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X-ray Sensitivity and Repair Capacity of a *polA1 exrA* Strain of *Escherichia coli* K-12

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A *polA1 exrA* strain of *Escherichia coli* K-12 was constructed. It was found to be more sensitive to aerobic or anoxic X irradiation than were mutants containing either *polA1* or *exrA* alone. The ability of *polA1 exrA* and related strains to repair X-ray-induced single-strand breaks in deoxyribonucleic acid DNA was examined. The *polA1* strain was deficient in type II (buffer) repair but not in type III (growth medium-dependent) repair. The *exrA* strain was not deficient in type II repair but was deficient in type III repair (similar to *rec* strains). The double mutant *polA1 exrA* was deficient in both type II and type III repair. Thus, the increased X-ray sensitivity of the *polA1 exrA* double mutant was correlated with its decreased ability to repair X-ray-induced single-strand breaks in DNA. We have tested the hypothesis that *polA rec* double mutants are not viable because they lack the types II and III systems for the repair of DNA single-strand breaks. Since the *polA1 exrA* strain is viable and is deficient in both of these repair processes, this hypothesis seems not to be correct.

Repair of X-ray-induced single-strand breaks in the deoxyribonucleic acid (DNA) of *Escherichia coli* K-12 has been shown to result from the action of at least three repair processes (C. D. Town, K. C. Smith, and H. S. Kaplan, submitted to Radiat. Res.). Type I repair acts preferentially on breaks produced by anoxic X irradiation and is complete within 1 to 2 min after irradiation, even at 0 C. Approximately 90% of the breaks remaining after type I repair are repaired by type II repair, which can occur in buffer and is complete within 10 to 20 min at room temperature. A medium-dependent repair process, termed type III repair, is able to repair a fixed number of the breaks (~2 per single-strand genome) in addition to those repaired by type II repair. Type III repair is complete within 40 to 60 min at 37 C. The characteristics of these three repair systems are summarized in Table 1.

The X-ray-sensitive *recA*, *recB*, and *recC* strains (10) have been shown to be deficient in the type III (medium-dependent) repair of X-ray-induced single-strand breaks (11; unpublished results). The *exrA* mutation, first isolated in the radiosensitive mutant B_{s-1} (7), is also deficient in type III repair, as judged from the results of McGrath and Williams (13). The *exrA* mutation results in a two- to fourfold decrease in proficiency of genetic recombination (3) compared with a 10^2-fold decrease for the *recB* or *recC* mutations and a 10^4-fold decrease for the *recA* mutation (8).

The *polA1* strain of *E. coli* K-12 is deficient in DNA polymerase I activity (2) and shows increased ultraviolet (UV) radiation (5) and X-ray (15, 18) sensitivity compared to the parent *pol*$^+$ strain. The X-ray sensitivity of the *polA* mutant appears to be related to the decreased ability of this strain to repair X-ray-induced single-strand breaks in DNA by type II repair (18).

The combination of the *recA* and *recB* mutations with *polA* have been found to be lethal (6, 14). The conditionally lethal, temperature-sensitive strains *polA12 recA* and *polA12 recB* have been prepared, and some of their properties have been described (14), but the reason for the inviability of these strains is as yet unknown. It was of interest to attempt the preparation of a *polA exrA* strain because of the similarity of the *recA*, *recB*, and *exrA* mutations in terms of defects in repair processes. If the deficiency in type III repair were a contributing cause of the lethality observed when *recA* or *recB* is placed in combination with *polA*, the *exrA* mutation might have a similar effect.

The *polA exrA* strain proved to be viable and is more X-ray sensitive than either the *polA* or

exrA strains. This paper reports the X-ray sensitivities and repair properties of these strains.

MATERIALS AND METHODS

Bacterial strains. The bacteria used, their genotypes, and the sources from which they were obtained are listed in Table 2.

Growth conditions. The transduction and conjugation techniques used were similar to those described by Monk and Kinross (14). The standard medium used for these procedures was LB broth (1).

For studies concerning the radiation sensitivity and repair capabilities of the various strains, a minimal medium (MM) supplemented with thiamine hydrochloride (0.5 μg/ml) was used (11). In addition, this was supplemented, as necessary, with L-methionine at 40 μg/ml and thymine at 10 μg/ml for overnight growth or at 2 μg/ml for exponentially growing cells.

LB and MM media with necessary supplements were solidified with 0.9% Oxoid agar-agar no. 3. The rhamnose MM agar and maltose MM agar used for the genetic experiments contained 0.4% of these sugars instead of glucose.

Survival after X irradiation. An overnight culture, grown in MM with thymine at 10 μg/ml, was diluted 1:50 in fresh MM containing thymine at 2 μg/ml. The cells were grown for several generations to 10^8 to 2×10^8 cells/ml, collected on 0.45-μm membrane filters (Millipore Corp.), washed with DTM buffer (MM without glucose), and resuspended in DTM buffer at 10^8 to 2×10^8 cells/ml. Irradiation was at room temperature with air or N_2 (~99.996% purity) bubbling prior to, and during, the X-ray exposure. The twin-tube 50 kV peak X-ray unit described by Loevinger and Huisman (12) was used at a dose rate of approximately 8.3 krad/min, depending upon the geometry of the irradiation vessels used. For some experiments, only one tube was operating, giving a dose rate of about 4.1 krad per min. Samples were diluted in buffer (11.7 g of Na_2HPO_4 and 7.1 g of KH_2PO_4 per liter, pH 7.0) and plated on MM agar supplemented as necessary.

Gradient centrifugation analyses. The procedures described by Town et al. (17) were adopted for these experiments with minor modifications. Cells were grown and irradiated as indicated above except that thymine-*methyl*-^3H (New England Nuclear; ~18 Ci/mmol) at 100 μCi/ml was present in the medium for exponentially growing cells.

Cells were incubated for 20 min at 37 C in DTM buffer after irradiation to allow completion of type II

TABLE 1. *Systems for the repair of X-ray-induced DNA single-strand breaks*[a]

Designation	Characteristics
Type I	Ultrafast (<2 min at 0 C); occurs in buffer; does not require DNA polymerase I; repairs ~ 75% of the X-ray-induced breaks produced under anoxic conditions but only ~ 25% of the breaks produced under aerobic conditions.
Type II	Fast ($T_{\frac{1}{2}}$ for aerobic X irradiation is ~10 min at 0 C, ~1 min at 37 C); occurs in buffer; is partially deficient in strains which lack DNA polymerase I; repairs ~90% of the breaks remaining after type I repair whether produced under aerobic or anoxic conditions.
Type III	Slow (40–60 min at 37 C); requires complete growth medium; controlled by *rec* and *exr* genes; does not require DNA polymerase I; repairs approximately two breaks per single-strand genome in addition to the breaks required by type II repair whether produced under aerobic or anoxic conditions.

[a] Compiled from C. D. Town, K. C. Smith, and H. S. Kaplan (19; submitted to Radiat. Res.) and present results.

TABLE 2. *List of strains*[a]

No.	Mating type	Genotype	Source
JG123	F⁻	W3110 *rha lacZ thy metE str malB*	J. D. Gross
JG78	Hfr R1	*rif metE polA1*	J. D. Gross
MM450	F⁻	W3110 *rha lacZ str recA56*	M. Monk
p3478	F⁻	W3110 *thy polA1*	J. Cairns
DY104	F⁻	W3110 *lacZ thy metE str malB*	This paper
DY105	F⁻	W3110 *lacZ thy metE str malB polA1*	This paper
DY98	F⁻	W3110 *lacZ thy metE str*	This paper
DY99	F⁻	W3110 *lacZ thy metE str exrA*	This paper[b]
DY100	F⁻	W3110 *lacZ thy metE str polA1*	This paper
DY101	F⁻	W3110 *lacZ thy metE str polA1 exrA*	This paper[b]
AB2494	F⁻	*thi metB his proA thr leu thy lex mtl xyl ara galK lacY str tsx sup*	P. Howard-Flanders
DY52	F⁻	*endA thy bio exrA*	—[b, c]

[a] Abbreviations are as used by Taylor (16).
[b] The *exrA* mutation was initially obtained from *E. coli* B_{s-1}.
[c] D. A. Youngs, Ph.D. thesis, The University of Michigan, Ann Arbor, 1971.

repair. Samples were then added to an equal volume of MM (with twice the normal concentrations of glucose and supplements) or DTM buffer and incubated for an additional 60 min. This allowed the measurement of the final extent of type II repair and a controlled measurement of the number of breaks repaired by the medium-dependent type III repair process. The cells were lysed by layering ~10^6 cells onto a 0.1-ml cap of 0.5% Sarkosyl (Geigy NL30) and 0.01 M ethylenediaminetetraacetic acid (EDTA) in 0.5 N NaOH (C. D. Town, K. C. Smith, and H. S. Kaplan, submitted to Radiat. Res.) on top of 4.8-ml linear gradients of 5 to 20% (wt/vol) sucrose in 0.1 N NaOH. After standing for 40 to 60 min, the gradients were centrifuged at 30,000 rpm for 105 min at 20 C in an SW50.1 rotor. The procedures for processing the gradients and analysing the data have been described (17).

Construction of strains. Strain JG123, *malB rha*, was used as the recipient in a cross with strain JG78, Hfr R1 *rha*$^+$ *polA1*. Exponentially growing cells were mixed at a donor to recipient ratio of 1:20 and incubated at 37 C without shaking for 15 min. Mating pairs were disrupted by mixing on a Vortex mixer, and dilutions were spread onto rhamnose MM agar supplemented with thymine, thiamine, methionine, and streptomycin at 100 μg/ml. The *rha*$^+$ recombinants were screened for the *polA1* marker by checking X-ray sensitivity, and the other markers were confirmed. Strains DY104, *pol*$^+$, and DY105, *polA1*, were retained. These strains were transduced to *mal*$^+$ with phage P1kc grown on the *exrA mal*$^+$ strain, DY52. The selection medium was maltose MM agar containing thiamine, thymine, and methionine. The *mal*$^+$ transductants were screened for X-ray sensitivity and were classified as *pol*$^+$ *exr*$^+$ (DY98), *pol*$^+$ *exrA* (DY99), *polA1 exr*$^+$ (DY100), and *polA1 exrA* (DY101). The unlinked markers *thy*, *lac*, *str*, and Rif^R were confirmed, as was sensitivity to phages P1 and λ.

Subsequently, it was demonstrated that phage P1kc grown on strains DY99 and DY101, but not DY98 or DY100, were able to cotransduce *mal*$^+$ and a radiation-sensitizing marker into a *malB exr*$^+$ strain (JG123) at a frequency of 70 to 85%. In addition, phage P1kc grown on the *pol*$^+$ *metE*$^+$ strain (AB2494) but not the *polA1 met*$^+$ strain (p3478) formed 21 to 55% radiation-resistant *met*$^+$ transductants, with either strain DY100 or strain DY101 as the recipient, indicating that strains DY100 and DY101 contain a radiation-sensitizing marker cotransducible with *metE*. These results support the classification of strains DY98, DY99, DY100, and DY101 given above.

RESULTS

Survival after X irradiation. The survival curves of the wild-type (DY98), *exrA* (DY99), *polA1* (DY100), and *polA1 exrA* (DY101) strains are shown in Fig. 1 for aerobic X irradiation and Fig. 2 for anoxic X irradiation. For comparison, the survival curves for the *recA56* (MM450) strain are also shown. The D_0 values obtained from these survival curves and

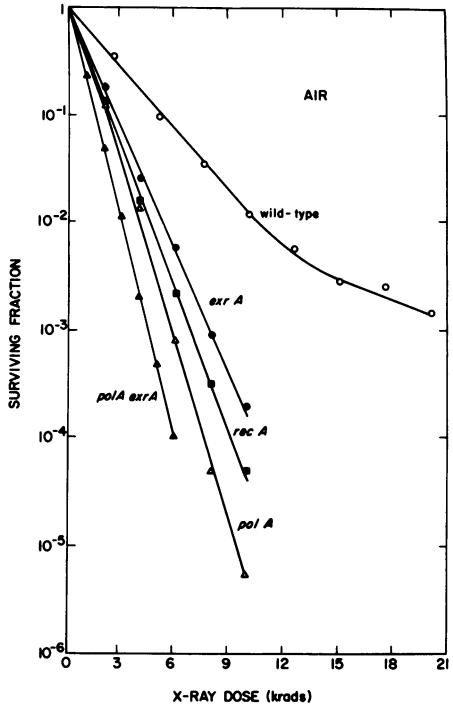

FIG. 1. *Survival after aerobic X irradiation. Symbols and strains are:* (○) *wild-type, DY98;* (●) *exrA, DY99;* (△) *polA1, DY100;* (▲) *polA1 exrA, DY101;* (■) *recA56, MM450. Cells were irradiated in DTM at room temperature, diluted in buffer, and plated on supplemented MM medium. The samples were bubbled with air prior to, and during, irradiation.*

the calculated oxygen enhancement ratios (OER) are listed in Table 3.

For aerobic X irradiation, the *polA1 exrA* strain was the most sensitive, with a D_0 value of about 0.6 krad. The D_0 values of the remaining strains increased in the order *polA1*, *recA56*, *exrA*, and wild type. The *polA1 exrA* and the *recA56* strains were the most sensitive to anoxic X irradiation, with D_0 values of 2.1 krad. D_0 values for the other strains increased in the order *exrA*, *polA1*, and wild-type.

The *recA* and *exrA* strains had OER values lower than that of the wild-type strain (Table 3). The OER values for the *polA1* and *polA1 exrA* strains, were about the same as that of the wild-type strain.

Repair of single-strand breaks. The ability of each of these strains to repair X-ray-induced single-strand breaks in DNA was measured by alkaline sucrose gradient centrifugation techniques. It was shown by Town, Smith, and Kaplan (submitted to Radiat. Res.) that type II repair was complete in the wild-type strain after approximately 15 min of incubation in buffer at room temperature. The

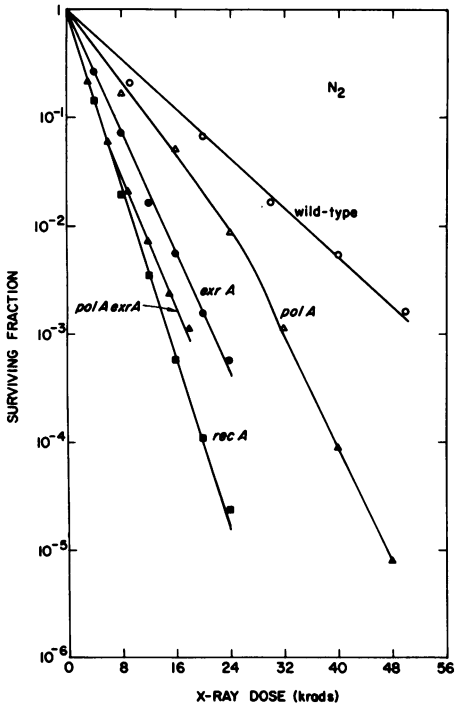

FIG. 2. *Survival after anoxic X irradiation. The symbols and procedures were the same as described in Fig. 1 except that cells were bubbled with N_2 instead of air.*

TABLE 3. *Calculated D_0 and OER values*

Strain	D_0 values (krad)[a]		OER[b]
	Aerobic X irradiation	Anoxic X irradiation	
Wild type (DY98)	2.3[c]	8.0	3.5
exrA (DY99)	1.2	3.1	2.6
polA1 (DY100)	0.8[d]	3.5[d]	4.4
polA1 exrA (DY101)	0.6	2.1[c]	3.5
recA56 (MM450)	1.0	2.1[c]	2.1

[a] Dose required to reduce survival by 63% on the exponential part of the survival curve. The D_0 values were obtained from the data shown in Fig. 1 and 2.

[b] OER (oxygen enhancement ratio) values were obtained by dividing the D_0 value for anoxic irradiation by the D_0 value for aerobic irradiation.

[c] The initial slope was used to calculate the D_0 value.

[d] The final slope was used to calculate the D_0 value.

repair in DTM buffer, characteristic of the *polA1* strain (DY100), was complete after about 20 min at 37 C or 40 min at room temperature (Fig. 3). The standard procedure used for further experiments was to incubate the cells either for 80 min in DTM buffer at 37 C to allow completion of type II (buffer) repair or for 20 min in DTM buffer followed by 60 min in minimal medium to allow the completion of medium-dependent type III repair.

Figure 4 shows the yield of breaks after incubation for 80 min at 37 C in DTM buffer. For comparison, the dashed line represents the yield of breaks remaining after the ultra-fast type I repair, as measured by Town et al. (19) for strains of *E. coli* K-12 closely related to those used in these experiments. The slope of the dashed line is 2.13 breaks per single-strand genome per krad. Strains containing the *polA1* mutation repaired 75% of the breaks remaining after type I repair, leaving 0.54 breaks per single-strand genome per krad (Fig. 4, middle line). The *pol*$^+$ strains repaired 91% of the breaks not repaired by type I repair, as judged from the ratio of slopes of the lower, solid line (0.19 breaks per single-strand genome) and the dashed line. In addition, the *pol*$^+$ strains repaired all the breaks produced by doses up to about 3.4 krad.

The amount of medium-dependent type III repair performed by each of the strains was calculated by subtracting the number of breaks

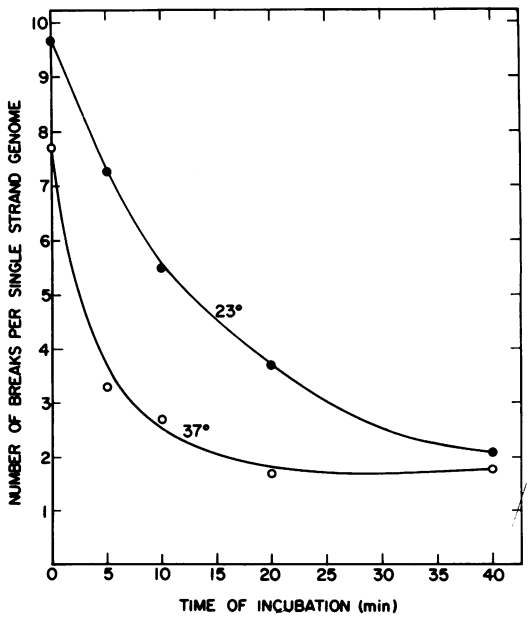

FIG. 3. *Influence of temperature on the rate of repair of X-ray-induced single-strand breaks in a polA1 strain (DY100) incubated in DTM. Cells were irradiated at room temperature (~23 C) and then incubated at the desired temperature. The X-ray dose used was 4 krad. Symbols are: (●) incubation at ~23 C; (○) incubation at 37 C.*

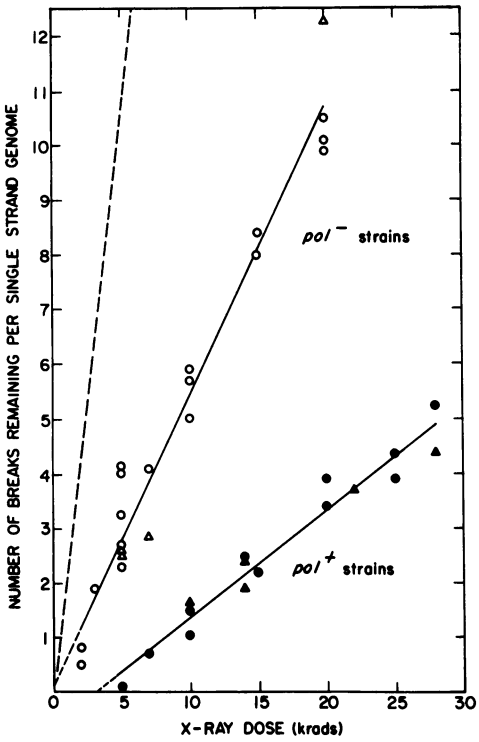

FIG. 4. *Repair of X-ray-induced single-strand breaks in DNA. Samples were incubated for 80 min in DTM at 37 C after X irradiation. Symbols and strains are:* (●) *wild-type, DY98;* (▲) *exrA, DY99;* (○) *polA1, DY100; and* (△) *polA1 exrA, DY101. The dashed line indicates the number of single-strand breaks remaining after the ultrafast, type I repair as measured by Town et al. (19).*

remaining after type III repair from the number left after type II repair, for a given X-ray dose. The results of several experiments for each strain are shown in Table 4. The wild-type strain (DY98) and the *polA1* strain (DY100) repaired an average of about 1.5 breaks per single-strand genome by type III repair. Strains containing the *exrA* mutation (DY99 and DY101) showed no significant type III repair. The average number of breaks per single-strand genome repaired by type III repair was −0.13 for the *exrA* strain (DY99) and 0.15 for the *polA1 exrA* strain (DY101).

DISCUSSION

Survival and repair capacities. The *polA1 exrA* double mutant is viable and is more X-ray sensitive than either the *polA1* or the *exrA* mutant (Table 3). The sensitivities of the *polA1 exrA* and related strains to UV radiation are currently under investigation.

The *exrA* strain is deficient in the medium-dependent type III repair of DNA single-strand breaks (Table 4) and in this respect is similar to the *recA*, *recB*, and *recC* strains (11; unpublished results). These data also clarify the results of McGrath and Williams (13) who observed no repair of single-strand breaks in the triple mutant *E. coli* B_{s-1} (Hcr^-, Fil^+, Exr^-) after incubation in growth medium, by demonstrating that the *exrA* mutation alone is sufficient to inactivate the type III repair system.

The *polA1* strain is partially deficient in type II repair (Fig. 4) but is not deficient in type III repair (Table 4). Therefore, DNA polymerase I is not required for type III repair.

The *polA1 exrA* double mutant is deficient in both type II (Fig. 4) and type III (Table 4) repair. This dual deficiency in repair is consistent with the enhanced sensitivity of this strain to X-ray-induced killing. These results are in

TABLE 4. *Extent of type III repair of X-ray-induced DNA single-strand breaks in strains of E. coli K-12*

Strain	X-ray dose (krad)	No. of single-strand breaks per single-strand genome[a]		
		Remaining after type II repair	Remaining after type III repair	Net amount of type III repair
Wild type (DY98)	14	2.5	1.5	1.0
	15[b]	2.5	1.5	1.0
	20	3.9	1.8	2.1
	20	3.2	1.4	1.8
	25[b]	3.9	3.2	0.7
exrA (DY99)	10	1.7	1.6	0.1
	14	2.4	2.6	−0.2
	22	3.7	4.0	−0.3
polA1 (DY100)	3[c]	2.0	0.0	2.0
	4[d]	1.2	0.2	1.0
	5[e]	3.2	1.2	2.0
	5	4.1	1.4	2.7
	7[c]	4.1	2.3	1.8
	8[d]	4.0	2.4	1.6
	10[e]	5.9	5.2	0.7
polA1 exrA (DY101)	5	2.5	2.5	0.0
	7	2.8	2.5	0.3

[a] Samples were incubated for either 80 min in DTM buffer at 37 C to allow completion of type II repair, or 20 min in DTM buffer and then 60 min in minimal medium at 37 C to allow completion of type III repair.

[b, c, d, e] Data with the same superscript are from the same experiment.

agreement with other findings which indicate that the chemical inhibition of type III repair in *polA* strains increases their sensitivity to X radiation. Thus, two irreversible inhibitors of type III repair, chloramphenicol (4) and dinitrophenol (E. Van der Schueren, K. C. Smith, and H. S. Kaplan, submitted to Radiat. Res.), sensitize *polA* strains to X radiation. In addition, the *lex* mutation, which is phenotypically quite similar to *exrA* (8), also sensitizes the *polA1* strain to killing by X radiation to the same extent as the *exrA* mutation (unpublished results).

Role of strand breaks in cell death. The number of single-strand breaks remaining after complete repair in the *polA1 exrA* strain (Fig. 4) was 0.54 per single-strand genome per krad (or 1.08 breaks per chromosome) for aerobic X irradiation. For a dose of 0.6 krad, the dose required per lethal event in this strain, there would remain 0.65 breaks per chromosome after complete repair. Thus, a maximum of 65% of the lethal damage could be due to unrepaired strand breaks.

The rate of production of double-strand breaks by aerobic X irradiation has been determined to be about 0.14 breaks per genome per krad in a closely related wild-type strain (C. D. Town, K. C. Smith, and H. S. Kaplan, manuscript in preparation). Assuming the same rate of break production in the *polA1 exrA* strain, this would result in about 0.08 double-strand breaks per 0.6 krad. Each double-strand break should be observed as two single-strand breaks. Thus, the value of 65% of the lethal damage which could be due to strand breakage should be corrected to 57%; 8% due to double-strand breaks and 49% to single-strand breaks. This would leave a balance of 43% of the lethal damage which cannot be accounted for by strand breakage and may be due to base damage.

The *uvr* mutants of *E. coli* K-12 are deficient in the excision repair of base damage induced by UV radiation and are also somewhat X-ray sensitive, indicating a possible involvement of the *uvr* gene products in the repair of X-ray-induced base damage (9). It has also been postulated that the X-ray sensitivity of the *recA* strain may be due in part to a deficiency in the repair of base damage (C. D. Town, K. C. Smith, and H. S. Kaplan, submitted to Radiat. Res.; unpublished results).

Similarly, the present results implicate both *exrA* and *polA* in some system for the repair of X-ray-induced damage in addition to DNA single-strand breaks. The *exrA* mutation sensitizes the wild-type strain by a factor of 1.9 (Table 3) but only sensitizes the *polA* strain by a factor of 1.3. Since in each case the *exrA* mutation results in a lack of type III repair, the increased sensitization of a wild-type strain to X rays by the introduction of an *exrA* mutation must be due to a defect in some additional repair system which is also dependent upon DNA polymerase I.

Repair of single-strand breaks by polA strains. Town et al. (18) demonstrated that no significant amount of repair of DNA single-strand breaks occurred in the *polA* strain during irradiation at 23 C. However, the present results indicate that considerable type II repair does occur in the *polA* strain if incubation in DTM buffer is continued after irradiation. Thus, this new repair process requires neither growth medium (type III repair) nor DNA polymerase I. Preliminary results suggest the involvement of DNA polymerase III in this repair process since it is absent in a *polA1 dnaE* strain at the restrictive temperature (unpublished results).

The involvement of type III repair in the viability of polA strains. Gross et al. (6) and Monk and Kinross (14) have reported that the *polA recA* and *polA recB* double mutants are not viable. The reason for this is not yet clear, but the hypothesis that we have tested is that these cells are inviable because they lack both the type II and type III repair processes for the repair of DNA single-strand breaks. Since the *polA exrA* strain is viable and is deficient in both of these repair processes, this hypothesis seems not to be true. Thus the function(s) of the *recA* and *recB* gene products necessary for the viability of *polA* strains remains to be determined.

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