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A76

Influence of a *uvrD* mutation on survival and repair of X-irradiated *Escherichia coli* K-12 cells

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The presence of a *uvrD* mutation increased the X-ray sensitivities of *E. coli* wild-type and *polA* strains, but had no effect on the sensitivities of *recA* and *recB* strains, and little effect on a *lexA* strain. Incubation of irradiated cells in medium containing 2,4-dinitrophenol or chloramphenicol decreased the survival of wild-type and *uvrD* cells, but had no effect on the survival of *recA*, *recB* and *lexA* strains. Alkaline sucrose gradient sedimentation studies indicated that the *uvrD* strain is deficient in the growth-medium-dependent (Type III) repair of DNA single-strand breaks. These results indicate that the *uvrD* mutation inhibits certain rec^+lex^+-dependent repair processes, including the growth-medium-dependent (Type III) repair of X-ray-induced DNA single-strand breaks, but does not inhibit other rec^+lex^+-dependent processes that are sensitive to 2,4-dinitrophenol and chloramphenicol.

1. Introduction

The lesions produced by ionizing radiation in DNA include single- and double-strand breaks (Town, Smith and Kaplan 1973 a), and several types of base damage (Cerutti 1975). These lesions are repairable to varying extents, depending on the repair capacity of the irradiated cell and on the chemical nature of the specific lesions in question.

The possibility that excision repair of DNA base damage occurs after ionizing irradiation has received much support from *in vitro* studies (Cerutti 1976). The work of Hariharan, Remsen and Cerutti (1975) indicates that such excision repair occurs both in bacterial and mammalian cell systems. The reports of endonuclease activities that act specifically on γ- or X-irradiated DNA (Paterson and Setlow 1972, Hariharan and Cerutti 1974, Strniste and Wallace 1975), indicate that the first step in this excision-repair process is probably an endonucleolytic incision near the radiation product, analogous to the U.V. excision-repair process (Grossman 1974).

DNA strand breaks that remain in the cell after repair is completed arise from three sources: (i) initial radiation-induced single-strand breaks, (ii) initial radiation-induced double-strand breaks, and (iii) enzymatically-induced single- and double-strand breaks arising during the excision repair of base damage. Two operationally-identifiable repair processes acting on DNA single-strand breaks have been described (Town *et al.* 1973 b): a growth medium-independent process (Type II repair), and a growth medium-dependent process (Type III

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repair). The ultrafast, Type I repair process postulated by Town *et al.* (1972) has now been shown to be due, at least largely, to non-enzymatic radiochemical processes (Roots and Smith 1974, Johansen 1975, Sapora, Fielden and Loverock 1975, Palcic and Skarsgard 1975) and will not be discussed here.

The increased sensitivities of several radiosensitive mutant strains of *E. coli* K–12 have been shown to be correlated with deficiencies in DNA strand-break repair. The *recA*, *recB* (Kapp and Smith 1970), *polC* (Hamelin, Youngs and Smith 1976), and *lexA* (Sedgwick and Bridges 1972, Youngs and Smith 1973) strains are deficient in the growth-medium-dependent (Type III) repair process, whereas the *polA* (Town *et al.* 1971, Youngs and Smith 1973) mutation results in a deficiency in the growth-medium-independent (Type II) repair process. The *polC* mutation also decreases the extent of Type II repair, but only if the cell also contains a *polA* mutation (Hamelin *et al.* 1976). In addition, post-irradiation incubation with 2,4-dinitrophenol (Van der Schueren *et al.* 1973 b), chloramphenicol (Ganesan and Smith 1972) or quinacrine (Fuks and Smith 1971) inhibits the Type III repair process, and sensitizes rec^+ lex^+ cells to killing by ionizing radiation.

The *uvrD* mutation was first described by Ogawa, Shimada and Tomizawa (1968) and was found to result in a marked sensitization to U.V.-radiation, and a lesser sensitizing effect after γ-irradiation. The *uvrD* mutation interferes with the rejoining of incision breaks in DNA produced during the U.V. excision-repair process (Shimada, Ogawa and Tomizawa 1968, Youngs *et al.* unpublished results), and also decreases the extent of post-replicational repair after U.V.-irradiation (Youngs and Smith 1976 a).

In this paper we have investigated the influence of a *uvrD* mutation on survival and repair processes of *E. coli* K–12 cells after ionizing irradiation. The results indicate that the *uvrD* mutation inhibits rec^+ lex^+-dependent repair processes, including the growth-medium-dependent (Type III) repair of DNA single-strand breaks, but does not inhibit other rec^+ lex^+-dependent processes that are sensitive to 2,4-dinitrophenol and chloramphenicol.

2. Materials and methods

2.1. *Bacterial strains*

The properties and sources of the strains of *E. coli* K–12 used in the present experiments are given in table 1. The transduction and mating techniques for the genetic crosses have been described (Youngs and Smith 1973). In each case the indicated nutritional marker was first selected and the presence or absence of the desired radiation-sensitizing mutation was subsequently determined by checking U.V., X-ray or MMS sensitivity.

2.2. *Media*

The growth medium used for all survival and repair experiments was the glucose-salts minimal medium (MM) described by Ganesan and Smith (1968). MM medium was supplemented as necessary with amino acids (10^{-3} M), thiamine hydrochloride (1·5 μg/ml), biotin (5 μg/ml), and thymine (10 μg per ml for overnight cultures and 2 μg per ml for exponentially-growing cells). MM medium was solidified by the addition of 1·6 per cent Difco Noble agar (MM agar).

Designation	Genotype†	Source
KH21	F⁻ *leuB bio metE thyA thyR rha lac str malB*	R. B. Helling
JC5088	Hfr KL16 *recA56 thr ilv spc str*	J. Gross
AB2497	F⁻ *thr leu thi arg his pro thyA thyR lac gal mtl xyl ara str tsx*	R. P. Boyce
AB2470	F⁻ *recB21 thr leu thi arg his pro lac gal mtl xyl ara str tsx*	R. P. Boyce
SR255	F⁻ *recB21 leu thr thi pro arg his lac ara gal mtl xyl str tsx thyR*	P_1·AB2470 × AB2497 (Select Thy⁺)
N14–4	F⁻ *uvrD3 trp gal str*	H. Ogawa
DY98	F⁻ *metE lac str thyA thyR*	Youngs and Smith (1973)
DY99‡	F⁻ *lexA101 metE thyA thyR lac str*	Youngs and Smith (1973)
DY100	F⁻ *polA1 metE thyA thyR lac str*	Youngs and Smith (1973)
DY182	F⁻ *thyA thyR lac str*	P_1·N14–4 × DY100 (Select Met⁺)
DY183	F⁻ *polA1 thyA thyR lac str*	P_1·N14–4 × DY100 (Select Met⁺)
DY184	F⁻ *uvrD3 thyA thyR lac str*	P_1·N14–4 × DY100 (Select Met⁺)
DY185	F⁻ *polA1 uvrD3 thyA thyR lac str*	P_1·N14–4 × DY100 (Select Met⁺)
MM450	F⁻ *recA56 lac rha str*	J. Gross
DY176‡	F⁻ *lexA101 thyA thyR lac str*	P_1·N14–4 × DY99 (Select Met⁺)
DY177‡	F⁻ *uvrD3 lexA101 thyA thyR lac str*	P_1·N14–4 × DY99 (Select Met⁺)
DY130	F⁻ *recB21 metE lac str thyR*	P_1·AB2470 × DY98 (Select Thy⁺)
DY175	F⁻ *uvrD3 thyA thyR leuB bio rha lac str malB*	P_1·N14–4 × KH21 (Select Met⁺)
DY187	F⁻ *uvrD3 recA56 leuB bio rha lac str malB thyR*	JC5088 × DY175 (Select Thy⁺)
DY189	F⁻ *uvrD3 recB21 leuB bio rha lac str malB thyR*	P_1·DY130 × DY175 (Select Thy⁺)

† Symbols are as used by Bachmann, Low and Taylor (1976).

‡ The *lexA101* notation is used to indicate the *exrA* mutation originally from *E. coli* B_{s-1} (Mount and Donch 1976).

Table 1. List of bacterial strains.

2,4-Dinitrophenol was obtained from Fisher Scientific Co., and chloramphenicol (chloromycetin) from Parke, Davis & Co. Fresh solutions were prepared for each experiment.

2.3. *Survival curves*

For the survival studies, overnight stationary-phase cultures were diluted in fresh MM medium to about 10^7 cells/ml, and grown for 3 to 6 hours at 37°C to exponential phase (1 to $1{\cdot}5 \times 10^8$ cells/ml). The cells were collected on a Millipore filter (0·45 μm pore size), and resuspended at the same cell concentration in DTM buffer (MM medium without glucose or supplements).

The irradiation procedure was generally the same as previously described (Van der Schueren *et al.* 1973 b). Five millilitre samples were placed in plastic Petri dishes (60 mm diameter), or in a Plexiglas chamber and irradiated at room temperature with a twin-tube, beryllium-window X-ray unit (50kVp, 50 and 48 mA, 0·3 mm Al filtration) at a dose-rate of ~8 krad/min, as measured by ferrous sulphate dosimetry. Samples irradiated in the Plexiglas chamber

were bubbled with air before and during irradiation. Alternatively, the cells were irradiated with γ-rays from an 8 kCi ^{137}Cs source under aerobic or anoxic conditions, as described by Bonura, Youngs and Smith (1975). After irradiation, the cells were diluted in 0·067 M phosphate buffer (pH 7·0), and plated on MM agar. Colonies were counted after incubation for 48 to 72 hours at 37°C.

For the experiments with 2,4-dinitrophenol (DNP) and chloramphenicol (CAP), a sample of cells in DTM buffer was added to an equal volume of DTM buffer containing the required concentrations of drug and supplements to constitute complete MM medium containing CAP (at 100 μg/ml) or DNP (at 3×10^{-3} M). The samples were incubated at 37° for 90 min (Van der Schueren *et al.* 1973 b, Ganesan and Smith 1972) before dilution and plating on MM agar. The survival of unirradiated cells after the drug treatment was 80 per cent or greater in each experiment.

All of the survival results shown are the average of two or more independent experiments.

2.4. *Alkaline sucrose gradients*

The extent of DNA single-strand breakage was determined using alkaline sucrose gradient techniques (Youngs and Smith 1976 b). An overnight culture was diluted to about 10^7 cells/ml in MM medium containing thymine-*methyl*-^3H (New England Nuclear, > 12 Ci/mmol) at 100 μCi/ml, with a total thymine concentration of 2 μg/ml. The culture was incubated to a final density of about $1·2 \times 10^8$ cells/ml, at which time the cells were collected and irradiated in the same way as for the survival experiments. After irradiation, samples were added to an equal volume of DTM buffer, or DTM buffer containing twice the concentration of glucose and required supplements to give complete MM medium. The samples were then incubated for 80 min at 37°C.

For the experiments described in figure 5, the cells were lysed using the method described by Town *et al.* (1973 b). A 0·05 ml sample of cells ($\sim 10^6$ cells) was layered onto a 0·1 ml lysis cap [0·5 per cent Sarkosyl (Geigy NL30), 0·01 M EDTA (ethylenediaminetetraacetic acid), and 0·5 N NaOH] just previously layered on top of a 4·8 ml linear alkaline sucrose gradient [5 to 20 per cent (wt/vol) sucrose in 0·1 N NaOH]. The samples were centrifuged after standing a minimum of 40 min at room temperature.

For the Type III repair experiments shown in figure 6, the cells were lysed using a lysozyme method similar to that described by Kapp and Smith (1970). Samples of 0·3 ml volume were placed on ice. When all samples had been taken, the following additions were made: 0·05 ml of 32 mM EDTA, 0·03 ml of 30 per cent sucrose in 0·6 M tris(hydroxymethyl)aminomethane (pH 8·1), and 0·08 ml of lysozyme at 1 mg per ml. The samples were then incubated for 5 to 15 min on ice and a volume of 0·05 ml was layered onto a 0·2 ml cap of 0·5 N NaOH, which had just been layered on top of a sucrose gradient (described above). These samples were centrifuged after standing for a minimum period of 10 min at room temperature. The lysozyme method seemed to give slightly larger DNA molecular weight values than the Sarkosyl technique.

The procedures for centrifugation, fractionating the gradients, processing the samples, and calculating DNA molecular weights have been described (Youngs and Smith 1976 b).

3. Results

The survival data are shown in figures 1–4, and are summarized in table 2. The *uvrD* mutation increased the sensitivity of the wild-type and *polA* cells to killing by aerobic X-radiation (figure 1). The *uvrD* mutation gave similar degrees of sensitization (~ 1·6-fold) in both the wild-type and *polA* backgrounds (table 2).

Strain	D_0 values (krad)†	O.e.r. or sensitization factor‡
^{137}Cs γ-irradiation		
Wild-type (aerobic)	3·7	O.e.r. = 3·68
Wild-type (anoxic)	13·6	
uvrD (aerobic)	2·4	O.e.r. = 2·92
uvrD (anoxic)	7·0	
X-irradiation		
Wild-type	3·0	
Wild-type + DNP	1·9	1·6
Wild-type + CAP	1·8	1·7
uvrD	1·8	1·7
polA	0·92	
polA uvrD	0·58	1·6
recA	1·14	
recA uvrD	1·14	1·0
recB	1·18	
recB uvrD	1·18	1·0
lexA	1·38	
lexA uvrD	1·20	1·2
uvrD + CAP or DNP	1·30	1·4

† The D_0 values (defined as the radiation dose required to reduce survival by a factor of *e* on the exponential part of the survival curve) were calculated from the survival curves in figures 1–4 as the dose required to reduce the surviving fraction from 1×10^{-2} to $3{\cdot}7 \times 10^{-3}$.

‡ The o.e.r. value is the ratio of the D_0 value under anoxic conditions to the D_0 value for aerobic conditions. The sensitization factor is the ratio of the D_0 value for the $uvrD^+$ strain or non-drug-treated sample to that for the *uvrD* strain or drug-treated sample, respectively.

Table 2. Survival of *uvrD* strains of *E. coli* K-12 after ionizing irradiation.

The sensitivities of the *recA* and *recB* strains were not altered by the additional presence of the *uvrD* mutation (figure 2). The *lexA* strain was only sensitized ~ 1·2-fold by the *uvrD* mutation (figure 2 and table 2), compared with the ~ 1·6-fold effect observed with the wild-type and *polA* strains. However, the *uvrD* strain was sensitized by the additional presence of a *polA*, *lexA*, *recA* or *recB* mutation (see table 2).

Incubation for 90 min in MM medium containing either CAP or DNP sensitized the wild-type and *uvrD* strains to killing by aerobic X-irradiation (figure 3). Both drugs showed about the same sensitizing effect; ~ 1·6 fold for wild-type cells and ~ 1·4-fold for *uvrD* cells (table 2). The level of survival observed with *uvrD* cells treated with DNP or CAP was very similar to that of the *lexA* strain (table 2).

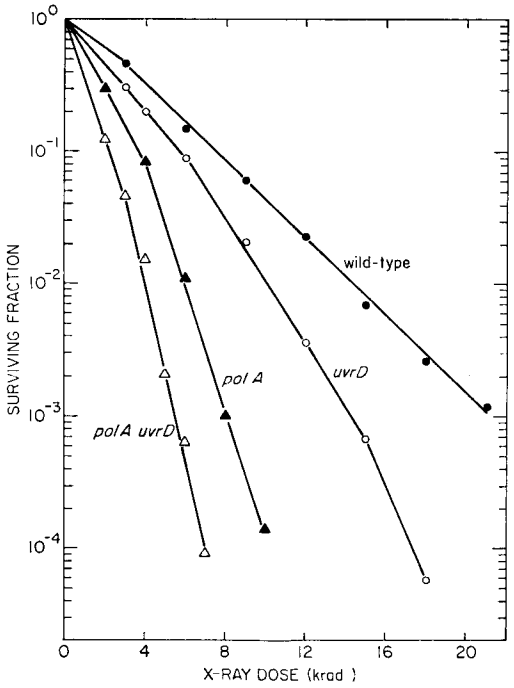

Figure 1. Survival of wild-type (DY182), *uvrD* (DY184), *polA* (DY183) and *polA uvrD* (DY185) strains of *E. coli* K-12 after X-irradiation. Samples were irradiated in DTM buffer with 50 kVp X-rays under aerobic conditions, and then were diluted and plated on MM agar to allow colony formation. Symbols: ● = wild-type; ○ = *uvrD*; ▲ = *polA*; △ = *polA uvrD*.

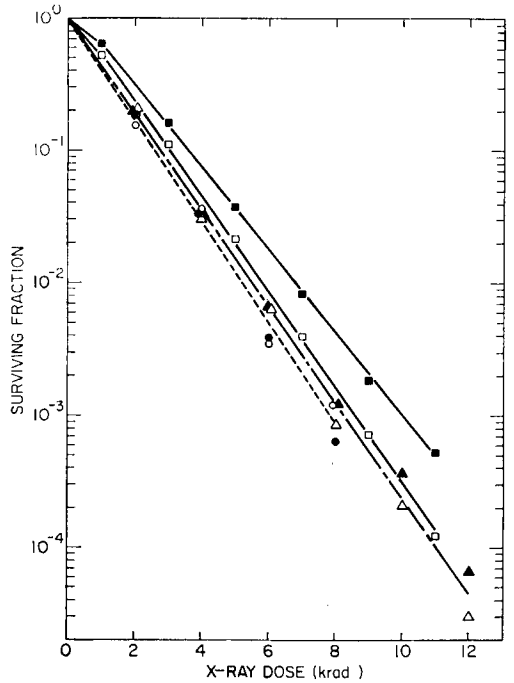

Figure 2. Survival of *recA* (MM450), *recA uvrD* (DY187), *recB* (DY130), *recB uvrD* (DY189), *lexA* (DY176) and *lexA uvrD* (DY177) strains of *E. coli* K-12 after aerobic X-irradiation. The procedure used is indicated in the legend to figure 1. Symbols: ● = *recA*; ○ = *recA uvrD*; ▲ = *recB*; △ = *recB uvrD*; ■ = *lexA*; □ = *lexA uvrD*.

The effect of the *uvrD* mutation on survival after ^{137}Cs γ-irradiation under aerobic and anoxic conditions was also examined (figure 4). The sensitizing effect of the *uvrD* mutation was greater for anoxic than for aerobic irradiation, as indicated by a lower oxygen enhancement ratio (o.e.r.) for the survival of *uvrD* cells than for wild-type cells (table 2).

The extent of repair of DNA strand breaks was determined for the *uvrD* strain and the related wild-type strain. The yield of unrepaired strand breaks observed for these two strains was not significantly different after incubation in DTM buffer, i.e. after completion of Type II repair (figure 5).

Post-irradiation incubation in MM medium rather than DTM buffer permits the occurrence of the growth-medium-dependent, Type III repair process, and resulted in the additional repair of DNA single-strand breaks in wild-type cells (figure 6 (*a*)). However, *uvrD* cells were deficient in the Type III repair process, as exemplified in figure 6 (*b*).

The extent of X-ray induced DNA degradation occurring in the wild-type and *uvrD* cells was determined (table 3). Samples were incubated for 80 min in DTM buffer at 37°C after X-ray doses ranging from 2 to 40 krad. In every case, the *uvrD* cells showed a more extensive breakdown of DNA to acid-soluble material than did the wild-type strain.

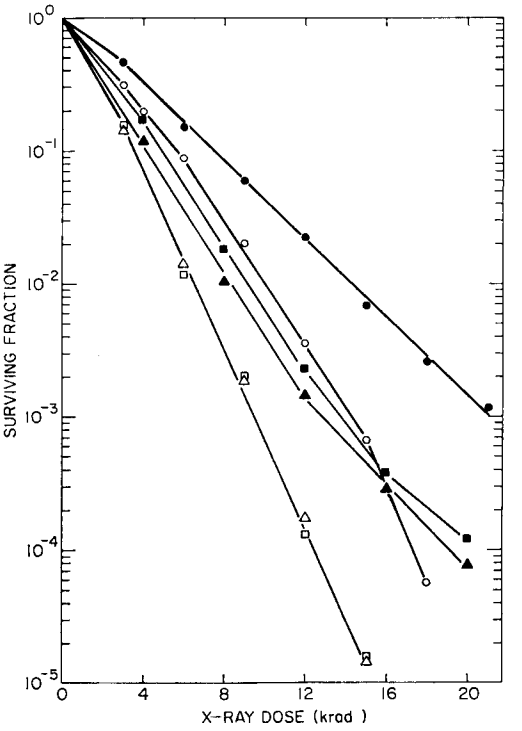

Figure 3. The effect of chloramphenicol (CAP) or 2,4-dinitrophenol (DNP) on the survival of wild-type (DY182) and *uvrD* (DY184) cells after aerobic X-irradiation. The procedure was the same as that given in the legend for figure 1, except that cells to be treated with CAP or DNP were incubated for 90 min at 37°C in MM medium containing the drug before dilution and plating. The drug concentrations used were 100 μg/ml for CAP, and 3×10^{-3} M for DNP. The symbols are: wild-type (closed symbols) and *uvrD* strains (open symbols): (●, ○)=no drug treatment; (■,□)=DNP treatment; (▲, △)=CAP treatment.

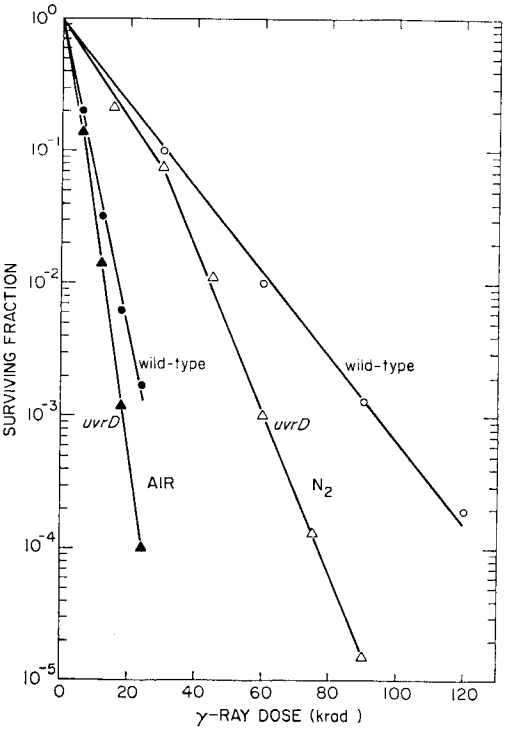

Figure 4. Survival of wild-type (DY182) and *uvrD* (DY184) strains of *E. coli* K-12 after aerobic and anoxic ^{137}Cs γ-irradiation. Samples were irradiated at room temperature in DTM buffer, and were bubbled with air or N_2 before and during irradiation. Samples were then diluted and plated on MM agar to allow colony formation. The symbols for aerobic and anoxic irradiation, respectively, are: ●, ○ = wild-type; ▲, △ = *uvrD*.

Strain	Percentage of DNA remaining TCA insoluble after aerobic X-ray doses (krad) of				
	2	5	10	20	40
Wild-type (DY182)	97	78	68	33	26
uvrD (DY184)	82	58	27	8	6

Samples were irradiated with the indicated doses and incubated for 80 min in DTM buffer at 37°C. Triplicate samples were placed on filter paper discs that had been soaked in 10 per cent trichloroacetic acid (TCA) and dried. The samples were processed as previously described (Youngs and Smith 1976 a). The values represent the average of two independent experiments.

Table 3. DNA degradation after X-irradiation.

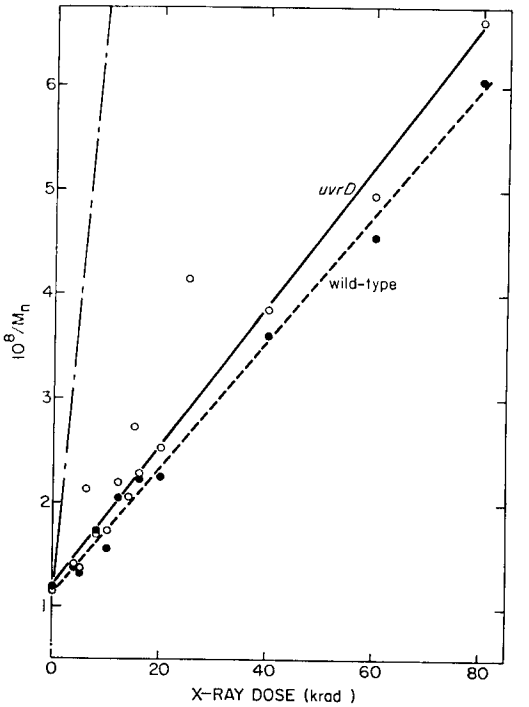

Figure 5. The Type II repair of DNA single-strand breaks in *E. coli* K-12 wild-type (DY182) and *uvrD* (DY184) strains after aerobic X-irradiation. The cells were irradiated as indicated in the legend for figure 1. Irradiated samples were incubated in DTM buffer for 80 min at 37°C to allow Type II repair to occur. The number-average molecular weight (Mn) of each DNA sample was then determined by alkaline sucrose gradient analysis. The inverse molecular weight values are plotted here as a function of the X-ray dose. Each point represents the average of at least two independent values. Lines were fitted to these data by linear-regression analysis, and have slopes that are equivalent to the yield of breaks per 10^8 daltons per krad. The slopes, their standard deviations, and the total number of data points for each line are: wild-type (●) $0{\cdot}061 \pm 0{\cdot}002$ per 10^8 per krad, $n=61$; *uvrD* (○) $0{\cdot}067 \pm 0{\cdot}003$, $n=89$. The initial yield of DNA single-strand breaks [1·16 per 10^8 daltons per krad (Youngs and Smith 1976 c)] is indicated (— — — —) for comparison.

4. Discussion

The presence of a *uvrD* mutation sensitized wild-type and *polA* cells to killing by ionizing radiation to similar extents (table 2). However, the *recA* and *recB* strains were not sensitized by the *uvrD* mutation, and the *lexA* strain was sensitized only slightly (table 2). These results suggest that the *uvrD* mutation inhibits repair processes that are present in wild-type and *polA* cells, but is largely deficient in *lexA* cells and completely lacking in *recA* and *recB* mutants; i.e. *uvrD* cells are deficient in a rec^+ lex^+-dependent process,

Our data also indicate that the *uvrD* mutation results in a greater sensitization to killing by anoxic γ-irradiation than aerobic γ-irradiation. This is indicated by the reduced o.e.r. value for *uvrD* cells (table 2). The *lexA* (Youngs and Smith 1973) and *recA* (Rupp, Zipser, von Essen, Reno, Prosnitz and Howard-Flanders 1970) mutations have also been shown to sensitize more extensively to anoxic than aerobic irradiation. These findings suggest that $uvrD^+$, rec^+,

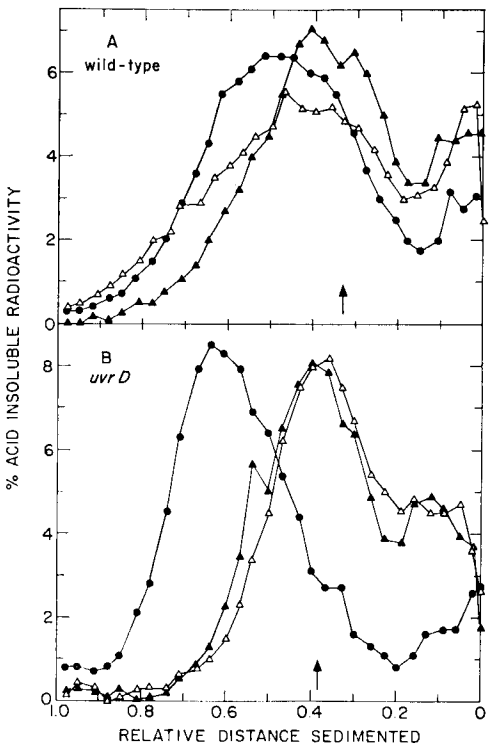

Figure 6. Type III repair of DNA strand breaks in *E. coli* K-12 wild-type (DY182) and *uvrD* (DY184) strains. The cells were irradiated as indicated in the legend to figure 1. Irradiated cells were incubated in DTM buffer or MM medium for 80 min at 37°C to allow repair to occur. The extent of Type III repair is indicated by the difference in position between the gradient profiles for DNA from irradiated cells incubated in MM medium and DTM buffer. Symbols are: 0 krad, 80 min incubation in MM medium (●); 22 krad, 80 min incubation in DTM buffer (▲); 22 krad, 80 min incubation in MM medium (△). The arrows indicate the sedimentation position for bacteriophage T_2 DNA. The Mn values for the unirradiated control samples were $0{\cdot}90 \times 10^8$ and $1{\cdot}04 \times 10^8$ daltons for the wild-type and *uvrD* strains, respectively.

lex$^+$-dependent repair processes are more important in the repair of anoxic than aerobic irradiation damage.

It has been demonstrated previously that the *recA*, *recB* (Kapp and Smith 1970, Youngs and Smith, unpublished results), and *lexA* (Sedgwick and Bridges 1972, Youngs and Smith 1973) strains are deficient in the growth-medium-dependent, Type III repair of DNA single-strand breaks after ionizing irradiation but are not deficient in the growth-medium-independent, Type II repair process. Since the survival results indicated that the sensitizing effect of the *uvrD* mutation involves the inhibition of a *rec*$^+$*lex*$^+$-dependent process, it seemed likely that the *uvrD* strain might be deficient in the Type III repair process but not in the Type II repair process. This proved to be the case (figures 5 and 6).

In addition to strand-break repair processes, certain results indicate that other *rec*$^+$*lex*$^+$-dependent processes are also important to cell survival (Town

et al. 1973 b, Youngs and Smith 1973, Van der Schueren *et al.* 1973 a). The current results that support this conclusion are as follows:

(i) The *uvrD* mutation (table 2) and treatment with CAP (Ganesan and Smith 1972) or DNP (Van der Schueren *et al.* 1973 b) sensitize only *rec*$^+$*lex*$^+$ cells, indicating that they inhibit *rec*$^+$*lex*$^+$-dependent repair processes. Yet, neither the *uvrD* mutation nor CAP or DNP treatment produces as large a sensitizing effect as do the *recA*, *recB*, or *lexA* mutations (table 2). Since *uvrD* (figure 6), CAP (Ganesan and Smith 1972), and DNP (Van der Schueren *et al.* 1973 *b*) are each able to block the *rec*$^+$*lex*$^+$-dependent Type III repair process, this suggests that the *recA*, *recB*, and *lexA* mutations must inhibit some repair function in addition to their known effect on Type III repair.

(ii) *uvrD* cells are sensitized by CAP or DNP treatment and, conversely, the *uvrD* mutation sensitizes CAP or DNP-treated wild-type cells (table 2). Since either the *uvrD* mutation or drug treatment alone is sufficient to block Type III repair, it appears that *uvrD*, CAP, and DNP must all block other processes in addition to Type III repair. In fact, a *uvrD* strain treated with CAP or DNP is approximately as sensitive as the *lexA* strain (table 2), suggesting that these other *rec*$^+$*lex*$^+$-dependent processes may be operationally split into two components, one inhibitable by the *uvrD* mutation and a second that is sensitive to CAP and DNP treatment.

These additional repair processes could include any process whose inhibition does not increase the final yield of strand breaks in DNA labelled before irradiation (the conditions of our alkaline sucrose gradient assay). This would include the repair of damage in DNA synthesized after irradiation, analogous to the post-replicational repair process occurring after U.V.-irradiation (e.g., Youngs and Smith 1976 a).

5. Summary

The DNA repair data indicate that the *uvrD* mutation results in a deficiency in the *rec*$^+$*lex*$^+$-dependent, Type III repair of DNA single-strand breaks, but has no significant effect on the Type II repair process. The survival data indicate that the *uvrD* mutation and CAP or DNP treatment, as well as the *recA*, *recB*, and *lexA* mutations, also result in a deficiency in cellular repair processes other than the Type III repair of DNA single-strand breaks.

Acknowledgments

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La présence d'une mutation *uvrD* augmente la radiosensibilité d'une souche sauvage *E. coli* et d'une souche *polA* mais n'a pas d'influence sur la sensibilité des souches *recA* et *recB* et peu d'effet sur une souche *lexA*. Une incubation de cellules irradiées dans un milieu contentant du dinitrophénicol −2, 4 ou du chloramphénicol diminue la survie de cellules de souche sauvage ou *uvrD*, mais n'influence pas la survie des souches *recA*, *recB* et *lexA*.

Des études portant sur la vitesse de sédimentation dans des gradients alkalins de sucrose indiquent que la souche *uvrD* est déficiente dans le processus de réparation de cassures mono-caténiques de l'ADN qui se passe normalement dans un milieu de croissance (type III).

Ces résultats indiquent que la mutation *uvrD* interfère avec des processus de réparation qui sont déterminés par les gènes *rec*$^+$ *lex*$^+$, entre autres la réparation dans un milieu de croissance de cassures mono-caténiques de l'ADN produites par des rayons-X mais n'influence pas les processus de réparation qui dépendent de *rec*$^+$ *lex*$^+$ et qui sont sensibles au dinitrophénol – 2, 4 et au chloramphénicol.

Die Anwesenheit einer *uvrD* Mutation erhöhte die Empfindlichkeit für Röntgenstrahlen beim *E. coli* wild Typ und der *polA* Linie, aber hatte keinen Einfluss auf die Strahlempfindlichkeit von *recA* und *recB* Linien, und wenig Einfluss bei einer *lexA* Linie.

Inkubation von bestrahlten Zellen in einem Medium das 2,4 Dinitrophenol enthalt, verringerte das Überleben von wild-typ und *uvrD* Zellen, aber hatte keinen Einfluss auf das Überleben von *recA*, *recB* und *lexA* Linien. Sedimentation in alkalischen Sucrose Gradienten deutete daraufhin, dass die *uvrD* Linie keine Reparatursprozesse von Einzelketten von DNA Bruchen, die normalwerweise im Wachstumsmedium auftritt, durchführen kann.

Diese Resultate deuten daraufhin, dass die uvrD Mutation bestimmte Reparatursprozesse, welche anhängig sind von *rec*$^+$*lex*$^+$, hindert. Bei diesen ist die Wachstumsmediumabhängige Reparatur von durch Röntgenstrahlen bedington Einzelketten Bruchen in DNA (Type III) mit einbergriffen. Die sonstigen *rec*$^+$*lex*$^+$ abhängigen Prozesse die empfindlich sind gegen 2,4 Dinitrophenol und Chloramphenicol werden aber nicht behindert.

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