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Characterization of the *Pseudomonas aeruginosa recA* Gene: the Les⁻ Phenotype

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The Les⁻ phenotype (lysogeny establishment deficient) is a pleiotropic effect of the *lesB908* mutation of *Pseudomonas aeruginosa* PAO. *lesB908*-containing strains are also (i) deficient in general recombination, (ii) sensitive to UV irradiation, and (iii) deficient in UV-stimulated induction of prophages. The *P. aeruginosa recA*-containing plasmid pKML3001 complemented each of these pleiotropic characteristics of the *lesB908* mutation, supporting the hypothesis that *lesB908* is an allele of the *P. aeruginosa recA* gene. The phenotypic effects of the *lesB908* mutation may be best explained by the hypothesis that the *lesB908* gene product is altered in such a way that it has lost synaptase activity but possesses intrinsic protease activity in the absence of DNA damage. The Les⁻ phenotype is a result of the rapid destruction of newly synthesized phage repressor, resulting in lytic growth of the infecting virus. This hypothesis is consistent with the observations that increasing the number of copies of the phage repressor gene by increasing the multiplicity of infection (i.e., average number of phage genomes per cell) or by introducing the cloned phage repressor gene into a *lesB908* mutant will also suppress the Les⁻ phenotype in a phage-specific fashion.

The *recA* gene of *Pseudomonas aeruginosa* PAO has been cloned and partially characterized (12, 13). The protein product of this gene mediates generalized homologous recombination in *P. aeruginosa*. The cloned gene restores recombination proficiency and UV resistance to *recA102* mutants of *P. aeruginosa* (6, 13). The *P. aeruginosa recA* gene product is required for UV induction of *P. aeruginosa* prophages F116L and D3. Lysogens of *recA102* mutants do not induce prophage upon exposure to UV irradiation. This property is also restored by the cloned wild-type gene (13).

P. aeruginosa is host to a diverse population of temperate bacteriophages (11). Holloway (9) and Miller and Ku (17) isolated several *P. aeruginosa* mutants that were deficient in lysogeny establishment (Les⁻). A subset of the Les⁻ mutations, exemplified by *lesB908*, has a pleiotropic phenotype. They are recombinationally deficient, more sensitive to UV and X-irradiation, and impaired in host-cell reactivation of UV-damaged bacteriophages (17). These characteristics of the *lesB908* mutation suggest that it may be an altered form of the *P. aeruginosa recA* gene.

The establishment of lysogeny by phage lambda of *Escherichia coli* is a complex process involving both phage- and host-encoded functions (8). The cI repressor protein is essential for both the establishment and the maintenance of lysogeny. The RecA protein of *E. coli* influences the lysogenic response by modulating the effective concentration of cI repressor. The RecA protein can be activated by DNA-damaging agents to mediate cleavage of the phage repressor by either acting as a specific protease (23) or stimulating the autocatalytic activity of the cI protein (15). As the actual mechanism of inactivation is unclear, we will here use the term RecA protease, merely for convenience. Alterations in the activity of RecA protein through mutation can influence both the establishment and the maintenance of lysogeny in *E. coli*. When a temperature-activatable form of the RecA protein is overproduced in a *recA441* (*tif-1*) *lexA*(Def) mutant, the frequency of the establishment of lysogeny is reduced (18). Lysogens of *recA*(Def) mutants do not release phage either spontaneously or after UV irradiation (3).

In *P. aeruginosa* the *lesB908* mutation alters both the establishment and the maintenance of lysogeny. Even though the establishment of lysogeny is greatly reduced in Les⁻ mutants, spontaneous induction of prophage occurs from the rare Les⁻ lysogen at detectable, although reduced, levels (9, 17), and exposure of lysogens to DNA-damaging agents such as UV irradiation does not increase the level of phage produced (29).

The *P. aeruginosa* temperate bacteriophage D3 has been shown to encode a repressor (*c1*) of lytic functions that is the direct functional analog of the lambda cI repressor (16). The Les⁻ phenotype can be suppressed by infecting the cell with phage F116L or D3 at a high multiplicity of infection (MOI; 9, 17), suggesting that a phage-encoded gene product required for the establishment of lysogeny (perhaps the *c1* repressor in the case of D3) is not produced, or is produced inefficiently, in *lesB908*-containing cells.

To investigate the hypothesis that *lesB908* is an allele of the *recA* gene, a recombinant plasmid carrying the *P. aeruginosa recA* gene (13) was mobilized into *P. aeruginosa* RM8, a strain containing *lesB908* (17). The resulting clones were examined for complementation of the various aspects of the pleiotropic mutant phenotype. In addition, a clone of the D3 *c1* repressor (16) was introduced into RM8 to determine if overproduction of this repressor would suppress the Les⁻ phenotype.

MATERIALS AND METHODS

Bacteria, plasmids, and bacteriophages. The bacterial strains used are listed in Table 1. Bacteriophages D3 and F116L are temperate phages which respond to the Les⁻ phenotype (17). Plasmids pKML3001 and pKML6 have been characterized previously. pKML3001 consists of a 2.3-kilobase-pair fragment of the *P. aeruginosa* PAO chromosome

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TABLE 1. *P. aeruginosa* strains

Strain	Plasmid (prophage)	Relevant genotype[a]	Source or reference
PAO303		*argB21*	17
JC9005	FP2	*pur-600*	17
RM8		*lesB908 argB21*	17
RM265		*recA102 leu-10*	13
RM2137	pKML6	*lesB908 argB21*	RM8
RM2183	pKML3001	*argB21*	PAO303
RM4114	pKML3001	*lesB908 argB21*	RM8
RM5003	(D3)	*argB21*	PAO303
RM5004	(D3)	*lesB908 argB21*	RM8
RM5005	pKML3001 (D3)	*lesB908 argB21*	RM4114

[a] Genotype symbols and abbreviations are as specified by Bachmann (1) except for *les*, which stands for lysogeny establishment deficiency (17).

which contains only the *P. aeruginosa recA* gene cloned into pCP13 (13). pKML6 expresses the *P. aeruginosa* phage D3 *cI* repressor gene from a 9-kilobase-pair fragment of phage DNA cloned into pME292 (16). FP2 is an IncP8 fertility factor which codes for mercury resistance and mobilizes the *P. aeruginosa* chromosome clockwise from 0 min on the genetic map (10).

Efficiency of lysogenization testing. (i) Qualitative tests. These tests were carried out as described by Miller and Ku (17). A small sample of phage lysate (approximately 10^9 PFU/ml) was streaked on an L-agar plate and allowed to dry. Cells to be tested were grown in L broth (17) and streaked perpendicular to the phage. Plates were incubated at 37°C for 16 h. The Les⁻ phenotype is defined as lack of growth at the phage-bacterium streak interface due to the failure to establish lysogeny (17).

(ii) Quantitative tests. Cells to be tested were grown in L broth at 37°C to approximately 20 Klett units, measured at 660 nm. The cells were harvested by centrifugation and suspended in an equal volume of TMN buffer (2). The cells were mixed with phage at several different MOIs and incubated at 37°C for 10 min to allow phage absorption. The infected cells were harvested by centrifugation in an Eppendorf Microfuge and suspended in an equal volume of TMN buffer. The cells were diluted in TMN buffer and plated on L agar to determine survival. The titer of the phage stock was determined with *P. aeruginosa* PAO303 as an indicator strain to estimate each MOI as accurately as possible. Colonies appearing after overnight incubation at 37°C were patched onto fresh L agar and, after a 4-h period of growth at 37°C, replica plated onto L agar seeded with approximately 10^7 CFU of *P. aeruginosa* PAO303 in 2 ml of lambda top agar (13). Lysogenized clones were identified by the spontaneous release of phage, which caused a clearing in the phage-sensitive PAO303 lawn.

Other methods. Triparental matings, transformations, conjugations, determination of UV sensitivity, preparation of phage stocks, and UV induction of prophages were done as described previously (2, 12, 13, 16).

RESULTS

Introduction of pKML3001 and pKML6 into RM8. Plasmid pKML3001, which contains the *P. aeruginosa* PAO *recA* gene on a 2.3-kilobase-pair chromosomal fragment, was introduced into RM8 with a triparental mating technique (13). Transconjugants containing pKML3001 were selected on L agar containing tetracycline (250 μg/ml). The presence of the *recA* gene was confirmed by demonstrating the ability of the tetracycline-resistant clones to grow on medium containing 0.01% (vol/vol) methyl methanesulfonate (13). Clones containing pKML3001 were examined to determine the ability of the cloned genes to complement the pleiotropic manifestations of the *lesB908* mutation.

Plasmid pKML6 (16) carries the phage D3 *cI* repressor gene. pKML6 was introduced into RM8 by transformation with selection for carbenicillin resistance as described previously (16). These clones were also examined for various phenotypic features associated with the *lesB908* mutation.

UV resistance. pKML3001 restores resistance to UV irradiation to strains of *P. aeruginosa* containing the *recA102* mutation (13). The ability of plasmids pKML3001 and pKML6 to restore resistance to UV irradiation to *P. aeruginosa* RM8 was tested. The presence of the cloned *recA* analog restored resistance to levels exceeding those exhibited by its isogenic Les⁺ (RecA⁺) parent, PAO303 (Fig. 1). The introduction of pKML3001 into PAO303 did not affect the frequency of killing of this strain by UV irradiation. As expected, the presence of plasmid pKML6 did not confer a UV-resistant phenotype to cells containing it (data not shown).

Recombinational proficiency. The ability of *lesB908* mutants containing pKML3001 or pKML6 to carry out homologous recombination was quantified after they were mated with the FP2 donor strain JC9005 (Table 2). The presence of the cloned *recA* gene restored recombinational proficiency to pKML3001 transformants of RM8. The presence of plasmid pKML6 did not restore the ability of *P. aeruginosa* RM8 to support homologous recombination.

Prophage induction. The levels of spontaneous and UV-stimulated induction to lytic growth of D3 prophage from lysogens of RM8 were determined (Table 3). Like other Les⁻ mutants (29), lysogens of RM8 were found to release phage spontaneously at reduced, but significant, levels. UV

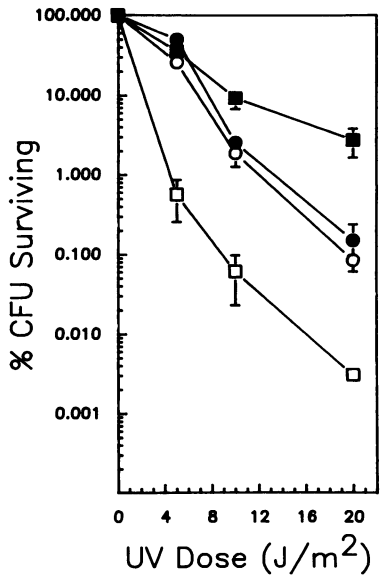

FIG. 1. Sensitivity to UV irradiation of *lesB908* mutants containing pKML3001. Cells were grown to a density of approximately 10^8 CFU/ml in Luria broth, pelleted, suspended in saline, and exposed to various fluences of UV irradiation. After appropriate dilution in saline, the irradiated cells were plated in duplicate on L agar and incubated at 37°C overnight in the dark. Mean values ± standard error are plotted. Symbols: ○, PAO303 (Rec⁺); ●, RM2183 (Rec⁺, pKML3001); □, RM8 (*lesB908*); ■, RM4114 (*lesB908*, pKML3001).

TABLE 2. Recombinational proficiency of *lesB908* mutants containing pKML3001 and pKML6[a]

Strain	Relevant characteristics	Expt no.[b]	Recombinational proficiency (*argB*$^+$ recombinants/10^6 donors)
PAO303	Rec$^+$	1	5.80
		2	8.75
RM8	*lesB908*	1	<0.02
		2	<0.02
RM4114	*lesB908*, pKML3001[c]	1	1.50
		2	2.30
RM2138	*lesB908*, pKML6[d]	1	<0.02
		2	<0.02

[a] JC9005 was the donor strain. Cells were mixed in a donor-to-recipient ratio of 1:1.2. Matings were performed for 2 h at 37°C without shaking.
[b] Two independent experiments are reported. Platings were done in duplicate.
[c] pKML3001 contains the *P. aeruginosa recA* analog.
[d] pKML6 contains the D3 *c1* gene.

irradiation did not increase the amount of phage produced. The presence of plasmid pKML3001 increased the amount of spontaneous release of phage D3 from lysogens to the level found after UV induction of wild-type *P. aeruginosa* cells. However, a significant increase in the amount of phage released from RM8 cells containing pKML3001 after UV irradiation was not observed.

Efficiency of lysogenization. Qualitative lysogeny establishment tests performed on *P. aeruginosa* RM8 with and without pKML3001 revealed an apparent suppression of the Les$^-$ phenotype by plasmid pKML3001 (Fig. 2). Therefore, the frequency of the establishment of lysogeny in RM8 with and without pKML3001 after infection by phage F116L was quantitated (Fig. 3). The presence of the cloned *recA* gene restored the ability of RM8 to be lysogenized at lower MOIs. The frequency of lysogenization was intermediate between those of Les$^-$ and wild-type strains at equivalent MOIs.

To determine if a plasmid containing the D3 *c1* gene would suppress the Les$^-$ phenotype by increasing the gene dosage of the phage repressor, pKML6 was introduced into RM8. The presence of pKML6 conferred a Les$^+$ phenotype to RM8 (Fig. 4). This suppression of the Les$^-$ phenotype was specific for cells infected by phage D3. The frequency of lysogenization by F116L was not affected by the presence of pKML6 in RM8.

DISCUSSION

The *P. aeruginosa recA*-containing plasmid pKML3001 complemented the various pleiotropic characteristics of the *lesB908* mutation, supporting the hypothesis that *lesB908* is an allele of the *P. aeruginosa recA* gene. Henceforth, we will refer to this allele as *recA908*.

Several alleles of the *E. coli recA* gene result in a phenotype similar to that of the *recA908* allele of *P. aeruginosa*. Tessman et al. (26–28) have genetically characterized both recombination-deficient, protease-constitutive (Rec$^-$ Prt^c) and recombination-proficient, protease-constitutive (Rec$^+$ Prt^c) alleles. Of the *recA* mutations of *E. coli* whose protein products have been biochemically characterized, the *recA142* mutation most closely resembles *P. aeruginosa recA908* in phenotype. The RecA142 protein is Rec$^-$ but retains a low level of protease activity (Prt$^\pm$), allowing low levels of spontaneous and UV-induced phage release from lambda lysogens (5, 22). In vitro RecA142 protein exhibits a reduced affinity of single-stranded DNA, which is the primary cause of the Rec$^-$ phenotype exhibited by *recA142* mutants of *E. coli* (4, 14). In addition, single-stranded DNA is presumed to be the signal required for activation of RecA protein to the proteolytic state (22) and is a coeffector for the protease activity in vitro (24).

The phenotypic effects of the *recA908* mutation can be explained by hypothesizing that the *recA908* gene product is altered in such a way that it has lost synaptase activity (Rec$^-$) but possesses intrinsic protease activity in the absence of DNA damage (Prt^c). The synaptase-deficient (Rec$^-$) phenotype of *P. aeruginosa recA908* mutants suggests that the protein produced from this allele may also have reduced affinity for single-stranded DNA (4). Merodiploids containing pKML3001 exhibit effects which may be interpreted as due to cooperative interaction between wild-type and mutant proteins, similar to the cooperative interactions exhibited by the *E. coli* RecA protein in many of its biochemical activities (14).

UV resistance was complemented to greater than wild-type levels in RM8 containing pKML3001 (Fig. 1). Importantly, the presence of the cloned *recA* gene did not result in enhanced UV resistance in the wild-type (PAO303) *P. aeruginosa* genetic background. If the expression of *recA* is inducible and autoregulated in *P. aeruginosa* in a manner analogous to that of the *E. coli recA* gene, the introduction of a regulated wild-type allele on plasmid pKML3001 into a protease-constitutive *recA908* background would be expected to lead to overexpression of the wild-type protein. It is clear that in *E. coli* the RecA protein affects the level of

TABLE 3. Induction of D3 prophage[a]

Strain	Relevant characteristics	Expt no.[b]	Phage titer (PFU/CFU)		Induction ratio
			Spontaneous	Induced	
RM5004	*lesB908*	1	1.0×10^{-4}	1.5×10^{-4}	1.5
		2	2.5×10^{-4}	2.5×10^{-4}	1.0
RM5005	*lesB908*, pKML3001[c]	1	2.30	3.30	1.4
		2	2.90	2.90	1.0
RM5003	Rec$^+$	1	0.07	1.43	20.4
		2	0.29	11.43	39.4

[a] Lysogens were grown to 20 Klett units (measured at 660 nm; approximately 7×10^7 CFU/ml) and induced by exposure to 10 J of UV light per m^2 followed by incubation for 2 h at 37°C in the dark.
[b] Two independent experiments are reported. All platings were done in duplicate.
[c] pKML3001 contains the *P. aeruginosa recA* analog.

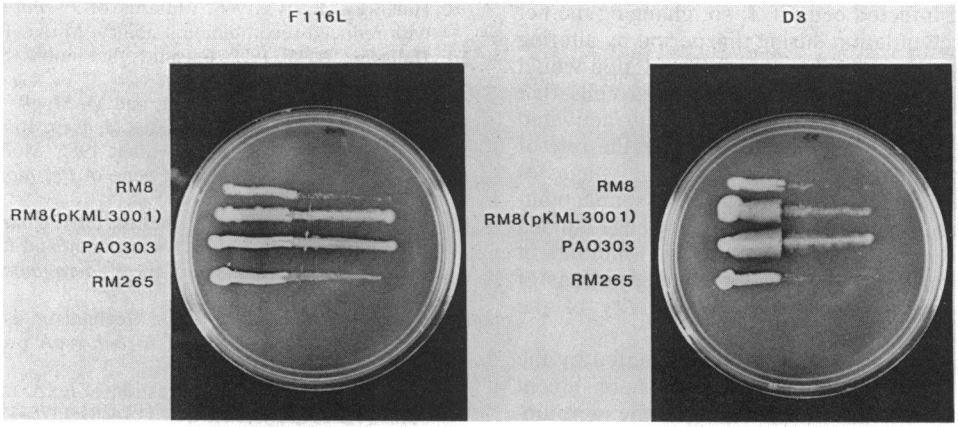

FIG. 2. Qualitative lysogeny establishment test. Strains were cross-streaked against phage F116L or D3 as described in the text.

cell survival after UV irradiation both by regulating gene expression of the SOS network (including the *recA* gene itself) and by acting in some uncharacterized capacity in several DNA repair systems (20, 30). A good correlation may be made between cell survival after UV irradiation and the amount of RecA protein in the cell (21). Tessman and Peterson (26) found that Rec⁻ Prt^c alleles of the *E. coli recA* gene were UV sensitive, whereas Rec⁺ Prt^c alleles showed enhanced resistance to UV irradiation compared with their wild-type parental strains.

The identification of an SOS box consensus sequence in the 5′ leader sequence of the *P. aeruginosa recA* gene (25) suggests that regulation of gene expression in response to stress may in fact occur. We have determined that expression of the *P. aeruginosa recA* gene is inducible in both *P. aeruginosa* and *E. coli* genetic backgrounds (T. A. Kokjohn and R. V. Miller, manuscript in preparation).

It is clear that in both *E. coli* and *P. aeruginosa* DNA damage-activated *recA* gene products interact with phage repressors and promote their destruction (13, 16, 23). This interaction must also be ongoing in the absence of inducing treatments, since the spontaneous release of phage also requires a functional *recA* gene product (19). *P. aeruginosa recA908* mutants supported spontaneous induction of prophage D3 at reduced, but significant, levels (Table 3). Possibly this is due to the reduced affinity of the RecA908 protein for single-stranded DNA. The level of spontaneous prophage induction in RM8 containing pKML3001 was significantly increased to levels equal to the fully UV-induced state of the wild-type isogenic parental strain. The increase in spontaneous levels of phage release in wild-type–*recA908* merodiploids appears to be a consequence of the specific characteristics of the RecA908 protein. Significantly, the introduction of pKML3001 into lysogens of *recA102*-containing strains causes an increase in spontaneous levels of release to only wild-type levels (13). UV irradiation of such wild-type–*recA102* merodiploids causes an additional increase in the level of phage production (13).

Whereas the maintenance of lysogeny depends on maintaining an effective concentration of repressor in the lysogen, it appears that the establishment of lysogeny is a function of the rate of repressor accumulation and, hence, of the elapsed time before an effective repressor concentration

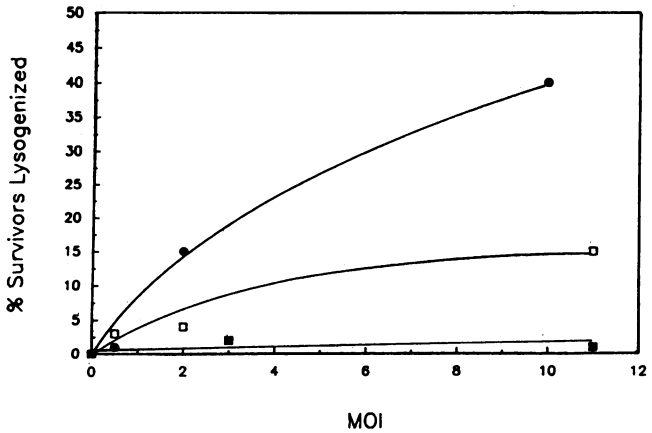

FIG. 3. Efficiency of lysogenization of *lesB908* mutants containing pKML3001. Strains were infected with temperate phage F116L at various MOIs, and the percentage of total input cells lysogenized was measured as described in the text. A representative experiment of two repetitions is shown. The data obtained from both sets of experiments were qualitatively the same. Symbols: ●, PAO303 (Les⁺); ■, RM8 (*lesB908*); □, RM4114 (*lesB908*, pKML3001).

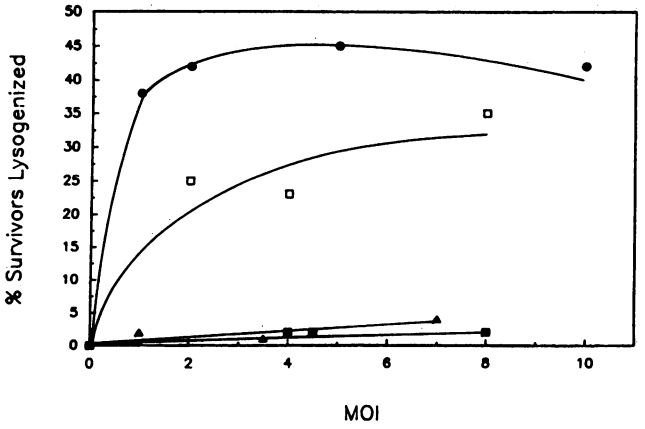

FIG. 4. Efficiency of lysogenization of *lesB908* mutants containing pKML6. Strains were infected with either temperate phage D3 or F116L at various MOIs, and the percentage of total input cells lysogenized was measured as described in the text. A representative experiment of two repetitions is shown. The data obtained in both sets of experiments were qualitatively the same. Symbols: ●, PAO303 (Les⁺, phage D3); ■, RM8 (*lesB908*, phage D3); □, RM2137 (*lesB908*, pKML6; phage D3); ▲, RM2137 (*lesB908*, pKML6, phage F116L).

is established in the infected cell (7). If so, changing the net rate of repressor accumulation during this period by altering either the rate of synthesis or the rate of degradation would be expected to alter the proportion of infected cells that become lysogenic. Because of its constitutively activated state, the RecA908 protein may act to increase the rate of destruction of repressor protein during the initial stages of phage infection, thus reducing the rate of repressor accumulation. Similar effects on the lysogenization of phage lambda have been observed in *lexA*(Def) *recA441* (*tif-1*) mutants of *E. coli* (18), which only accumulate low concentrations of repressor due to the increased protease activity of the overproduced RecA441 protein (19).

The Les$^-$ phenotype was partially complemented by the introduction of pKML3001, which allows the establishment of lysogeny by phage F116L at lower MOIs in the merodiploid. The presence of plasmid pKML3001 may allow the complementation of the Les$^-$ phenotype by producing wild-type *recA* gene product (not activated) which competes with the mutant (activated) protein for phage repressor. Repressor molecules interacting with wild-type, nonactivated RecA molecules may be protected from destruction by the RecA908 protein. Hence, the effective rate of accumulation of repressor may be increased, allowing establishment of lysogeny in a statistically larger subpopulation of infected cells. Similarly, Mount (19) found that a *recA*$^+$-*recA441* merodiploid accumulated a level of lambda repressor between the levels obtained in *recA441* mutants and wild-type haploid cells.

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LITERATURE CITED

1. **Bachmann, B. J.** 1983. Linkage map of *Escherichia coli* K-12, edition 7. Microbiol. Rev. **47**:180–230.
2. **Cavenagh, M. M., and R. V. Miller.** 1986. Specialized transduction of *Pseudomonas aeruginosa* PAO by bacteriophage D3. J. Bacteriol. **165**:448–452.
3. **Clark, A. J.** 1973. Recombination deficient mutants of *Escherichia coli* and other bacteria. Annu. Rev. Genet. **7**:67–86.
4. **Cox, M. M., and I. R. Lehman.** 1987. Enzymes of general recombination. Annu. Rev. Biochem. **56**:229–262.
5. **Dutreix, M., A. Bailone, and R. Devoret.** 1985. Efficiency of induction of prophage λ mutants as a function of *recA* alleles. J. Bacteriol. **161**:1080–1085.
6. **Früh, R., J. M. Watson, and D. Haas.** 1983. Construction of recombination-deficient strains of *Pseudomonas aeruginosa*. Mol. Gen. Genet. **191**:334–337.
7. **Gussin, G. N., A. D. Johnson, C. O. Pabo, and R. T. Sauer.** 1983. Repressor and Cro protein: structure, function, and role in lysogenization, p. 93–121. *In* R. W. Hendriz, J. W. Roberts, R. W. Stahl, and R. A. Weisberg (ed.), Lambda II. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.
8. **Herskowitz, I., and D. Hagen.** 1980. The lysis-lysogeny decision of phage λ: explicit programming and responsiveness. Annu. Rev. Genet. **14**:399–445.
9. **Holloway, B. W.** 1966. Mutants of *Pseudomonas aeruginosa* with reduced recombination ability. Mutat. Res. **3**:452–455.
10. **Holloway, B. W.** 1979. Plasmids that mobilize bacterial chromosome. Plasmid **2**:1–9.
11. **Holloway, B. W., J. B. Egan, and M. Monk.** 1960. Lysogeny in *Pseudomonas aeruginosa*. Aust. J. Exp. Biol. **38**:321–330.
12. **Kokjohn, T. A., and R. V. Miller.** 1985. Molecular cloning and characterization of the *recA* gene of *Pseudomonas aeruginosa* PAO. J. Bacteriol. **163**:568–572.
13. **Kokjohn, T. A., and R. V. Miller.** 1987. Characterization of the *Pseudomonas aeruginosa recA* analog and its protein product: *rec-102* is a mutant allele of the *P. aeruginosa* PAO *recA* gene. J. Bacteriol. **169**:1499–1508.
14. **Kowalczykowski, S. C.** 1987. Mechanistic aspects of the DNA strand exchange activity of *E. coli* recA protein. Trends Biochem. Sci. **12**:141–145.
15. **Little, J. W.** 1984. Autodigestion of lexA and phage λ repressors. Proc. Natl. Acad. Sci. USA **81**:1375–1379.
16. **Miller, R. V., and T. A. Kokjohn.** 1987. Cloning and characterization of the *c1* repressor of *Pseudomonas aeruginosa* bacteriophage D3: a functional analog of phage lambda cI protein. J. Bacteriol. **169**:1847–1852.
17. **Miller, R. V., and C.-M. C. Ku.** 1978. Characterization of *Pseudomonas aeruginosa* mutants deficient in the establishment of lysogeny. J. Bacteriol. **134**:875–883.
18. **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.
19. **Mount, D. W.** 1979. Isolation and characterization of mutants of λ*recA* which synthesize a hyperactive *recA* protein. Virology **98**:484–488.
20. **Ossanna, N., K. R. Peterson, and D. W. Mount.** 1986. Genetics of DNA repair in bacteria. Trends Genet. **2**:55–58.
21. **Quillardet, P., P. L. Moreau, H. Ginsburg, D. W. Mount, and R. Devoret.** 1982. Cell survival, UV reactivation and induction of prophage λ in *Escherichia coli* K12 overproducing *recA* protein. Mol. Gen. Genet. **188**:37–43.
22. **Roberts, J. W., and R. Devoret.** 1983. Lysogenic induction, p. 123–144. *In* R. W. Hendrix, J. W. Roberts, R. W. Stahl, and R. A. Weisberg (ed.), Lambda II. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.
23. **Roberts, J. W., and C. W. Roberts.** 1975. Proteolytic cleavage of bacteriophage λ repressor in induction. Proc. Natl. Acad. Sci. USA **72**:147–151.
24. **Roberts, J. W., and C. W. Roberts.** 1981. Two mutations that alter the regulatory activity of *E. coli recA* protein. Nature (London) **290**:422–424.
25. **Sano, Y., and M. Kageyama.** 1987. The sequence and function of the *recA* gene and its protein in *Pseudomonas aeruginosa* PAO. Mol. Gen. Genet. **208**:412–419.
26. **Tessman, E. S., and P. Peterson.** 1985. Plaque color method for rapid isolation of novel *recA* mutants of *Escherichia coli* K-12: new classes of protease-constitutive *recA* mutants. J. Bacteriol. **163**:677–687.
27. **Tessman, E. S., and P. K. Peterson.** 1985. Isolation of protease-proficient, recombinase-deficient *recA* mutants of *Escherichia coli* K-12. J. Bacteriol. **163**:688–695.
28. **Tessman, E. S., I. Tessman, P. K. Peterson, and J. D. Forestal.** 1986. Roles of RecA protease and recombinase activities of *Escherichia coli* in spontaneous and UV-induced mutagenesis and in Weigle repair. J. Bacteriol. **168**:1159–1164.
29. **van de Putte, P., and B. W. Holloway.** 1968. A thermosensitive recombination-deficient mutant of *Pseudomonas aeruginosa*. Mutat. Res. **6**:195–203.
30. **Walker, G. C.** 1984. Mutagenesis and inducible responses to deoxyribonucleic acid damage in *Escherichia coli*. Microbiol. Rev. **48**:60–93.