

INHIBITION OF THE *exrA* GENE-DEPENDENT BRANCH OF THE DNA EXCISION REPAIR SYSTEM IN *ESCHERICHIA COLI* K-12 BY 2,4-DINITROPHENOL

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Abstract—Ultraviolet (UV)-irradiated *E. coli* K-12 wild-type cells were sensitized by a post-irradiation treatment with 10^{-2} M 2,4-dinitrophenol (DNP). This effect was not seen in strains carrying a *uvr* mutation, suggesting that DNP interferes with the excision repair process. The *polA* strain was sensitized to the same extent as the wild-type strain, while the *exrA* strain was not affected by DNP treatment.

Recombination deficient strains (*recA*, *recB* and *recA recB*) were protected by DNP treatment after UV irradiation. This protection was abolished by the addition of a *uvr* mutation (i.e., in strains *recA uvrB* and *recB uvrB*).

Alkaline sucrose gradient sedimentation studies showed that DNP treatment interfered with the rejoining of DNA single-strand breaks induced by the excision repair process. This interference was apparently specific for the *exr* gene-dependent branch of the *uvr* gene-dependent excision repair process, since the *uvr* and *exr* strains were not sensitized while the wild-type and *polA* strains were sensitized.

INTRODUCTION

Bacteria have a remarkable capacity for the repair of ultraviolet (UV)-induced DNA damage (Howard-Flanders, 1968; Smith, 1971; Setlow and Setlow, 1972); the two main dark-repair processes are excision repair (Setlow and Carrier, 1964; Boyce and Howard-Flanders, 1964) and post-replication repair (Rupp and Howard-Flanders, 1968; Howard-Flanders *et al.*, 1968).

The excision repair process is dependent on the *uvr* gene products for the initial incision step (Boyce and Howard-Flanders, 1964). Survival data (Monk *et al.*, 1971; Witkin and George, 1973) and sedimentation studies (Kanner and Hanawalt, 1970; Paterson *et al.*, 1971) indicate that DNA polymerase I is involved in the subsequent steps of the excision repair pathway. Cooper and Hanawalt (1972) demonstrated that, during the DNA resynthesis step of excision repair, newly synthesized patches of two distinct sizes were produced. The formation of large patches appeared to be dependent on the *recA recB* genes, while DNA polymerase I appeared to be involved in the production of shorter patches of repair-replicated

DNA. The existence of different branches of excision repair has also been reported by Youngs and Smith (1973) who found that the *polA* and *exrA* gene products act in independent branches of the *uvr* gene-dependent excision repair process, as shown by viability and sedimentation experiments. It has not yet been determined if this *exrA*-dependent branch is different from the previously described (Cooper and Hanawalt, 1972) *recA* and/or *recB*-dependent branch.

Several drugs are known to sensitize bacterial cells to killing by UV radiation. This was shown for acriflavine (Alper, 1963), caffeine (Harm, 1967), chloramphenicol (Forage and Gillies, 1969; Ganesan and Smith, 1972), actinomycin D (Reiter *et al.*, 1966), reductone (Alcantara-Gomes *et al.*, 1970) and quinacrine (Z. Fuks and K. C. Smith, unpublished observations). The use of repair inhibitors in combination with repair-deficient mutants has proven to be a very powerful tool for elucidating steps in repair systems and in evaluating the importance of repair in survival.

2,4-Dinitrophenol (DNP) not only uncouples oxidative phosphorylation, but also complexes with many proteins, and thus interferes with their function (see references in Lakchaura and Jagger, 1972). DNP was found to sensitize *E. coli* K-12 cells to X rays by interfering with the growth-

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medium-dependent repair of DNA single-strand breaks (Van der Schueren *et al.*, 1973). DNP was also shown to sensitize *Micrococcus lysodeikticus* cells to UV irradiation and to lower the survival of UV-irradiated phage when the host cells were treated with DNP (Elder and Beers, 1965). Lakchaura and Jagger (1972), however, found that *E. coli* B cells showed a higher survival when plated on a medium containing DNP. This protective effect seemed to be due to a mechanism of growth inhibition, as is true for photoprotection.

The experiments presented here provide evidence that a 90 min post-irradiation treatment with 10^{-2} M DNP sensitizes UV-irradiated cells by interfering with the *exr* gene-dependent branch of the *uvr* gene-dependent excision repair process.

MATERIALS AND METHODS

Bacterial strains. All strains used were derivatives of *E. coli* K-12. The strains and their characteristics are listed in Table 1.

Media. The cells were grown in supplemented minimal medium (SMM) (Ganesan and Smith, 1968) which contained DTM buffer (Kaplan *et al.*, 1962), glucose, and the necessary supplements required by the different strains.

SMM was solidified when necessary with 1.6% Difco noble agar (SMM-agar).

Survival curves. Overnight cultures were diluted 1:100 in SMM and incubated at 37°C. Cells grown for about three cell doublings to a density of $\sim 10^8$ cells/ml were considered to be in log-phase growth. Cells were collected on a Millipore filter (0.45 μ m) and resuspended in DTM buffer to a density of about 2×10^8 cells/ml. The UV source was a General Electric germicidal lamp (8 W) emitting primarily at 254 nm with an exposure rate of 0.967 W m⁻², as determined with an International Light germicidal photometer (No. IL-254). The exposure rate was reduced with a perforated screen to 0.055 W m⁻² for

the sensitive mutants. Samples of 10 ml were irradiated in an open Petri dish (diameter 9 cm) while shaking on a platform shaker. To prevent photoreactivation, the irradiation and handling of the cells were carried out under General Electric "gold" fluorescent lights.

The cells for the control survival curves were diluted in phosphate buffer (0.067 M, pH 7.0) and plated on SMM-agar. For the drug treatment, the cells were diluted 1:1 into SMM with twice the normal amount of glucose and supplements, and twice the required DNP concentration (the final concentration of DNP used for most experiments was 10^{-2} M). At the end of the incubation, samples were diluted in phosphate buffer and plated on SMM-agar. Colonies were counted after incubation at 37°C for 48–72 h.

DNP was obtained from Fisher Scientific Company. Fresh solutions were prepared at room temperature for each experiment.

Sedimentation studies. An overnight culture of the wild-type strain (AB2497) was diluted 1:100 into SMM containing 100 μ Ci thymine-methyl-³H (New England Nuclear; 17.6 Ci/mmol) per ml. The total thymine concentration was 2 μ g/ml. Cells were grown to log phase, collected on a Millipore filter (0.45 μ m), resuspended in DTM buffer and UV irradiated as for the survival experiments. After irradiation, samples were diluted 1:1 into SMM with twice the normal amount of glucose and supplements, and, when desired, 2×10^{-2} M DNP. The irradiated cells were then incubated at 37°C in SMM with or without 10^{-2} M DNP.

At various times during the incubation, samples were taken to determine the number of DNA single-strand breaks present. The samples were immediately diluted in buffer to $\sim 10^7$ cells/ml of which 0.05 ml was layered onto a 0.1 ml cap of 0.5% Sarkosyl (Geigy NL30) and 0.01 M EDTA in 0.5 N NaOH on top of a 4.8 ml linear alkaline sucrose gradient (5–20% weight to volume in 0.1 N NaOH) (Town *et al.*, 1973). Thirty min after layering the last sample the gradients were centrifuged at 30,000 rev/min at 20°C in a Beckman Spinco ultracentrifuge (SW50.1 rotor) for 105 min. The procedures for fractionating and processing the gradients have been described (Kapp and Smith, 1970).

Table 1. *Escherichia coli* K-12 derivatives used*

Designation	Relevant genotype	Other markers	Reference or source
AB2497	wild-type	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Howard-Flanders and Boyce (1966)
AB2487	<i>recA13</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Howard-Flanders and Boyce (1966)
SR78	<i>recB21</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Kapp and Smith (1970)
SR111	<i>recA13 recB21</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Kapp and Smith (1970)
AB2499	<i>uvrB5</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Howard-Flanders <i>et al.</i> (1966)
AB2498	<i>uvrC34</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Howard-Flanders <i>et al.</i> (1966)
SR159	<i>recB21 uvrB5</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	A. K. Ganesan
SR58	<i>recA56 uvrB5</i>	<i>arg his leu pro thi thr thy ara gal lac mtl xyl tsx str</i>	Ganesan and Smith (1969)
JG138	<i>polA1</i>	<i>thy rha lac str</i>	Monk <i>et al.</i> (1971)
DY95	<i>exrA†</i>	<i>thy rha lac str</i>	D. A. Youngs

*Abbreviations are as used by Taylor (1970). All strains were of the F⁻ mating type.

†The *exr* mutation, originally isolated in *E. coli* strain B, appears to be the same as the *lex* mutation, originally isolated in *E. coli* strain K-12 (for references see Mount *et al.*, 1972).

RESULTS

Survival studies. The increase in the number of colony-forming units of unirradiated *E. coli* K-12 wild-type cells was almost completely inhibited during a 90 min treatment with $3 \times 10^{-3} M$ DNP (Fig. 1). However, treatment with $10^{-2} M$ DNP killed about 20% of the control cells. The survival of UV-irradiated cells was not affected by treatment with DNP at concentrations up to $3 \times 10^{-3} M$, but post-irradiation treatment with $10^{-2} M$ DNP produced a sharp reduction in the survival of UV-irradiated cells (Fig. 1).

Figure 2 shows the kinetics of the sensitization of UV-irradiated cells during incubation in growth medium with $10^{-2} M$ DNP. No change in viability was observed after a treatment of 10 or 20 min, but longer incubation times led to a rapidly decreasing viability which leveled off after about 60 min. Even a 30 min incubation in DNP before UV irradiation did not decrease this latency period (i.e., a 20 min treatment was still required, before any effect on survival appeared) (results not shown).

When UV-irradiated cells were incubated in SMM before adding the DNP, they very rapidly became resistant to the DNP effect (Fig. 3). There was no latency period, and a post-irradiation incubation of 10 min before adding the DNP

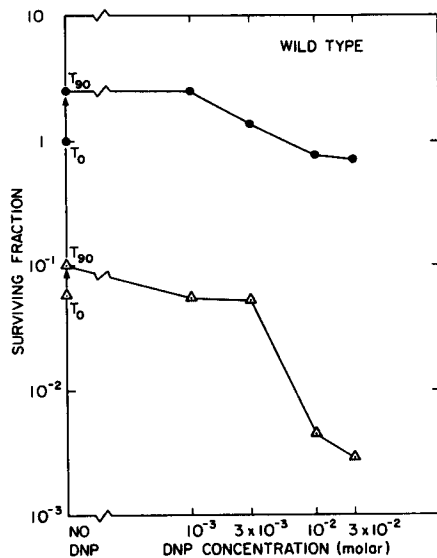


Figure 1. Post-irradiation treatment of *E. coli* K-12 wild-type cells (AB2497) with DNP. Cells in exponential growth were UV-irradiated ($130 J m^{-2}$) and incubated for 90 min in growth medium with different concentrations of DNP. The numbers of unirradiated and irradiated cells at the beginning of the incubation are indicated (T_0). (●) Unirradiated cells treated with DNP; (△) irradiated cells treated with DNP.

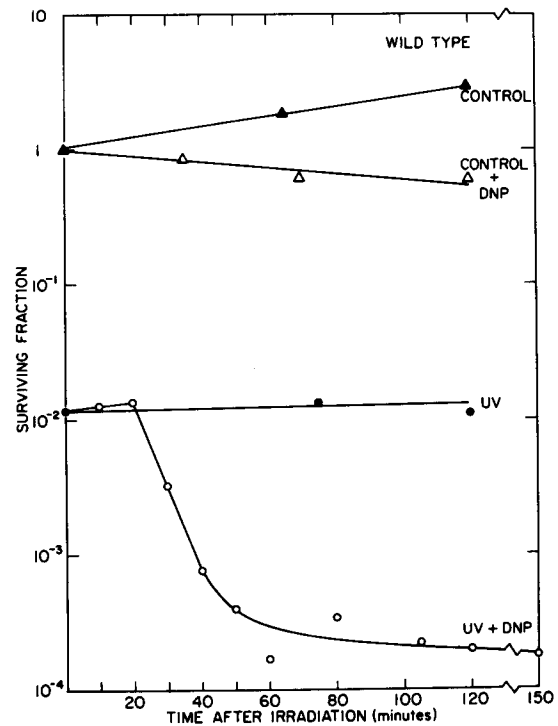


Figure 2. Kinetics of the sensitization of UV-irradiated *E. coli* K-12 wild-type cells (AB2497) by DNP. Exponentially growing cells were UV irradiated ($140 J m^{-2}$) and incubated in growth medium, with or without $10^{-2} M$ DNP. At various times after irradiation, samples were diluted and plated to determine colony forming ability. (▲) Unirradiated cells; (△) unirradiated cells, DNP-treated; (●) irradiated cells; (○) irradiated cells, DNP-treated.

decreased its effect on viability by more than 50 per cent.

The influence of $10^{-2} M$ DNP on the survival of UV-irradiated wild-type cells is shown in Fig. 4. The main effect was a reduction of the shoulder while the final slopes of control and DNP-treated cells were very similar. At a survival level of 10 per cent for untreated cells ($100 J m^{-2}$) DNP reduced the number of viable cells by a factor of 10; at a 1 per cent survival level the sensitization factor was 25.

This sensitizing effect of DNP on UV-irradiated cells was not found in excision deficient cells; the survival of *uvrB* (Fig. 5) and *uvrC* (results not shown) strains was identical whether or not the cells were treated with DNP after UV irradiation.

In *polA1* cells, however, an effect similar to that seen in the wild-type cell was observed (Fig. 6). Here the DNP removed the shoulder of the survival curve, but the curves of the treated and non-

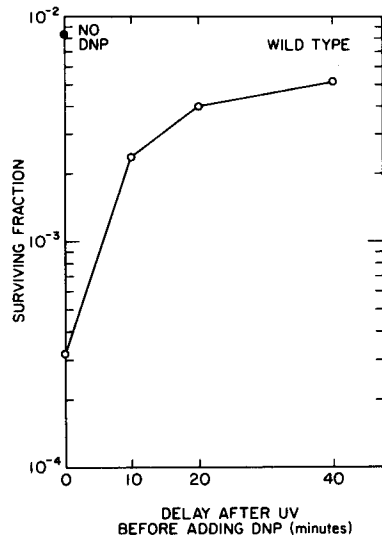


Figure 3. Effect of delay in adding DNP after UV irradiation on the survival of *E. coli* K-12 wild-type cells (AB2497). After UV irradiation (140 J m^{-2}) cells were incubated in growth medium without DNP and at the times indicated 10^{-2} M DNP was added and samples were incubated for an additional 90 min. Survival of cells without incubation or DNP treatment is shown.

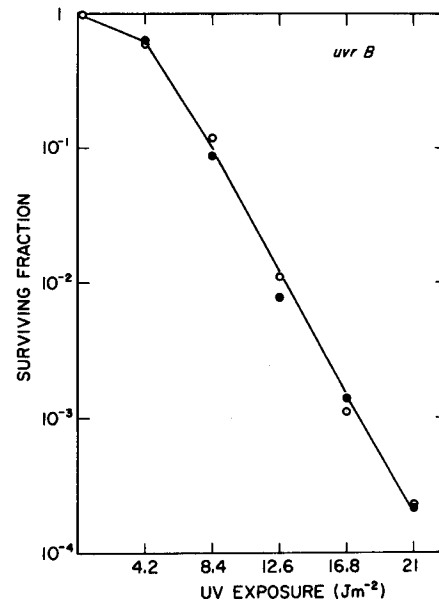


Figure 5. Survival of UV-irradiated *E. coli* K-12 *uvrB* (AB2499) cells with or without DNP (10^{-2} M) treatment. Procedure and symbols as in Fig. 4.

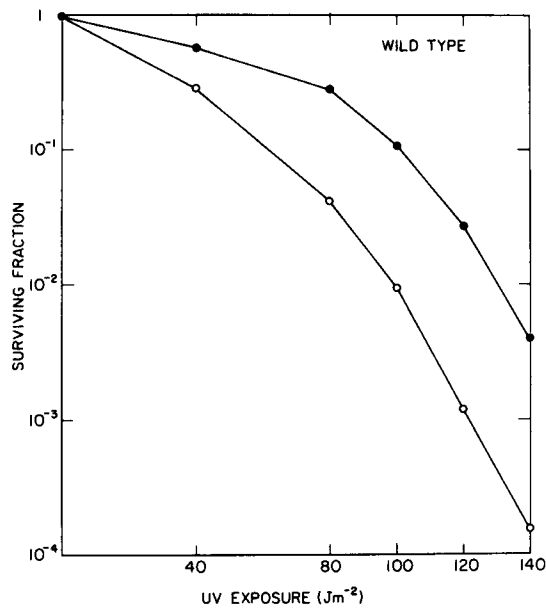


Figure 4. Survival of UV-irradiated *E. coli* K-12 wild-type cells (AB2497) with or without DNP treatment. After UV irradiation one sample was plated immediately while another sample was incubated for 90 min in 10^{-2} M DNP before plating. (●) Control cells; (○) DNP-treated cells.

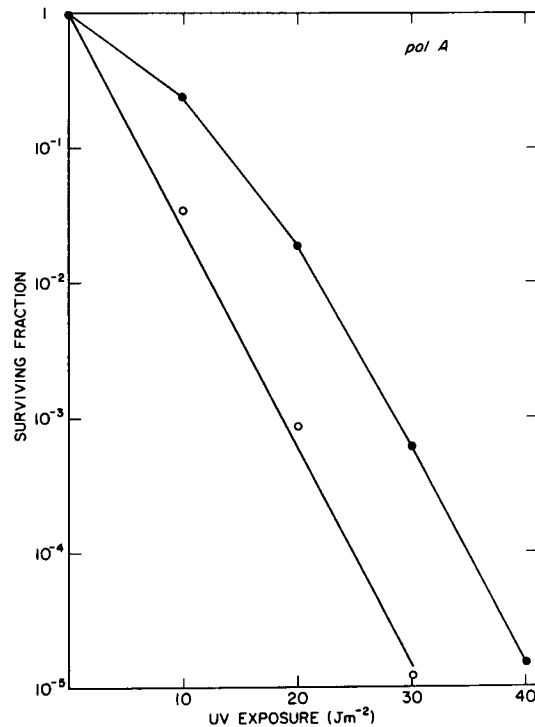


Figure 6. Survival of UV-irradiated *E. coli* K-12 *polA* (JG138) cells with or without DNP (10^{-2} M) treatment. Procedure and symbols as in Fig. 4.

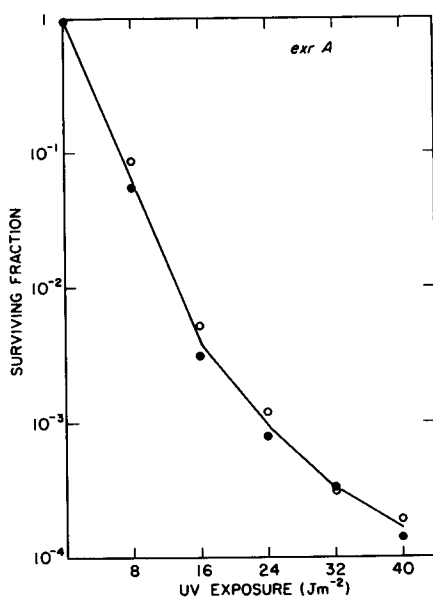


Figure 7. Survival of UV-irradiated *E. coli* K-12 *exrA* (DY95) cells with or without DNP ($10^{-2} M$) treatment. Procedure and symbols as in Fig. 4.

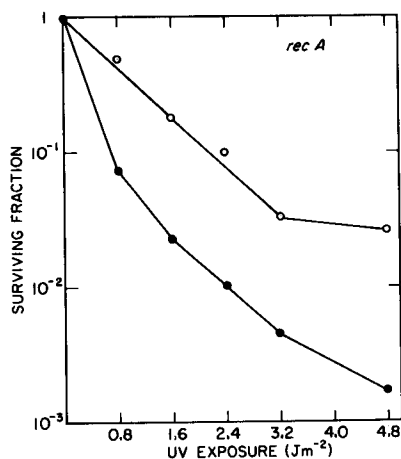


Figure 8. Survival of UV-irradiated *E. coli* K-12 *recA13* (AB2487) cells with or without DNP ($10^{-2} M$) treatment. Procedure and symbols as in Fig. 4.

treated cells were parallel below the 1 per cent survival level and represented about a 30-fold difference in survival.

The survival of the *exrA* strain was not affected by DNP treatment after UV irradiation (Fig. 7), nor was that of a *polA1 exrA* (DY101; Youngs and Smith, 1973) double mutant (data not shown).

A protective effect was found for recombination deficient cells (Figs. 8 and 9). The survival of *recA*

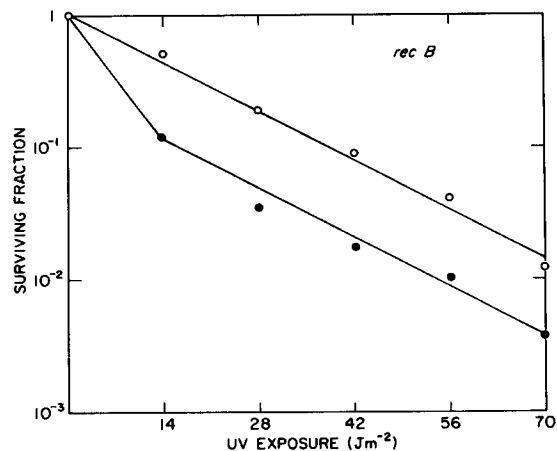


Figure 9. Survival of UV-irradiated *E. coli* K-12 *recB21* (SR78) cells with or without DNP ($10^{-2} M$) treatment. Procedure and symbols as in Fig. 4.

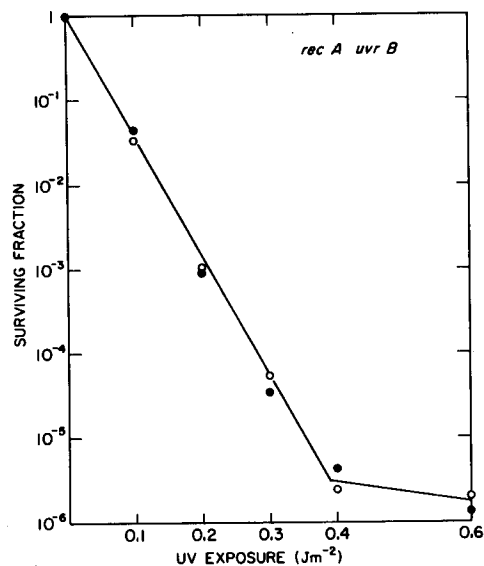


Figure 10. Survival of UV-irradiated *E. coli* K-12 *recA uvrB* (SR58) cells with or without DNP ($10^{-2} M$) treatment. Procedure and symbols as in Fig. 4.

cells was higher by a factor of about 10 when they were treated with DNP after UV irradiation (Fig. 8). The amount of protection increased progressively with DNP treatment time up to 90 min (results not shown). The *recB* strain was also protected by DNP after UV irradiation although the degree of protection was not as large as in *recA* cells (Fig. 9). The *recA recB* double mutant was also protected (results not shown).

The *recA* and *recB* strains, which were normally protected against UV by DNP treatment, did not

show this effect when a *uvr* mutation was also present. Thus, the DNP treatment had no effect on UV survival in the *recA uvrB* strain (Fig. 10) or the *recB uvrB* strain (data not shown).

Sedimentation studies. When wild-type cells (AB2497) were incubated in SMM after a UV

exposure of 60 J m^{-2} the number of incision breaks in the DNA reached a plateau level after about 15 min. The DNA profile started shifting back toward the control position after about 45 min, and complete repair was seen after 90 min (results not shown). Similar data have been reported for *E. coli*

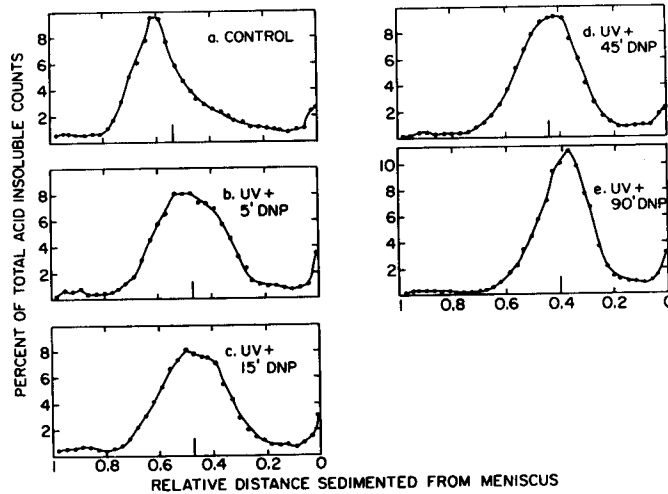


Figure 11. Effect of 10^{-2} M DNP on the production and repair of single-strand breaks in DNA of *E. coli* K-12 wild-type (AB2497) cells after UV irradiation. Cells were prelabeled with ^3H -thymine, washed, resuspended in DTM buffer and UV irradiated (60 J m^{-2}). An unirradiated sample was used as control (a). After irradiation cells were diluted into growth medium (SMM) with 10^{-2} M DNP. Samples were taken after 5 min (b), 15 min (c), 45 min (d), and 90 min (e). Cells were lysed, centrifuged and processed as described in Materials and Methods.

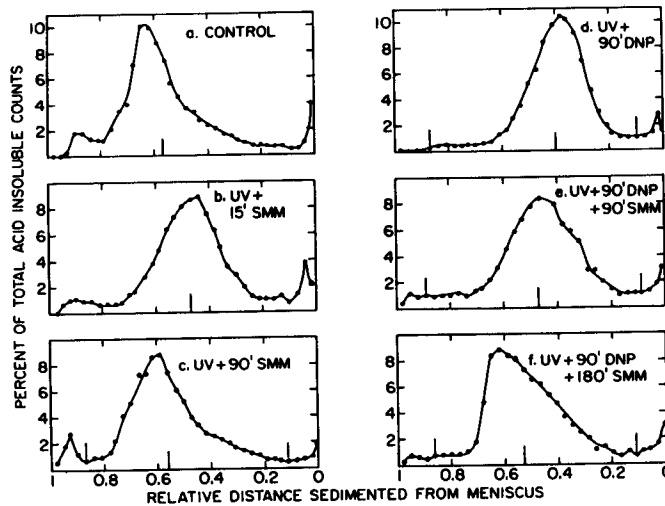


Figure 12. Effect of 10^{-2} M DNP on production and repair of single-strand breaks in DNA of *E. coli* K-12 wild-type (AB2497) cells after UV irradiation (60 J m^{-2}). Cells are prelabeled with ^3H -thymine, washed and resuspended in DTM buffer. (a) Control, (b) 15 min SMM incubation after UV, (c) 90 min SMM incubation after UV, (d) UV irradiation followed by 90 min treatment with 10^{-2} M DNP, (e) UV irradiation followed by 90 min DNP treatment and an additional 90 min incubation in SMM after removal of the DNP, and (f) UV irradiation followed by 90 min DNP treatment and 180 min incubation in SMM after removal of the DNP.

B/r (Setlow, 1967). The DNP-treated cells, however, showed an increase in the number of incision breaks during the entire period of DNP treatment, pointing to an imbalance between the production and repair of breaks (Fig. 11). After washing out the DNP, the cells began to repair the single-strand breaks which had accumulated. However, the rate of repair was much slower than that observed in non-treated cells, and even after 180 min the repair was not complete (Fig. 12).

A 20 min treatment of UV-irradiated cells with DNP, which had no influence on the survival of the cells (Fig. 2), did not interfere with the rate of repair of incision breaks after removal of the DNP (results not shown).

DISCUSSION

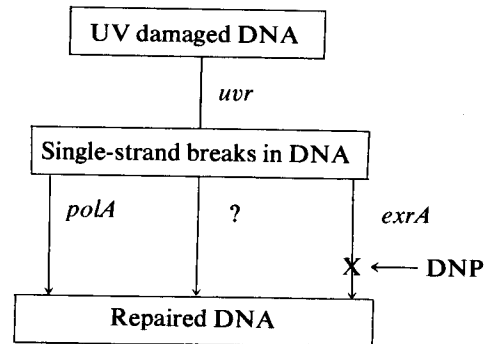
DNP at a concentration of 10^{-2} M sensitized UV-irradiated wild-type cells at the 1 per cent survival level by a factor of about 50. Treatment with 3×10^{-3} M DNP, a concentration which maximally sensitized these cells to X radiation (Van der Schueren *et al.*, 1973), did not affect the survival of UV-irradiated cells (Fig. 1). This post-irradiation sensitizing effect on UV-irradiated cells was essentially complete after 60 min of incubation, but during the first 20 min there was no effect on survival (Fig. 2). If UV-irradiated cells were incubated in growth medium before DNP treatment, they very rapidly became resistant to the sensitizing effects of the DNP treatment (Fig. 3). It thus seems that the DNP must be present immediately after irradiation although the sensitization effect only appears after 20 min of treatment. The main effect of the DNP treatment on the shape of the survival curves is to reduce the shoulder while having little effect on the final slopes (Fig. 4).

Cells carrying a *uvrB* (Fig. 5) or *uvrC* (data not shown) mutation were not sensitized by DNP treatment after UV irradiation, indicating that DNP interferes with a step in the excision repair pathway. This is consistent with earlier data (Alcantara-Gomes *et al.*, 1970) which showed that redutone, a keto-aldehyde which uncouples oxidative phosphorylation, sensitizes bacterial cells to UV irradiation only when they are Hcr^+ .

Incision breaks can be repaired by more than one mechanism. DNA polymerase I is involved in one of these pathways (Kanner and Hanawalt, 1970; Paterson *et al.*, 1971; Cooper and Hanawalt, 1972; Youngs and Smith, 1973). In addition, both an *exrA* strain (Youngs and Smith, 1973) and a *recA recB* strain (Cooper and Hanawalt, 1972) have

been shown to be deficient in a branch of the *uvr* gene-dependent excision repair process which is at least partially independent of the action of DNA polymerase I. It is not yet clear whether the *recA recB*-dependent and the *exrA*-dependent branches are different.

The *polA* strain was sensitized by DNP treatment to the same extent as the wild-type strain (Fig. 6). The *exrA* strain, however, was not affected by DNP treatment (Fig. 7). These data, and those cited above, imply that DNP does not interfere with the DNA polymerase-I-dependent branch of the *uvr* gene-dependent excision repair process, but it does inhibit the *exr* gene-dependent branch. Our results are summarized in the following diagram:



With recombination-deficient strains the results were totally different. The *recA* (Fig. 8), *recB* (Fig. 9) and *recA recB* (data not shown) strains showed an appreciable enhancement in survival when incubated in DNP after UV irradiation. The amount of protection was not the same for the different strains and seemed to increase with the UV sensitivity of the strain. The protective effect of DNP on UV-irradiated *rec* cells was apparently due to an effect on an excision repair system since no protection was found in strains *recA uvrB* (Fig. 10) or *recB uvrB* (data not shown).

It has been shown that holding *recA* cells in buffer before plating them on growth medium enhances their survival (Ganesan and Smith, 1968). This phenomenon, called liquid-holding recovery, was shown to be dependent in *E. coli* K-12 on the presence of functional *uvr* genes (Ganesan and Smith, 1969). It seems that the DNP treatment could have a protective effect analogous to this process. Since the protective effect of DNP observed in a *recA* strain was dependent on the presence of functional *uvr* genes, it is possible that it is dependent on the excision repair function of

DNA polymerase I. This, however, leaves unexplained the fact that *recB* cells are also protected by DNP treatment after UV irradiation, although they show little liquid-holding recovery (Ganesan and Smith, 1968).

Certain cells become more resistant to the lethal action of 254 nm radiation if they are pre-illuminated with 300–380 nm radiation. This phenomenon, termed photoprotection, appears to be due to the temporary inhibition of normal growth which extends the period in which repair systems may operate. Lakchaura (1972) has shown that only *uvr⁺ rec⁻* mutants of *E. coli* K-12 exhibit this phenomenon. Thus, the same genetic requirements appear to hold for photoprotection and DNP-induced protection.

Since the survival data suggest that DNP interferes with an *exr* gene-dependent branch of the *uvr* gene-dependent excision repair process, the repair of UV-induced single-strand breaks (incision breaks) in the DNA was examined by sedimentation techniques. DNP treatment of UV-irradiated cells resulted in the accumulation of a greater number of breaks than normal (Fig. 11), suggesting that there was an imbalance between the production and the repair of DNA single-strand breaks. The repair of incision breaks was inhibited while DNP was present (Fig. 11). Although this inhibition proved to be partially reversible (Fig. 12), the rate of repair after removing the DNP was much slower than the rate seen in untreated cells.

The survival and sedimentation data thus suggest that the sensitizing effect of DNP on UV-irradiated cells is due to an impairment of the excision repair process. More specifically, the survival data suggest that DNP acts on an *exr* gene-dependent branch of the *uvr* gene-dependent excision repair process.

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