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# *exrB:* a *malB*-linked gene in *Escherichia coli* B involved in sensitivity to radiation and filament formation

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SUMMARY

PAM 26, a radiation-sensitive mutant of *Escherichia coli* strain B, is described. Its properties are attributable to a mutation in a gene, *exrB*, which is cotransducible with *malB*. It differs from *uvrA* (also *malB*-linked) derivatives of strain B in being sensitive to 1-methyl-3-nitro-1-nitroso-guanidine and $\gamma$-radiation, and in being able to reactivate UV-irradiated phage T3. It differs from *exrA* (also *malB*-linked) derivatives of strain B in forming filaments during the course of normal growth as well as after irradiation. When *exrB* was transduced into a K12 (*lon*$^+$) strain, filaments did not form spontaneously. Three-point transductions established the order of markers as *metA malB exrB*. Based on an analysis of the frequency of wild-type recombinants in a reciprocal transduction between *exrA* and *exrB* strains, it was inferred that they are not isogenic and that the order of markers is *malB exrA exrB*.

## 1. INTRODUCTION

Two genes have been found in *Escherichia coli* B which are linked to *malB* and are involved in resistance to radiation. These are *exrA* (e.g. Bs2) and *uvrA* (e.g. Bs12) (Hill & Simson, 1961; Hill & Feiner, 1964; Donch & Greenberg, 1968; Chung & Greenberg, 1968). *exrA* strains are more sensitive to ultraviolet radiation (UV) and X-rays and to chemicals such as methylmethane sulphonate and 1-methyl-3-nitro-1-nitrosoguanidine (NG) than parental strain B. Most importantly for purposes of this paper, *exrA* suppresses the radiation induced filamentation observed in parental strain B and attributable to *lon* (Donch, Green & Greenberg, 1968). *exrA* strains are HCR$^+$, able to rescue a portion of some UV-irradiated phage. The gene *lex* confers phenotypic properties corresponding to those of *exrA* (Howard-Flanders & Boyce, 1966; Mount, Low & Edmiston, 1972). *uvrA* strains are sensitive to UV but not to X-rays or methylmethane sulphonate and are HCR$^-$. *uvrA* does not suppress UV-induced filamentation in *lon* strains.

Among radiation-sensitive mutants of strain B, isolated following treatment with NG in separate experiments, were two, PAM 26 and PAM 443, which are HCR$^+$ and form filaments not only after UV irradiation but spontaneously. The gene responsible for this phenotype, which we shall call *exrB*, is cotransducible by

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phage P1 with *malB*. A preliminary description of PAM 26 is the subject of this report.

## 2. METHODS

(*a*) *Media.* The minimal medium used for transductions was Davis and Mingioli (1950) (DM) broth to which Bacto agar (Difco) was added at a final concentration of 2 % and glucose or maltose (for Mal$^+$ selection) at 0·5 %. Amino acids were used in concentrations of 50 $\mu$g/ml. Complete (JN) broth consisted of tryptone 5 g, NaCl 5 g, glucose 1 g, and yeast extract (Difco) 5 g/l. of deionized water. Viable counts and UV survival curves were done with this medium without glucose, to which 1·5 % Bacto agar (Difco) was added.

(*b*) *Bacteria.* Bacterial strains used are described in Table 1.

Table 1. *Strains of bacteria used*

| Strain | Markers relevant to radiation sensitivity | Other relevant markers | Source |
|---|---|---|---|
| | | *E. coli* derivatives | |
| B | *lon* | *malB* | Ruth Hill |
| B251 | *lon* | *malB*$^+$ | W. Arber |
| PAM 26 | *exrB lon* | *malB* | This paper |
| PAM 443 | *exrB lon* | *malB* | This paper |
| PAM 444 | *exrB lon* | *malB*$^+$ | By transduction from B251 |
| Bs2 | *exrA lon* | *malB* | Ruth Hill |
| PAM 23 | *exrA lon* | *malB*$^+$ | From Bs2 by transduction from B251 |
| Bs12 | *uvrA lon* | *malB* | Ruth Hill |
| | | *E. coli* K12 derivatives | |
| AB1157 | Wild-type | *argD* | E. Adelberg |
| PAM 5764 | Wild-type | *metA* | AB1157 from AB1191 by transduction |
| AB1191 | Wild-type | *metA* | E. Adelberg |
| PAM 5717 | *exrA* | *metA*$^+$ | PAM 5764 by transduction from Bs2 |
| PAM 5725 | *exrB* | *metA*$^+$ | PAM 5764 by transduction from PAM 444 |

Abbreviations are as recommended by Demerec, Adelberg, Clark & Hartman (1966).

(*c*) *Phage.* P1b*vir* has been described earlier (Donch & Greenberg, 1968). It grows on both strain B and its derivatives and K12, and transduces almost equally well between these strains in either direction. It will be referred to as P1. Transducing phage were carried through three successive passages in the donor strain prior to being used for transduction. Phage T3, used for HCR studies, was grown in strain B.

(*d*) *Transduction.* Transductions were performed essentially as described by Lennox (1955) and by Donch & Greenberg (1968). PAM 26 grows very slowly and does not attain a high titre. Therefore, overnight cultures were centrifuged for 10 min at 5000 rev/min, the supernatant decanted, fresh broth added to original

volume, and incubated for 2 h. At this point P1 was added at a multiplicity of 0·1–1·0. The preparation was shaken gently for 40 min and then centrifuged. The supernatant was decanted and the pellet resuspended in 1/4 the original volume; 0·1 ml of this or an appropriate dilution of it in water were plated on selective medium and incubated at 37 °C for 2 days. Preparations handled identically but with no P1 added were used to determine frequency of revertants. For the two loci transduced, *malB*$^+$ and *metA*$^+$, the reversion frequency was nil.

Transductants were purified by streaking on selecting medium, single clones were isolated, generally grown in JN broth, and tested for unselected markers of the donor. *mal*$^+$ transductants of PAM 26 were sometimes unstable, so these were grown in liquid selecting medium and reisolated on selecting medium.

The selecting medium for transduction to prototrophy was DM. Mal$^+$ selection was done on DM with maltose (0·3 %) as carbon source. Auxotrophic markers were tested by spotting overnight cultures with a capillary tube on DM medium deficient in the nutritional requirement. Mal as an unselected marker was tested by spotting cultures onto Brom Thymol Blue (BTB) Difco broth solidified with 2 % Bacto (Difco) agar of JN agar containing $2{\cdot}0 \times 10^{-4}$ % triphenyl tetrazolium chloride.

(*e*) *Ultraviolet sensitivity*. Methods for testing ultraviolet sensitivity have been described in detail (Greenberg, 1964). The terms 'resistance' or 'sensitivity' are used relative to the resistance or sensitivity of the recipient. The rapid spot test was used for examining large numbers of transductants, but definitive survival curves were made on a sufficient number to confirm the interpretation of the spot tests.

(*f*) *Effect of γ-rays*. This was performed at the MRC Radiobiology Unit, Harwell, England, by the method described by Green, Bridges & Riazuddi (1973).

(*g*) *Host cell reactivation*. Phage T3 was diluted to $5 \times 10^7$ pfu/ml in buffered saline and irradiated with UV 924 ergs/mm$^2$; 0·1 ml of the irradiated phage was spread on JN agar and test cultures added as spots. HCR$^+$ cultures were eliminated; HCR$^-$ exhibited only an occasional plaque.

Unless stated otherwise all incubations were done at 37 °C.

## 3. RESULTS

(*a*) *Sensitivity to radiation and NG*. Fig. 1 shows the survival of strains PAM 26, Bs1, Bs12 and Bs2 after exposure to UV. PAM 26 is much more sensitive to UV than its parent strain B, somewhat more sensitive than Bs2 and less sensitive than Bs12.

*uvr* mutations do not confer sensitivity to ionizing radiation (Hill & Simson, 1961) though *exr* does. Fig. 1 also shows that PAM 26 is more sensitive to γ-radiation than parental strain B. *uvrA* does not confer more sensitivity to monofunctional alkylating agents such as 1-methyl-3-nitro-1-nitrosoguanidine (NG) but *exrA* does (Witkin, 1967). Results in Fig. 2 show that PAM 26 and Bs2 were more sensitive to NG than strain B. PAM 26 is also sensitive to methylmethanesulphonate and ethylmethansulphonate. PAM 26 differs from Bs12 (*uvrA*) in

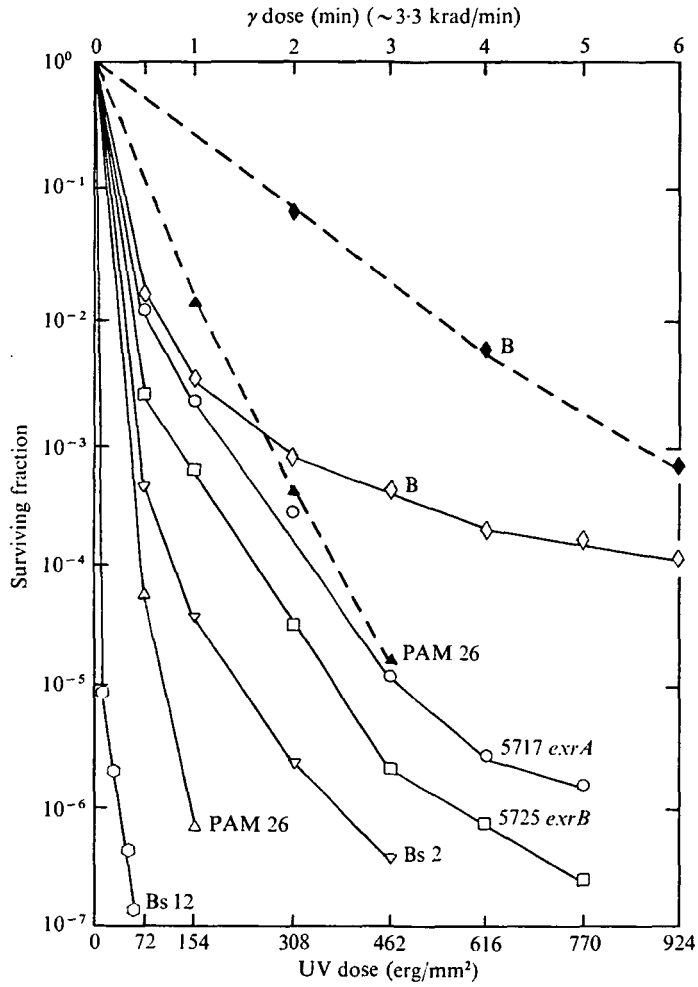


**Fig. 1.** Survival after UV radiation of strains B, Bs2, Bs12, PAM 26, K12 strains PAM 5717 (*exrA*) and PAM 5725 (*exrB*), open symbols, solid lines. Survival after γ-radiation of strains B and PAM 26, closed symbol, dotted lines.

being more sensitive to ionizing radiation and to monofunctional alkylating agent than strain B. In these respects it resembles Bs2 (*exrA*).

(*b*) *Filamentation.* Strain B forms filaments when irradiated but not under normal growth conditions. Bs12 forms some filaments under normal growth conditions but extensive filamentation occurs only following radiation. Bs2 (*exrA*), on the other hand, does not form filaments even when irradiated (Donch *et al.* 1968). PAM 26 forms abundant filaments when grown in either complex or minimal medium. Filaments were observed when PAM 26 was grown at 37 or 42 °C or in the presence of pantoyl lactone, all of which are known to inhibit filamentation in irradiated strain B.

No detailed studies of the kinetics of filament formation have been made with PAM 26. It can be stated only that about half the population of log phase or

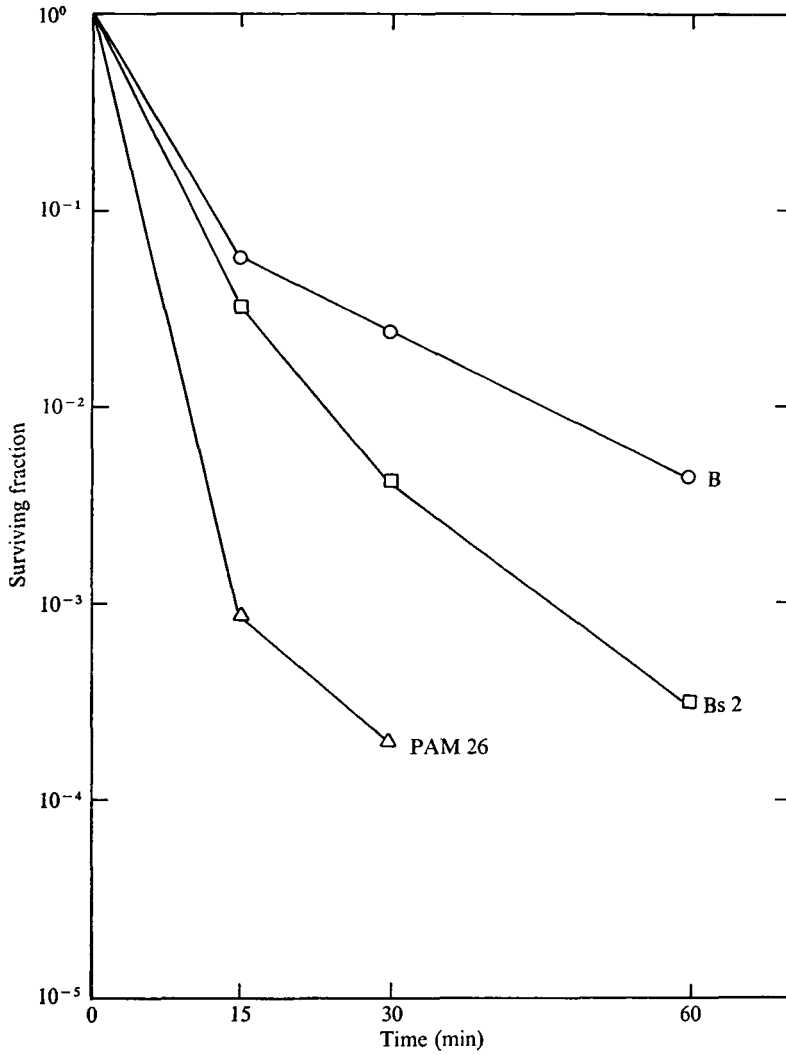


Fig. 2. Survival after treatment with 1-methyl-3-nitro-1-nitrosoguanidine 10 $\mu$g/ml in complete (JN) broth of strains B, Bs2, and PAM 26.

mature cultures of PAM 26 are filaments, as is that of colonies on agar at all stages of growth. To examine the kinetics of filamentation it would be necessary to isolate 'normal' cells and follow their history. Without this sort of experimental evidence it can be proposed only that all cells in a culture form filaments, the 'normal' cells arising from the filaments by some process of budding. Such a process would maintain the high proportion of filaments, or cells becoming filaments, observed at all stages of growth. It would also account for the observed slow rate of growth of cultures as measured in colony-forming units, as well as the low colony-forming ability of mature cultures despite high turbidity.

(c) *Host-cell reactivation*. When phage T3 were irradiated with UV and plated in three hosts, B, Bs12 and PAM 26, more phage survive when plated on PAM 26

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(and B) than when plated on Bs12. PAM 26 therefore reactivates some irradiated T3 and is HCR+.

(*d*) *Linkage to* malB. *uvrA* and *exrA* have been shown to be linked to *malB* (Greenberg, 1964, 1967; Donch & Greenberg, 1968). P1 was used to transduce *malB*+ from B251 (*malB*+ *exrB*+) to PAM 26 (*malB exrB*) with results shown in Table 2. Ninety-four per cent of the *malB*+ transductants of PAM 26 were at least as resistant to UV as the donor and did not filament spontaneously. The UV survival curves of five of these, randomly chosen, were indistinguishable from that of strain B251. Five (0·5 %) of the transductants were even more resistant to UV than strain B. The UV survival curves of these were indistinguishable from those of strain B/r. These probably arise from among mutants of PAM 26 in which its *lon* function was suppressed. This observation complicates interpretation of the behaviour of PAM 26 and will be discussed more fully in a later publication. For the present it does not detract from the point that *exrB*+ is cotransducible with *malB* at a frequency similar to that reported for *exrA* (Chung & Greenberg, 1968, referred to in that paper as *uvr2*).

A donor for the reciprocal transduction, PAM 26 *malB*+ *exrB* × B *malB exrB*+, was obtained by selecting one transductant from the previous experiment which was *malB*+ *exrB*. Forty one per cent of the *malB*+ transductants of strain B were as sensitive to ultraviolet radiation as PAM 26 (Table 2). All the sensitive transductants filamented spontaneously. It is clear that the frequencies of transduction of *exrB* and *exrB*+, assuming for the present that we are observing the transfer of one cistron, are not the same. It is possible that *exrB* strains survive less well than *exrB*+ strains or that the piece of host DNA picked up by the transducing P1 particle varies with the state of the *exrB* cistron. Whatever the explanation for lack or reciprocity, it is clear that *exrB*, like *uvrA* and *exrA*, is closely linked to *malB* and that the tendency to form filaments spontaneously during growth accompanies *exrB*.

Table 2. *Frequency of donor phenotype* (*UVr or UVs*) *transduced with* malB+ *by P1*

| Donor | Recipient | Mal+ transductants | Donor phenotype (UV) (%) |
|---|---|---|---|
| B251 | PAM 26 (*exrB*) | 1194 | 94 |
| PAM 444 (*exrB*) | B | 300 | 41 |

(*e*) *Three-factor transductional crosses. uvrA* is known to be cotransducible with *metA* (Howard-Flanders, Boyce & Theriot, 1966; Schwartz, 1966) as is *exrA* (Donch & Greenberg, 1968). The order of markers proposed is *metA malB exrA uvrA* (Chung & Greenberg, 1968). Three factor crosses were done using P1 grown on PAM 26 (*metA*+, *malB*, *exrB*) to transduce *metA*+ to the K12 derivative, PAM 5764 (*metA*, *malB*+, *exrB*+). The transductants were examined for the unselected donor markers *malB* and UV sensitivity (*exrB*). The results are shown in Table 3. Seven per cent of the *metA*+ transductants inherited *malB* from the donor, but only 2 % inherited *exrB*. All those which were *exrB* were also *malB*.

Table 3. *Frequency of PAM 26 markers* (malB, exrB) *transduced with* metA$^+$ *to PAM 5764* (metA, malB$^+$, exrB$^+$)

| | Frequency donor markers (%) | |
|---|---|---|
| *met*$^+$ transductants | *malB exrB*$^+$ | *malB exrB* |
| 522 | 7 | 2 |

Table 4. *Frequency* (%) *of donor markers,* exrA *and* exrB, *in transductions between PAM 26 and Bs2* (*100* mal$^+$ *transductants selected in each experiment*)

| | | | Transductants | | |
|---|---|---|---|---|---|
| | | | | UV-sensitive | |
| Expt. | Donor | Recipient | UV-resistant | Filamenting | Non-filamenting |
| I | PAM 444 *exrB* | Bs2 | 1 | 2 | 97 |
| II(*a*) | PAM 23 *exrA* | PAM 26 | 38 | 42 | 20 |
| II(*b*) | PAM 23 *exrA* | PAM 26 | 35 | 30 | 35 |

The order of markers is, therefore, *metA*, *malB*, *exrB*, and places *exrB* on the same side of *malB* as *exrA* and *uvrA* (Taylor, 1970).

The survival curves following UV irradiation of the *exrB* derivative of K12, PAM 5725, and an *exrA* derivative, PAM 5717, made in a similar way are shown in Fig. 1. The *exrB* derivative appears to be slightly more sensitive than the *exrA* strain, but this difference is of doubtful significance. It seems safe to say that, were *exrB* isolated in K12, no more note would be made of it than that it was a trivial variant of *exrA*.

(*f*) *Filamentation in K12* exrB. The difference between strain B and K12 relative to sensitivity to UV is that the former is *lon*, the latter *lon*$^+$. *lon* accounts for the filamentation of UV-irradiated strain B. *lon* derivatives of K12 have been isolated (Howard-Flanders, Simson & Theriot, 1964) and these are sensitive to UV and form filaments on irradiation. It was of interest to observe whether *exrB* derivatives of K12 *lon*$^+$ filamented. A microscopic examination of an overnight broth culture of PAM 5725 showed the population contained a few filaments usually at a frequency of 0·1–0·5 %. To determine the filament-inducing capacity of UV, a drop of a resting broth culture of PAM 5725 was placed on a JN agar plate, irradiated with 144 ergs/mm$^2$ and incubated overnight at 37 °C. When a surviving colony was teased apart with a toothpick and examined with a phase-contrast microscope at a magnification of 200× many short filaments were seen. No such filaments were observed with PAM 5717.

(*g*) *Transductions between Bs2 and PAM 26.* To determine the relationship between *exrA* and *exrB* the reciprocal transductions presented in Table 4 were performed: P1 PAM 23 × PAM 26 and P1 PAM 26 *mal*$^+$ × Bs2. In each transduction 100 *mal*$^+$ transductants were selected. The transductants, after purification, were examined for UV sensitivity and for spontaneous filamentation in broth

cultures incubated at 37 °C for 16–18 h and after irradiation on agar plates. The column headed $UV^S$ will be discussed first.

When Bs2 was used as donor 97% of the transductants of PAM 26 were non-filamenting and were indistinguishable in UV sensitivity from Bs2. These are considered to have incorporated the *exrA* gene. But there is no knowledge of the UV resistance and filamenting properties of a possible *exrA exrB* double mutant. The 97 UV-sensitive, non-filamenting recombinants could therefore include *exrA* and *exrA exrB* genotypes. If the latter were present, then the *exrA* phenotype would be epistatic to *exrB*. To establish the presence of double mutants would require an experiment in which one might attempt by transduction into strain B to demonstrate the presence of each cistron. Such tests were not undertaken.

When PAM 26 was used as donor, 42% of the *mal*$^+$ transductants were phenotypically indistinguishable from PAM 26; they were UV-sensitive and filamented spontaneously. Twenty per cent were still phenotypically like the recipient strain. Again there is no prior knowledge of whether either class includes an *exrA exrB* genotype.

Turning now to the column $UV^r$, one (of 100) transductants in the cross P1 Bs2 × PAM 26 was indistinguishable in its response to UV from strain B from which both Bs2 and PAM 26 were derived. In the reciprocal transduction 38% were like B. This phenotype is the result of a recombination between *exrA* and *exrB*. It is worth noting that the frequency of wild-type recombinants was high (38% and 35%) in two tests when PAM 26 was used as a donor, whereas the frequency was low (1%) when Bs2 was donor. The high frequency of wild-type recombinants in one direction suggests, but does not prove, that *exrA* and *exrB* are not allelic and that the order of markers is *malB exrA exrB*, since this configuration would require no extra cross-overs to achieve a wild-type genotype in cross I but would in cross II.

## 4. DISCUSSION

We are unaware of a description of a strain of *E. coli* with the phenotypic and genetic properties of PAM 26. It is sensitive to UV, X-rays and NG, which, in part, probably describes all radiation-sensitive mutants except *uvr* strains. It also differs from the latter in being HCR$^+$. The gene *exrB*, mutation in which is associated with all the properties of PAM 26 relevant to radiation sensitivity, is closely linked to *malB* and to the right of it on the conventional map of *E. coli*. In this respect, as well as in its response to radiation and chemicals, PAM 26 resembles an *exrA* (or *lex*) strain. However, it differs from *exrA* strains in one significant respect. Whereas the presence of an *exrA* gene in a *lon* strain, such as B, inhibits the formation of filaments following UV irradiation, PAM 26 forms filaments, not only after these treatments, but also spontaneously. Furthermore, though an *exrB lon*$^+$ K12 strain did form spontaneous filaments at low frequency, virtually the entire population exhibited a defect in cell division leading to elongated cells, when irradiated with UV. This suggests that *exrB* produces a defect in cell division

elicited by UV, independent of the defect associated with *lon* but additive with it, since *lon exrB* cells filament spontaneously. Since an *exrB lon*$^+$ strain does not filament spontaneously, such a phenotype appears to require both *lon* and *exrB*. Unless one examined mutants of the *exr* type for elongation after UV irradiation, or transferred the gene into a *lon* strain, one would miss *exrB* mutants, which may in fact exist among collections of *exrA* and *lex* mutants.

The immediate cause of spontaneous filamentation is unknown, but in PAM 26 (*exrB lon*) all the conditions known to prevent filamentation in irradiated *lon* strains, such as elevated temperature of incubation, growth in minimal medium, and presence of pantoyl lactone, do not significantly reduce filamentation. What completely eliminates the tendency to filament is the *sul* gene (Donch, Chung & Greenberg, 1969). Deferring detailed discussion of this to a later publication, we wish merely to state, that strain B/r into which *exrB* is transduced, though sensitive to UV, does not form filaments either spontaneously or after irradiation.

There is an awareness of the possibility of a cluster of genes near *malB* involved in radiation resistance (Sedgwick & Bridges, 1972), though until now this has been a cluster of two, *exrA* (*lex*) and *uvrA* (Donch & Greenberg, 1968; Mount, Low & Edmiston, 1972; Howard-Flanders *et al.* 1966). One might add the *ts DNA* gene found by Fangman & Novick (1968) in strain FA 22, considered now one of many mutants of *tsDNAB* linked to *malB*, described as sensitive to X-rays but not UV (Fangman & Novick, 1968). Mutants of *tsDNAB*, defective in DNA replication at elevated temperature, form lethal filaments at these temperatures, though the defect in cell division can be separated in some revertants from inhibition of DNA synthesis (Fangman & Novick, 1968). It is possible that *exrB* is an allele of *tsDNAB* or *exrA* or *uvrA* and this remains to be determined by further genetic studies. Preliminary results indicate that *exrB* and *tsDNAB* are not identical, and therefore there appears to be a cluster of now three or possibly four genes in the *malB* region all involved in radiation resistance or cell division or both. Definitive proof based on complementation would probably be impossible to obtain because *exrB* like *exrA* and *lex* is dominant (Mount, Low & Edmiston, 1972, and unpublished). We have not been able to recognize in the present studies strains of the genotype *exrA exrB*. All the recombinants examined were identifiable phenotypically with previously known strains.

The fact that *exrB* was isolated independently on two occasions would indicate it is probably not the result of two mutations. This is further confirmed by the fact that the tendency to filament in strains with a *lon* background, the chief distinguishing characteristic of *exrB* strains, accompanies the *exrB* gene in transduction. We shall show in a later publication that *exrB* reverts to *exrB*$^+$ at a frequency of about $2 \times 10^{-6}$, which observation further precludes the possibility that the phenotypes of PAM 26 and PAM 443 result from two *malB*-linked mutations.

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