4, 5, 8, 24, 27, 28). It is generally believed that the SOS response is normally repressed and that the various functions are triggered in response to a common regulatory signal emitted as a consequence of a specific block in DNA replication (10, 25, 28). Expression of SOS functions can be modified by mutation in either of two genes; certain mutations in *recA* or *lexA* prevent their induction, whereas others, such as the *tif-1* mutation (in *recA*) and the *tsl* and *spr* mutations (in *lexA*), allow at least some of these functions to be expressed constitutively (4, 18, 20, 21, 23). Consequently, the products of these two genes are thought to regulate the SOS response (9, 11, 28).

The *recA* gene product has now been identified as protein X, which is synthesized in large amounts in SOS-induced cells and which is almost certainly responsible for the proteolytic cleavage of λ prophage repressor in induced lysogens (9, 17, 26). Mutations in *recA* were first identified in strains that were completely defective for genetic recombination (7), which implies that protein X is also involved in this process. Indeed, in the pathway concept for genetic recombination formulated by A. J. Clark (6), the *recA* gene product is accorded a very fundamental role. Lloyd and Low (13) proposed that the *recA* gene product is involved in the initiation of crossover between homologous DNA molecules. The increased level of *recA* gene product in SOS-induced cells raises the possibility that the potential for recombination may be enhanced. The results given below demonstrate that elevated expression of SOS functions in F⁻ strains dramatically alters the genotype of recombinant progeny recovered from crosses with Hfr donors in a way which suggests that the number of genetic exchanges between Hfr and F⁻ DNA per unit length of chromosome is greatly elevated.

MATERIALS AND METHODS

Strains. The *E. coli* K-12 strains used are listed in Table 1. Gene symbols are as described by Bachmann et al. (2), except that *str* is retained to designate resistance to streptomycin. The *sfiA* mutation prevents filamentation in strains carrying *tif-1* but is not thought to interfere with conjugal recombination (10).

λ⁺ (wild-type) was obtained from Charles Radding. λ*ind* and λ*imm*^{434} were kindly provided by Peter Emmerson. These phages were used to check the prophage induction phenotype of the F⁻ strains, employing the spot lysis procedure described by Mount (20).

Media. Luria broth (LB) and agar have been described (14). Minimal medium was 56/2 salts (14) and was used in liquid form as dilution buffer or solidified with 1.5% (wt/vol) agar (Difco) for plates.

Cultures. Overnight cultures were routinely grown in LB medium from single colonies on LB-agar plates. Since the *thr-1* allele in the F⁻ strains is prone to reversion, care was taken with strains GC3217 and DM1187, which have mutator activity (20), to ensure that only those overnight cultures containing low numbers of spontaneous Thr⁺ revertants were used for inoculating mating cultures.

Matings. Overnight cultures of donor and recipient

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

Strain	Sex	Relevant genotype				Other markers	Source	Reference
		tif	*sfi*	*lex*	*spr*			
AB1157	F^-	+	+	+	+	*thi-1 thr-1 leu-6 proA2 his-4 argE3 lacY1 galK1 ara-14 mtl-1 xyl-5 tsx-33 supE44 str-31*	K. B. Low	1
DM49	F^-	+	+	A3	+	As AB1157	D. W. Mount	22
GC3217	F^-	1	A11	+	+	As AB1157 but *lac*$^+$ *ilv*(Ts) *sup*$^+$	P. T. Emmerson	10
DM1180	F^-	1	A11	A3	+	As GC3217 but *arg*$^+$	P. T. Emmerson	20
DM1420	F^-	+	A11	A3	51	As GC3217 but *arg*$^+$ *his*$^+$	D. W. Mount	20
DM1187	F^-	1	A11	A3	51	As GC3217 but *arg*$^+$	P. T. Emmerson	20
3000	Hfr (Hayes)	+	+	+	+	*thi-1 rel-1*	K. B. Low	1
KL226	Hfr (Cavalli)	+	+	+	+	*rel-1 tonA2* $T2^r$	K. B. Low	1
E5014	F-prime	+	+	+	+	F128 *lac*$^+$ *proAB*$^+$/*thi-1* Δ(*proB-lac*)$_{XIII}$ *mal-24 spc-12 supE50*	J. H. Miller via K. B. Low	

[a] Spontaneous expression of SOS functions is constitutive in DM1187 (20), is elevated in GC3217 at 37°C (J. George, personal communication), but is prevented by *lexA3* (*spr*$^+$) in DM49 and DM1180 (9, 11). Strain DM1420 synthesizes high levels of protein X (P. T. Emmerson, unpublished data) but does not spontaneously express other SOS functions (20). The *sfi* mutation prevents loss of viability due to filamentation in strains expressing SOS functions (10). *sfiA11* is located near *pyrD* (2, 10).

strains were diluted approximately 40-fold into fresh LB medium. The dilutions were shaken at 37°C until the absorbance at 650 nm, as measured with a Bausch and Lomb Spectronic 20 spectrophotometer, reached 0.38 to 0.40 (ca. 2×10^8 cells per ml). Donor and recipient were then mixed in a ratio of 1:5, and incubation continued at 37°C with very gentle agitation. Mating was for 30 min with the F-prime strain E5014, 75 min with Hfr Cavalli, and 85 min with Hfr Hayes, before the mating mixtures were chilled on ice. Samples of suitable dilutions were then pipetted into molten soft agar (0.75% [wt/vol] agar [Difco Laboratories] in distilled water), and mating pairs were separated by the blending procedure of Low and Wood (15) before plating on supplemented minimal agar for the selection of Pro^+, Thr^+, or Gal^+ recombinants in crosses with E5014, Hfr Cavalli, and Hfr Hayes, respectively. Counterselection of the donor cells was achieved with 100 μg of streptomycin sulfate (Sigma Chemical Co.) per ml in the plate agar. Mating mixtures were diluted at least 100-fold before plating for recombinants in order to limit transfer of broth medium and therefore reduce the chance of selecting revertants (27). Each F^- culture grown for mating experiments was tested for reversion of the *thr-1* and *galK1* alleles. Samples of the F^- cells were incubated in parallel with the mating mixtures, and 0.05-ml volumes were then plated, with or without prior dilution, on minimal agar selective for the appropriate revertant class. Transfer of some broth medium with undiluted samples ensured that spontaneous reversion frequencies were not underestimated. Colonies on selective plates were scored after 48 to 72 h at 37°C.

Analysis of recombinant genotype. Single colonies from selective plates were transferred in regular arrays to fresh plates of the same medium, incubated overnight, and then replica-plated (12) onto various media designed to reveal the presence of unselected donor alleles. Selection was always maintained on the replica plates except when the arabinose fermentation characteristic of Gal^+ progeny from the HfrH crosses was being tested, in which case arabinose MacConkey medium was used. Replica plates were scored after 18 h at 37°C, and the phenotype of each recombinant was recorded for analysis.

Failure to grow on LB-agar plates containing 0.2 to 0.5 μg of mitomycin C (Sigma) per ml was used as an indicator of the *lexA* mutation in strains DM49 and DM1180.

RESULTS

To test whether changes in the level of protein X synthesis had any effect on genetic recombination, conjugal crosses were performed between Hfr donors and a number of F^- recipients which differ in their ability to express SOS functions (Table 1). The crosses were analyzed both for the proficiency of recombinant formation and for the genotype of the recombinants generated. The design of the appropriate crosses was complicated by the location of the *lexA* (*spr*), *recA* (*tif*), and *sfiA* genes on the *E. coli* chromosome (2). In crosses with wild-type Hfr strains, some transfer of at least one of these loci to the recipient strains could hardly be avoided if conjugation were to continue long enough for the yield of recombinants to be sufficiently greater than the yield of spontaneous revertants. To limit any undue bias that might consequently have arisen, the recipient strains were crossed both with Hfr Cavalli and Hfr Hayes. Neither of these Hfr's transfers wild-type alleles affecting protein X synthesis particularly early but, more importantly, they have different origins and di-

rections of chromosome transfer (2), factors which have previously been shown to affect the linkage of unselected markers in crosses with recipients altered in respect of *recA* function (13). Secondly, matings were interrupted and selection was made in both cases for donor markers proximal to *lexA*, *recA*, or *sifA*.

Recombination proficiency. Since Hfr alleles transferred to F^- cells are normally inherited with high efficiency, increasing the level of the *recA* gene product was not expected to alter the frequency of recombinant formation to any great extent. The values obtained for the recombination proficiency of the recipient strains (Table 2) were consistent with this. Strains carrying *tif-1* or *spr-51* were found to give slightly greater numbers of recombinants, but strain DM1187, which carries both of these mutations, proved to be somewhat deficient for recombination, particularly in crosses with HfrC. Comparison of the results obtained with strains DM49 and DM1180 revealed that the *lexA3* (*spr*$^+$) mutation reduced recombination proficiency irrespective of whether the recipient carried *tif*$^+$ or *tif-1*.

Genetic analysis of the recombinant progeny. Advantage was taken of the fact that the F^- recipients (Table 1) were multiply marked to examine whether altered SOS expression had any effect on the genotype of the recombinants generated in Hfr crosses. Samples of the progeny selected in each cross were tested for the presence of several unselected, proximal donor markers. The progeny obtained with recipient strains able to synthesize a higher than normal level of protein X (GC3217, DM1420, DM1187) showed reduced linkage of donor alleles (Tables 2 and 3). The genetic constitution of these progeny (Table 3) indicated that genetic exchanges between Hfr and F^- DNA per unit length of chromosome were much more frequent. In particular, there was a large increase in the number of progeny showing evidence of multiple exchanges. This effect, however, was diminished in the HfrH × DM1187 cross, since the majority of the recombinants obtained had failed to inherit any of the unselected donor markers available for analysis. A clearer indication of the increased frequency of genetic exchanges was obtained from examination of those progeny which had inherited an unselected marker and in which at least one further marker located between this and the selected marker was available for analysis. For instance, of the Thr$^+$ Leu$^+$ progeny obtained in the crosses with HfrC, 4.8, 5.0, and 30% were Ara$^-$ when the recipients were DM1420, GC3217, and DM1187, respectively, compared with only 0.6% when the recipient was AB1157. A similar effect was observed among the Gal$^+$ Leu$^+$ progeny of the HfrH crosses. In this instance, the frequencies of Pro$^-$ recombinants were 3, 30, 35, and 57%, respectively, with AB1157, DM1420, GC3217, and DM1187. Table 4 gives a summary of this analysis for all the progeny capable of revealing multiple-exchange events.

Although all the mutant strains used in this work are closely related to AB1157, the more immediate ancestor for GC3217, DM1180,

TABLE 2. *Recombination proficiency of various recipient strains and frequency of unselected Hfr markers*

Recipient strain	Selected progeny (Str^r)	RDI[a]	No. analyzed	Frequency of unselected Hfr markers (%)			
				thr$^+$	*ara*$^+$	*leu*$^+$	*pro*$^+$
(a) × HfrC							
AB1157	Thr$^+$	1	600		92.2	88.7	79.3
DM49[b]	Thr$^+$	0.38	400		89.0	89.0	80.5
GC3217	Thr$^+$	2.4	600		76.0	72.5	59.0
DM1180	Thr$^+$	0.68	400		85.8	85.5	71.3
DM1420	Thr$^+$	1.95	400		78.5	74.0	59.8
DM1187	Thr$^+$	0.097	700		29.7	16.7	25.3
(b) × HfrH							
AB1157	Gal$^+$	1	500	92.0	87.8	88.2	88.5
DM49	Gal$^+$	0.23	300	64.3	64.0	66.3	75.3
GC3217	Gal$^+$	2.0	600	38.2	41.2	44.8	40.7
DM1180	Gal$^+$	0.54	300	51.0	51.7	54.3	60.3
DM1420	Gal$^+$	2.1	400	67.5	60.0	60.2	50.6
DM1187	Gal$^+$	0.73	700	5.7	8.4	10.4	18.8

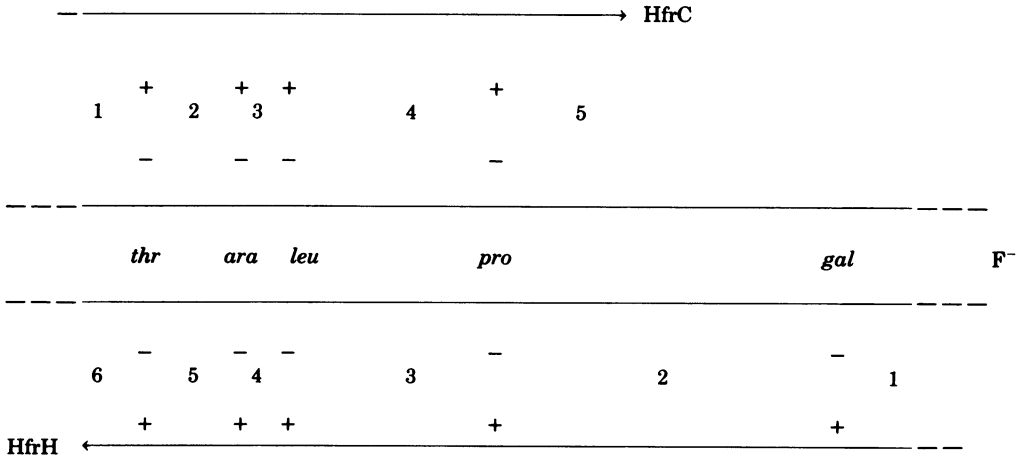
[a] RDI, Recombination deficiency index calculated from progeny in Hfr cross/progeny in F′ cross and expressed as a fraction of that obtained with AB1157.

[b] 15% of the Thr$^+$ recombinants obtained in this cross also inherited *lexA*$^+$, as indicated by their resistance to mitomycin C.

TABLE 3. *Genetic constitution of recombinant progeny*

Crossover regions[a]	Genotype					% of total recombinants in strain:					
	thr	*ara*	*leu*	*pro*	*gal*	AB1157	DM49	GC3217	DM1180	DM1420	DM1187
(a) × HfrC											
1 + 2	+	−	−	−		5.0	9.0	16.3	10.5	13.7	60.0
1 + 3	+	+	−	−		3.2	0.3	3.7	1.0	3.5	6.3
1 + 4	+	+	+	−		12.0	10.0	19.0	16.3	21.3	6.3
1 + 5	+	+	+	+		76.2	78.0	49.8	68.3	49.3	5.7
Multiples[b]						3.6	2.7	11.2	3.9	12.2	21.7
(b) × HfrH											
1 + 2	−	−	−	−	+	4.8	22.0	35.8	34.7	19.5	70.6
1 + 3	−	−	−	+	+	1.6	9.7	7.3	8.3	5.5	12.7
1 + 4	−	−	+	+	+	0.2	2.3	1.7	1.0	0.5	1.1
1 + 5	−	+	+	+	+	0.6	1.7	2.5	3.7	2.0	3.1
1 + 6	+	+	+	+	+	83.8	60.7	22.5	43.7	38.3	0.1
Multiples[b]						9.0	3.6	30.2	8.6	34.2	12.4

[a] The arrangement of genetic markers and crossover regions in the HfrC (a) and HfrH (b) crosses can be depicted as follows (2):



[b] Includes all classes of progeny showing a crossover in region 1 plus any three of the remaining four regions (a); and region 1 plus any three of the remaining five regions, or all six regions (b).

DM1420, and DM1187 is strain JM1 (4, 5, 20). The crosses with HfrC and HfrH were therefore repeated with JM1 as recipient. Analysis of the recombinant progeny obtained did not reveal any major differences between AB1157 and JM1 crosses, except that more (2.5%) of the Thr^+ Leu^+ progeny from the HfrC × JM1 cross were Ara^-.

Since recombinant progeny were not purified to single colonies before being tested for unselected donor markers, differences in the frequencies of multiple-exchange events could have arisen if a significant proportion of the progeny were of mixed genotype, particularly if the frequency of these mixed colonies varied from one cross to another. Whenever possible, the Lac and Ara phenotypes were scored by using indicator medium (MacConkey) in addition to defined minimal medium. In this way, certain mixed clones could be detected, but their frequency was insignificant in the crosses with HfrC (data not shown). With HfrH, mixed clones were more frequent (2 to 3% of the total progeny), but these did not appear to vary in number according to the recipient used, nor among any particular subclass of progeny in any one cross. Although the genotype of a small number of the recombinant progeny may have been incorrectly assigned, this alone would not have been enough to account for the large differences in the frequencies of multiple-exchange events observed in the various crosses.

Since DM1187 and GC3217 are mutator strains (20), many of the progeny recovered from the crosses could have been revertants rather than recombinants. This would be consistent

TABLE 4. *Adjusted frequency of multiple crossovers*

Recipient strain	Total progeny suitable for analysis[a]		% showing multiple crossovers[b]	
	HfrC cross	HfrH cross	HfrC cross	HfrH cross
AB1157	551 (91.8)	468 (93.6)	4.0	9.6
DM49	363 (90.7)	205 (68.3)	3.0	5.4
GC3217	480 (80.0)	341 (56.8)	14.0	53.1
DM1180	354 (88.5)	171 (57.0)	4.2	15.2
DM1420	331 (82.8)	300 (75.0)	14.8	45.7
DM1187	236 (33.7)	117 (16.7)	64.4	73.5

[a] This includes only those progeny with a crossover in region 4 or 5 of the HfrC cross or in region 4, 5, or 6 of the HfrH cross, and therefore excludes the first two progeny classes displayed in Table 3. Figures in parentheses are percentage of total progeny analyzed (Table 2).

[b] As defined in footnote *b* of Table 3.

with the fact that more than half of the progeny obtained with DM1187 had not inherited any of the unselected donor alleles tested (Table 3). However, reversion controls revealed that no more than 0.1% of the Gal^+ progeny obtained in the HfrH cross were due to selection of spontaneous revertants. The *thr-1* allele reverted more readily. Nevertheless, spontaneous revertants accounted for less than 1% of the progeny selected in the crosses with HfrC, except when the recipient was DM1187, in which case they formed 2 to 3% of the total progeny. This level of spontaneous reversion was insufficient to account for the drastic reduction in linkage observed with DM1187, though reversion induced as a result of the fact that mating itself may have promoted SOS functions, including mutagenesis, cannot be ruled out. However, mating-induced reversion would have had to be very efficient to account for a large proportion of the total progeny selected, since, even in the least efficient cross (HfrC × DM1187), these were obtained at a frequency of approximately 1.2% per Hfr donor cell in the mating mixture.

Analysis of the progeny obtained with strain DM49 provided evidence that a *lexA* (*spr*$^+$) mutation reduces the frequency of multiple exchanges in the merozygotes. This effect of a *lexA* mutation was even more apparent with strain DM1180, in which the effect of *tif-1* on the genotype of the recombinant progeny was largely suppressed. In crosses with HfrH, the linkage of unselected donor markers was also reduced. A similar effect was reported by Lloyd and Low (13) for crosses involving HfrH and recipient strains having reduced *recA* gene function.

DISCUSSION

This paper describes some consequences of constitutive synthesis of high levels of the *recA* gene product, protein X (9), for recombination in *tif* and *spr* merozygotes. By using multiply marked recipient strains and analyzing the genotype of recombinants recovered from crosses with Hfr donors, elevated protein X synthesis was shown to be associated with an increase in the frequency of genetic exchanges between Hfr and F^- DNA in the merozygotes. Recombination proficiency, however, increased only slightly. By contrast, the inability to induce protein X synthesis associated with a mutation in *lexA* (9, 11) was found to slightly reduce recombination proficiency. In addition, it reduced the frequency of progeny showing multiple genetic exchanges, although in the cross with HfrC the effect was quite small. What is more significant is that the *lexA* mutation appeared to suppress the effect of *tif-1* on the recombinant genotype.

These results suggest that the frequency of crossover between Hfr and F^- DNA is strongly dependent on the level of the *recA* gene product in merozygotes. This conclusion has also been reached from studies with mutations in *recA* that confer a leaky Rec^- phenotype (13). However, the *spr-51* mutation alone suffices to promote constitutive synthesis of protein X (9; P. Emmerson, K. Powell, S. West, and K. Botcherby, *In* I. Molineux and M. Kohiyama, ed., *DNA Synthesis, Present and Future*, in press). Yet the genotype of the recombinant progeny was much more dramatically altered when the recipient strain (DM1187) also carried *tif-1*. Castellazzi et al. (5) proposed that to reach its active configuration the *recA* gene product must interact with an effector molecule normally present in a low amount within the cell. Furthermore, they proposed that the *tif-1* mutation increases the affinity of the *recA* protein for this effector. The results obtained with the *tif-1 spr* strain (DM1187) may therefore reflect not only an increase in the level of the *recA* gene product, but also the fact that more of it was in its active configuration. This interpretation would agree with the observation by Mount (20) that spontaneous expression in *spr* strains of those SOS functions requiring *recA* gene function is dependent on *tif-1*.

Although increasing *recA* gene function would seem to promote crossover events, the relatively high yields of recombinants obtained in crosses with *lexA* (*spr*$^+$) recipients suggests that integration of Hfr DNA into the recipient chromosome can proceed fairly efficiently without induction of the *recA* gene product. However, the possibility that a limited residual capacity for activating *recA* remained in the *lexA* strains cannot be ruled out. What is fairly clear from the results described here is that the *recA* activity which promoted increased crossing over is

the same as that necessary for inducing SOS functions (28). If crossing over were mediated by an activity of the *recA* protein which is permanently expressed, as proposed by Castellazzi et al. (5), its frequency in *spr* mutants would have been independent of *tif-1*.

A striking feature of the crosses with DM1187 was the extent to which Hfr markers failed to appear among the progeny selected. In crosses with wild-type recipients, a donor marker proximal to the selected marker is normally inherited with high efficiency, even when they are not very closely linked. The increased frequency of crossover events evident in strain DM1187 would certainly have the effect of reducing linkage, but unless the efficiency of integrating donor DNA fragments was also reduced, more of the progeny should have inherited at least one of the unselected donor alleles tested. The slightly reduced recombination proficiency of this strain, particularly in relation to those carrying either *tif-1* or *spr-51* alone, suggests that integration may indeed have been less efficient. This could have been due to the reduction in the interval between crossover events, which would have the effect of reducing the Hfr DNA to short fragments. Reducing the size of donor DNA fragments in merozygotes may well reduce the efficiency of integration (19). An alternative explanation for the genotype of the recombinants obtained with DM1187 is that recombination proceeds via a recombination pathway (6) that is not operative in wild-type strains. For instance, there is some evidence that the RecF pathway (6) is dependent on both $recA^+$ and $lexA^+$ (R. G. Lloyd and S. Johnson, unpublished data; A. J. Clark, personal communication), and therefore may be an inducible function expressed constitutively in SOS-induced strains. Recombinant progeny generated via the RecF pathway may very well differ from those produced by the RecBC pathway operative in wild-type strains (3, 16).

ACKNOWLEDGMENTS

I am grateful to P. T. Emmerson and D. W. Mount for supplying some of the *E. coli* strains used, and to Sandra Johnson for her expert technical assistance.

This work was supported by a grant from the Science Research Council.

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