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A mechanism for rich-medium inhibition of the repair of daughter-strand gaps in the deoxyribonucleic acid of UV-irradiated *Escherichia coli* K12 *uvrA*

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Summary

Ultraviolet-irradiated *Escherichia coli* K12 *uvrA(B,C)* cells show higher survival if plated on minimal growth medium (MM) rather than on rich growth medium (RM). This phenomenon has been referred to as 'minimal medium recovery' (MMR). UV-irradiated (4 J/m^2) *uvrA* cells showed a similar rate of protein synthesis, whether incubated in MM or RM, however, they showed a severe depression in DNA synthesis when incubated in MM that lasted for about 30 min, and the normal rate of DNA synthesis was not reestablished until about 60 min after irradiation. When a sample of these same cells was switched to RM immediately after UV-irradiation, there was only a slight slowing of DNA synthesis, and the normal rate of synthesis was reestablished by 60 min. An additional *mmrA* mutation or growth retardation by valine blocked both this extra DNA synthesis in RM, and the inhibitory effect of RM on survival. These findings suggest that the absence of a marked delay in DNA synthesis observed in RM may be responsible for the inhibitory effect of RM on the survival of UV-irradiated excision-deficient cells. Two hypotheses, which are not mutually exclusive, are proposed and supported by data to explain why a fast rate of DNA synthesis after UV-irradiation partially inhibits postreplication repair and enhances cell lethality.

The survival of ultraviolet-irradiated, minimal medium-grown *uvrA*, *uvrB* and *uvrC* cells of *Escherichia coli* K12 is higher when the cells are plated on minimal growth medium (MM) rather than on rich growth medium (RM) [e.g., yeast extract–nutrient broth (Ganesan and Smith, 1968; Barfknecht and Smith, 1977; Sharma et al., 1982), or MM plus Casamino Acids or plus 13 pure amino acids (Sharma et al., 1982)]. This higher survival on MM is called 'minimal-medium recovery' (MMR) (Smith, 1971). The reduced survival on RM is due in part to the partial inhibition by RM of the repair of DNA daughter-strand gaps formed after UV-irradiation (Sharma et al., 1982). The DNA repair associated with MMR is largely inducible in nature, and requires functional *recA* and *lexA* genes (Sharma and Smith, 1983). Recently we have isolated a mutation, *mmrA1*, which blocks MMR in a *uvrA* strain, and does so by increasing the survival of UV-irradiated cells plated on RM (Sharma et al., 1983). This increase in UV radiation survival in RM of the *uvrA mmrA* strain has been correlated with an increase in the repair of DNA daughter-strand gaps in RM (Sharma et al., 1983).

The molecular bases for (i) the accumulation of more unrepaired DNA daughter-strand gaps in *uvrA* cells in RM, and (ii) how a mutation in the

mmrA gene prevents these responses to RM are not known. However, the data reported here suggest that the shorter lag in DNA synthesis observed in UV-irradiated cells incubated in RM versus MM may be responsible for the enhanced cell killing in RM. The *mmrA1* mutation, or growth retardation due to the presence of valine without isoleucine in the RM, blocked the earlier onset of DNA synthesis after UV-irradiation in RM-incubated cells relative to MM-incubated cells, and also blocked the lethal effects of RM. Two hypotheses, which are not mutually exclusive, are proposed to explain the effect of RM on survival. One hypothesis is that a slow rate of DNA synthesis favors the recombinational repair of DNA daughter-strand gaps, possibly because of the longer juxtaposition of the two daughter DNA duplexes. The relative efficiencies of postreplication repair in RM and MM support this hypothesis. The second hypothesis is that a continued high rate of DNA synthesis in RM may selectively utilize one or more key proteins in DNA synthesis that are in short supply, and are also required for postreplication repair. Data for a *uvrB ssb-1* strain support this hypothesis.

Materials and methods

Bacterial strains. The strains of *E. coli* K12 used in this study are listed in Table 1.

Media and growth conditions. The minimal growth medium (MM), salts buffer (DTM), and minimal plating medium have been described (Sharma and Smith, 1983). The rich growth media (RM) were: YENB plates were Difco yeast extract (0.75%) and Difco nutrient agar (2.3%); YENB liquid was yeast extract (0.75%) and nutrient broth (0.8%); CAA plates were Difco Casamino Acids (CAA, vitamin assay grade) added to the MM plating medium at 2 mg/ml; 13AA medium was the following amino acids (each at 1 mM) aspartic acid, arginine, glutamic acid, glycine, histidine, leucine, lysine, methionine, phenylalanine, threonine, tyrosine, valine and isoleucine added to either liquid or plating MM. In our earlier study (Sharma et al., 1982), we used the mixture of 13 pure amino acids at the concentrations found in CAA. However, both mixtures of 13 AA yielded essentially the same UV-radiation survival for *uvrA* cells (strain SR1097) (data not shown). 12AA medium was the same as 13AA but without isoleucine.

Logarithmic-phase cultures were obtained and

TABLE 1

LIST OF STRAINS OF *E. coli* K12 [a]

Strain	Relevant genotype	Other characteristics	Source, reference
SR250	*uvrB5*	*thyA36 deo(C2?) leuB19 metE70 lacZ53 rha-5 rpsL151*	Youngs and Smith (1976)
SR281	*uvrB5*	*thyA36 deo(C2?) leuB19 lacZ53 rha-5 rpsL151*	Youngs and Smith (1976)
SR305	*Δ(uvrB-chlA) recF143*	*thyA36 deo(C2?) leuB19 lacZ53 rha-5 malB45 rpsL151*	D.A. Youngs (DY243)
SR1007	*Δ(uvrB-chlA) ssb-1*	*thyA36 deo(C2?) leuB19 lacZ53 rha-5 rpsL151*	Wang and Smith (1982)
SR1040	*uvrB5*	Same as SR281	T.V. Wang
SR1041	*uvrB5 recF143*	Same as SR281	T.V. Wang
SR1097	*uvrA6*	*thyA36 deo(C2?) leuB19 lacZ53 rha-5 bioA2 rpsL151*	Sharma and Smith (1983)
SR1098	*uvrA6 mmrA1*	Same as SR1097	Sharma and Smith (1983)
SR1203	*uvrB5 recB21 recF143*	*thyA deo(C2?) leuB19 lacZ53 rha-5 rpsL151*	T.V. Wang

[a] Genotype symbols are those used by Bachmann (1983). All strains are F^- and λ^-.

prepared for irradiation as previously described (Sharma and Smith, 1983).

UV-irradiation. The source and method for UV-irradiation (254 nm) have been described (Sharma and Smith, 1983). For viability measurement, irradiated and nonirradiated cell suspensions were diluted in phosphate buffer (PB) (Wang and Smith, 1981), and plated on MM and RM plates. Incubation was 1–3 days, except for 12AA plates, which were incubated for 4–5 days before survivors were scored.

Measurement of protein synthesis. Since both the 13AA and MM contain leucine at 1 mM, in our protein synthesis studies, leucine was withdrawn from the 13AA mixture in order to avoid further dilution of the [^{3}H]leucine present. Fresh overnight cultures were diluted into MM containing leucine at 1 mM and [^{3}H]leucine at 4 μCi/ml (L-[4,5-^{3}H(N)]leucine, 59.8 Ci/mmole, New England Nuclear Corp.), and shaken at 37°C until an OD_{650} (Zeiss PMQ II spectrophotometer) of ca. 0.4 was reached. At this time, the cells were UV irradiated and immediately diluted 8-fold into MM or MM plus 13AA (minus leucine), each containing [^{3}H]leucine as above, and again shaken at 37°C. Both before and after UV-irradiation, 0.2-ml cell samples were periodically removed and precipitated with cold 10% trichloroacetic acid (TCA). The precipitates were collected on membrane filters (Millipore EHWP, 0.5-μm pore size) presoaked in 0.1 M leucine, and the radioactivity was determined by liquid scintillation counting [Omnifluor (New England Nuclear Corp.) at 4 g/l in toluene].

Measurement of DNA synthesis. DNA synthesis was measured by the incorporation of [^{3}H]thymine into DNA (Smith and O'Leary, 1968; Smith, 1969). DNA was labeled by growing the cells for at least 4 generations in MM containing thymine at 2 μg/ml and [*Me*-^{3}H]thymine at 2 μCi/ml (50 Ci/mmole, Amersham Corp.). After UV-irradiation in their growth medium, the cells were diluted 8-fold into 3 different growth media (i.e., MM, MM + 13AA, MM + 12AA) each containing the same specific activity of [^{3}H]thymine, and incubation was continued at 37°C. Both before and after irradiation, 0.2-ml samples were periodically removed and treated as described above for protein synthesis (except that the filters were presoaked in thymine at 1 mg/ml).

Measurement of DNA degradation. DNA degradation was measured by the loss of TCA-precipitable radioactivity from prelabeled DNA. DNA was prelabeled in MM as described above for DNA synthesis. At an OD_{650} of ca. 0.4, cells were filter-harvested, washed with DTM and resuspended in homologous nonradioactive MM at an OD_{650} of 0.2. These cells were shaken at 37°C until an OD_{650} of ca. 0.4 was reached (i.e., the cells were 'chased' for one doubling time). The cells were then UV-irradiated while in medium, diluted 8-fold into both nonradioactive MM and RM (MM + 13 AA), and incubation was continued at 37°C. Samples were removed and treated as described above for DNA synthesis.

Results

Fig. 1 shows the effect of plating media on the survival of UV-irradiated *uvrA* and *uvrA mmrA* strains. Note that the survival of the *uvrA mmrA* strain is the same on both MM and RM (i.e., YENB, CAA or 13AA) plates (Fig. 1B), and equals the survival of the *uvrA* strain on MM plates (Fig. 1A).

Since MMR is largely an inducible repair phenomenon (Sharma and Smith, 1983), we thought that RM might affect postirradiation protein synthesis in a *uvrA* strain, and the *mmrA* mutation might block this effect of RM. To observe the relationship between MMR and postirradiation protein synthesis in MM and RM, we measured radioactive leucine incorporation into proteins after 4 J/m^2 of UV-radiation. The *uvrA* and *uvrA mmrA* cells, incubated either in MM or RM (i.e., MM + 13AA), showed similar rates of protein synthesis during the 100 min of incubation after UV-irradiation (data not shown). From this experiment, we conclude that RM does not inhibit MMR by affecting the rate of total protein synthesis after UV-irradiation.

Our next hypothesis was that the rate of DNA synthesis might be enhanced in RM, and this might interfere with postreplication repair, and thus cause cell lethality. To test this, we measured the rate of postirradiation DNA synthesis in MM and RM (i.e., MM plus 13AA). For unirradiated cells, a shift to RM did not affect the rate of DNA

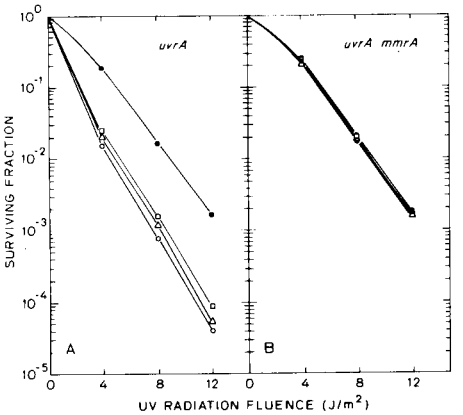

Fig. 1. UV-radiation survival curves of *E. coli* K12 cells that were grown to logarithmic phase in MM, irradiated in PB, and plated on MM (●), YENB (○), MM plus CAA (△), and MM plus 13AA (□). (A) *uvrA6* (SR1097), (B) *uvrA6 mmrA1* (SR1098). Each data point represents the average of at least 2 Expts.

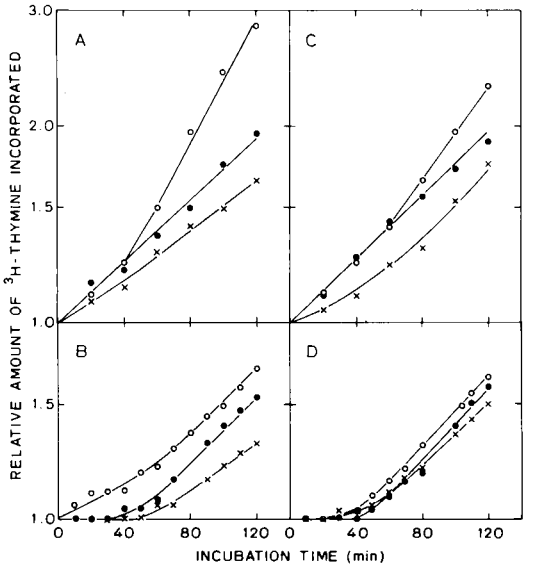

Fig. 2. Effect of culture media on DNA synthesis kinetics in nonirradiated and UV-irradiated cells. Cells were grown in MM containing 2 μCi/ml of [^{3}H]thymine before irradiation (for at least 4 generations) as well as during and after irradiation. At an OD_{650} of ca. 0.4, the culture was UV-irradiated (254 nm) with 4 J/m^2, and immediately diluted 8-fold into radioactive media with the same specific activity; MM (●), MM plus 13AA (○) or MM plus 12AA (×). Unirradiated controls were treated similarly. (A) *uvrA6* (SR1097), unirradiated; (B) *uvrA6* (SR1097), irradiated; (C) *uvrA6 mmrA1* (SR1098) unirradiated; (D) *uvrA6 mmrA1* (SR1098), irradiated. The data are from a representative experiment for each strain.

synthesis for the first 40 min for *uvrA* cells (Fig. 2A), and for the first 60 min for *uvrA mmrA* cells (Fig. 2C). UV-irradiated (4 J/m^2) *uvrA* cells, incubated in MM, showed a severe depression in DNA synthesis, and then, after about 60 min of incubation, synthesized their DNA at a rate similar to that of unirradiated cells; however, cells that were incubated in RM showed only a slight slowing in the rate of DNA synthesis, and were back to the rate observed for unirradiated cells in MM by about 60 min (Fig. 2B). Similar results were obtained for *uvrB* cells (strain SR281) (data not shown). The *uvrA mmrA* cells did not show this effect of RM on DNA synthesis (Fig. 2D). Under these same experimental conditions, UV-irradiated *uvrA* cells showed a 2% DNA degradation in MM and a 7% DNA degradation in RM (Table 2). This suggests that the data in Fig. 2 reflect the kinetics of new DNA synthesis, rather than degradation and resynthesis.

We wished to determine if the continued DNA synthesis observed in RM (versus MM) after UV-irradiation meant that DNA lesions were not blocking the replication fork. If this were so, then one should be able to detect larger sizes of pulse-labeled DNA. Log-phase cells grown in MM were UV-irradiated while in MM. Immediately after UV-irradiation, or after a 10-min postirradiation

TABLE 2

DNA DEGRADATION IN UV-IRRADIATED *uvrA6* CELLS OF *E. coli* K12 [a]

Postirradiation incubation (min)	Relative amount of [^{3}H]thymine in TCA-precipitable fraction of DNA	
	MM	RM
0	1.00	1.00
10	1.00	0.98
20	0.99	0.95
40	0.99	0.95
60	0.98	0.95
80	0.99	0.93
100	0.99	0.91
120	0.98	0.93

[a] UV-radiation fluence was 4 J/m^2. Each data point represents the average of 2 Expts. The RM was MM + 13AA.

incubation in MM, the cells were transferred to MM and to RM, and immediately pulse-labeled with [^{3}H]thymidine for 10 min. In all cases the molecular weight of the pulse-labeled DNA was the same whether the pulse was immediate or delayed by 10 min, and whether it was synthesized in MM or RM (data not shown). This suggests that there was no measurable translesion DNA synthesis in RM, and that the extra DNA synthesized in RM represented the production of more DNA daughter-strand gaps than produced in MM. However, if translesion DNA synthesis occurred at only a few lesions, we may not have detected this on our gradients.

Our next hypothesis was that a continued high rate of DNA synthesis in RM might selectively utilize one or more key proteins in DNA synthesis that are in short supply and are also required for postreplication repair. Therefore, agents that reduce the rate of DNA synthesis should protect UV-irradiated excision-deficient cells from killing in RM. To test this hypothesis, we took advantage of the fact that valine is bacteriostatic for *E. coli* K12, and isoleucine counteracts this effect (Felice et al., 1979). It is important to mention that although we did observe a severe inhibition of growth by valine, we were able to grow all of our cells with the same plating efficiency on 12AA plates (i.e., 13AA medium minus isoleucine) as on MM plates. However, it took about 5 days to observe colonies on the 12AA plates. Earlier workers used an incubation period of 2–3 days to conclude incorrectly that there was no growth on MM plates containing valine (Glover, 1962).

The presence of valine in the absence of isoleucine in what may otherwise be considered to be RM (i.e., 12AA medium) did slow down the rate of DNA synthesis (Fig. 2), and it enhanced the survival of UV-irradiated *uvrA* cells (Fig. 3A), but had no effect on the survival of *uvrA mmrA* cells, which are not sensitive to RM (Fig. 3B). The slowing of growth by valine also eliminated the toxicity of RM on UV-irradiated *uvrB* cells (strains SR250 and SR1040), *uvrB recF* cells (strains SR305 and SR1041) and *uvrB recB recF* cells (strain SR1203) (data not shown).

These data suggest that the slowing down of DNA synthesis immediately after UV-irradiation (observed when cells are incubated in MM) helps UV-irradiated excision-deficient cells to do more postreplication repair. In an attempt to explain the molecular basis of this observation, we took ad-

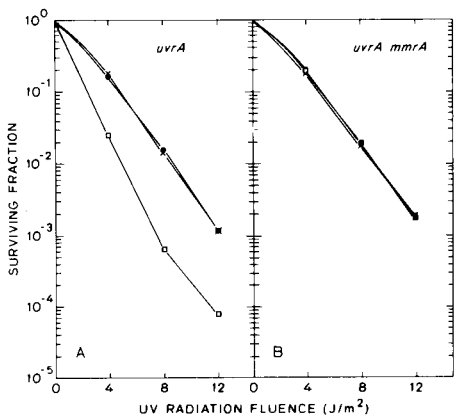

Fig. 3. Effect of growth inhibition by valine (MM plus 12AA) on the UV-radiation survival of cells of *E. coli* K12. Cells were grown in MM, irradiated in PB, and plated on MM (●), MM plus 12AA (contains valine without isoleucine) (×), and MM plus 13AA (contains isoleucine) (□). (A) *uvrA6* (SR1097), (B) *uvrA6 mmrA1* (SR1098). Each data point represents the average of at least 2 Expts.

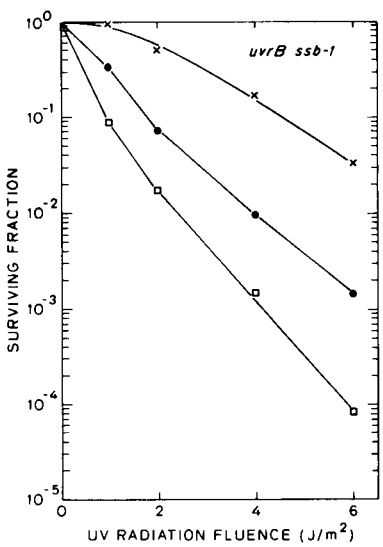

Fig. 4. The effect of various plating media on the survival of UV-irradiated *uvrB ssb-1* (SR1007) cells of *E. coli* K12. Cells were grown in MM, UV irradiated in PB, and plated on MM (●), MM plus 12AA (contains valine without isoleucine) (×), and MM plus 13AA (includes isoleucine) (□). Results are the average of 2 Expts.

vantage of an *E. coli* K12 strain that is partially defective in DNA single-strand binding (SSB) protein (Chase, 1984). The SSB protein is coded for by the *ssb* gene, is essential for DNA replication (Glassberg et al., 1979; Meyer et al., 1979), and is involved in postreplication repair but not in excision repair (Lieberman and Witkin, 1981; Whittier and Chase, 1981; Wang and Smith, 1982). Using a *uvrB ssb-1* strain (SR1007), our hypothesis was that, by slowing down the rate of DNA synthesis, we should reduce the requirement for SSB protein in DNA replication, thereby making more of the limited amount of SSB protein that is present in this strain available for postreplication repair. If this were true, and knowing that the rate of DNA synthesis in the presence of valine inhibition is even slower than in MM (Fig. 2), we expected not only to eliminate the lethality seen in RM, but also to see more repair and higher survival (compared to *uvrB ssb-1* cells plated on MM) in the presence of valine inhibition (i.e., on 12AA plates). Our predictions using the *uvrB ssb-1* strain were confirmed (Fig. 4).

Discussion

Our results suggest that the inhibitory effects of RM (compared to MM) on survival and postreplication repair in UV-irradiated, excision-deficient strains of *E. coli* K12 is due to the lack of a severe depression in DNA synthesis in RM after UV-irradiation. The nearly complete cessation of DNA synthesis for about 30 min for UV-irradiated *uvrA* cells incubated in MM appears to represent the optimum conditions for the successful completion of postreplication repair, since the further slowing of DNA synthesis in the presence of valine did not enhance survival, except under certain conditions (i.e., a partial deficiency in SSB protein) that will be discussed below. Our data showing that the size of the pulse-labeled DNA was the same in MM and RM, both before and after UV-irradiation (without repair incubation), rule out the possibility that translesion DNA synthesis could explain the extra DNA synthesized in RM, and suggest that this extra DNA synthesis in RM just resulted in the formation of more DNA daughter-strand gaps.

To explain how perturbations in the rate of DNA synthesis in RM could inhibit postreplication repair and cause cell killing, we propose the following two hypotheses, which are not mutually exclusive.

Hypothesis 1. If DNA synthesis is slowed after UV-irradiation, as it is when cells are held in MM after UV-irradiation, the DNA daughter-strand gaps can be repaired with high efficiency shortly after they are formed, perhaps because of the favorable juxtaposition of the other daughter DNA duplex. However, if DNA synthesis remains near the rate that is normal for unirradiated cells (as in UV-irradiated cells incubated in RM), the DNA daughter-strand gaps may quickly move further away from the growing point and the favorable juxtaposition of the daughter duplexes may be lost, making gap filling repair processes more difficult to accomplish.

Previously published data (Sharma et al., 1982, 1983) on the repair of DNA daughter-strand gaps in MM and RM showed that the cells incubated in RM accumulated more unrepaired DNA daughter-strand gaps when compared to the cells incubated in MM. These data suggest that the presence of RM during postirradiation incubation decreased the efficiency of the DNA daughter-strand gap filling process and support this hypothesis.

Hypothesis 2. The absence of an immediate delay in postirradiation DNA synthesis in RM may partially inhibit postreplication repair and enhance cell killing in UV-irradiated, excision-deficient strains of *E. coli* K12 by selectively tieing up in DNA synthesis one or more key proteins (e.g., SSB) that may be in short supply and are also required for postreplication repair.

The severe inhibition of growth by valine slowed the rate of DNA synthesis of UV-irradiated cells in 12AA medium compared with cells suspended in MM (Fig. 2), and abolished the toxicity of RM for the Mmr^+ strains. This treatment protected the *uvrB ssb-1* strain from killing in RM, in fact, the survival of UV-irradiated *uvrB ssb-1* cells plated on 12AA medium (i.e., with valine inhibition) was much higher than for cells plated on MM (Fig. 4). This greater survival can be explained if we consider the cellular functions of the SSB protein (Chase, 1984). The SSB protein is essential for DNA replication (Glassberg et al., 1979; Meyer et al., 1979), and is also involved in postreplication repair (Lieberman and Witkin, 1981; Whittier and

Chase, 1981; Wang and Smith, 1982). When growth is slowed in *ssb* cells by valine, the rate of DNA synthesis is slower than in MM, and thus less SSB protein should be required for DNA replication, thereby making more SSB protein available to the cells for postreplication repair. The presumed availability of extra SSB protein at slower DNA synthesis rates seems to be an advantage to the *uvrB ssb-1* strain, since it is reflected in terms of a higher survival after UV-irradiation (Fig. 4).

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