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SENSITIVITY OF DNA REPAIR-DEFICIENT STRAINS OF *ESCHERICHIA COLI* K-12 TO VARIOUS FUROCOUMARINS AND NEAR-ULTRAVIOLET RADIATION

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Abstract—Survival curves were obtained for DNA repair-deficient strains of *Escherichia coli* K-12 (*polA1*, *uvrB5*, and *recA56*) exposed to near-ultraviolet radiation [black light (BL)] in the presence of the DNA cross-linking agent 8-methoxypsoralen (8-MOP) or in the presence of photosensitizers forming primarily monoadducts with DNA [angelicin; 3-carbethoxypsoralen (3-CPs); 5,7-dimethoxycoumarin (DMC)], and after exposure to blue light (BluL) in the presence of 8-MOP or 3-CPs. An interpretation of these data suggests that DNA polymerase I is required for the major pathway of monoadduct repair, but appears to play little or no role in the repair of 8-MOP cross-links. The *uvrB* and *recA* strains were very sensitive, both to the cross-linking agent and to the monoadduct formers. The markedly different results for BL plus DMC or 3-CPs compared to angelicin suggests that the DMC and 3-CPs monoadducts are repaired by a different mechanism than are the angelicin monoadducts, or else DMC and 3-CPs undergo photochemical side reactions that produce DNA lesions other than the expected monoadducts. From photochemical evidence, we predicted that fewer 8-MOP monoadducts should be converted to cross-links by BluL vs BL; this appears to be the case. 3-CPs showed dramatically different biological results when irradiated with BL vs BluL, suggesting that 3-CPs may form more types of photoproducts than the expected monoadducts; BluL, however, appears to favor monoadduct formation.

INTRODUCTION

The photosensitization of DNA to near-ultraviolet (near-UV) radiation (320–400 nm) by bifunctional furocoumarins involves a sequence of events initiated by the formation of an equilibrium complex with DNA that is light-independent, the production of two types of monofunctional adducts to DNA in the first photochemical step and the subsequent photochemical conversion of some of these monoadducts to interstrand DNA cross-links (e.g. the review of Song and Tapley, 1979). Since psoralen is not a symmetrical molecule, it forms two types of monoadducts that differ in chemical structure and absorption spectra. They are the covalent, cycloaddition products linking either the 4′,5′-carbon atoms or the 3,4-carbon atoms of the furocoumarin with the 5,6-carbon atoms of a pyrimidine. The 4′,5′-monoadducts of psoralen absorb below ~380 nm, while the 3,4-monoadducts absorb below ~330 nm (Musajo and Rodighiero, 1972). Therefore, wavelengths between 380 and 400 nm should generate only monoadducts, wavelengths between 330 and 400 nm can generate both types of monoadducts but only convert the 4′,5′-monoadducts to cross-links, while wavelengths below ~330 nm can generate both types of monoadducts and convert both to cross-links. These conclusions are consistent with the results of Chatterjee and Cantor (1978) who found that wavelengths from 380–400 nm lead to only 4′-aminoethyl-4,5′,8-trimethyl psoralen monoadducts with DNA and that subsequent irradiation at 350 nm induced cross-linking of about half of the monoattached furocoumarin. These photochemical reactions are diagramed in Fig. 1. From these considerations, the BL lamps, whose spectral output is shown in Fig. 2, are expected to convert both types of monoadducts

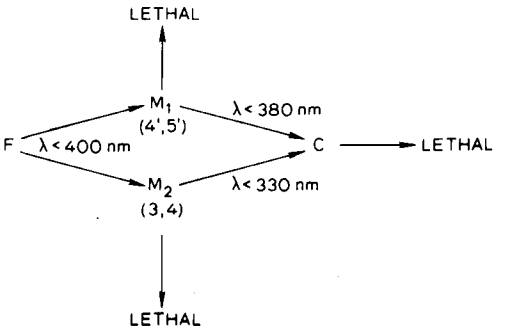

Figure 1. Scheme of the two-step photochemical reactions that lead in the first step to the formation of two types of furocoumarin monoadducts, followed by their photochemical conversion to interstrand DNA cross-links. The symbols designate the furocoumarin (F), complexed with DNA in the dark, the 4′,5′-monoadducts (M_1), the 3,4-monoadducts (M_2) and the cross-links (C).

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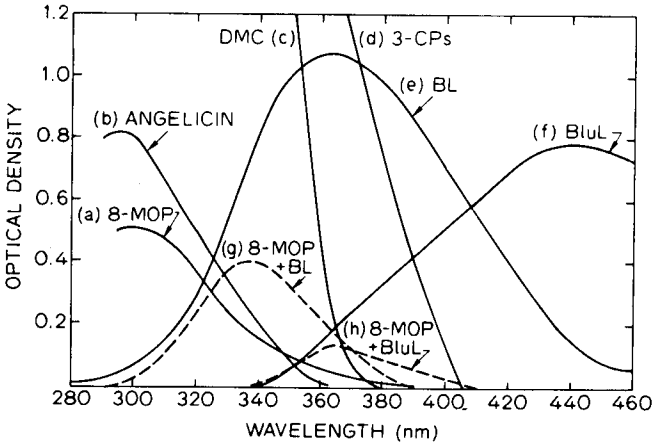

Figure 2. Optical density (1 cm) of photosensitizers: 10 μg/m/ 8-methoxypsoralen (a), 20 μg/m/ angelicin (b), 50 μg/m/ 5,7-dimethoxycoumarin (c), 50 μg/m/ 3-carbethoxypsoralen (d). Spectral distributions of G.E. Type BL lamps (e) and G.E. Type B lamps (f) (arbitrary scales). Dashed lines: effective absorption spectra for 8-methoxypsoralen exposed to black light (g) and blue light (h) lamps (arbitrary scales). These spectra were obtained by numerical integration of the 8-methoxypsoralen (10 μg/m/) absorption over the spectral intensity distribution of the lamps (manufacturer's data); corrected for the transmission of the Kimex dish top, and based on the experimental solution thickness of 0.15 cm.

to cross-links, whereas the BluL lamps (Fig. 2) should cause the formation of both types of monoadducts, and convert only the 4',5'-monoadducts to cross-links.

The photochemical pathway also depends on the structure of the furocoumarin used. 8-Methoxypsoralen (8-MOP) (Fig. 3) forms 4',5'-monoadducts and 3,4-monoadducts, both of which can be converted to DNA cross-links when excited within their respective absorption bands (Song and Tapley, 1979). Angelicin (Fig. 3) and its derivatives only form monoadducts because of the angular shape of the molecule (Bordin *et al.*, 1975, 1978, 1979). 5,7-Dimethoxycoumarin (DMC) can only form 3,4-monoadducts because the furan ring is absent (Fig. 3). Similarly, 3-carbethoxypsoralen (3-CPs) might be expected to form only 4',5'-monoadducts in view of the blocked 3,4-double bond (Fig. 3).

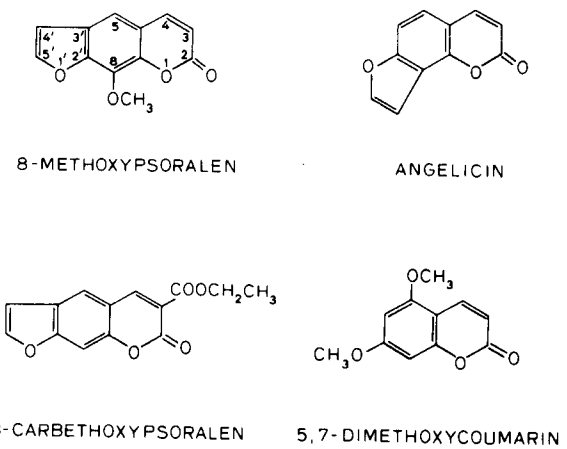

Figure 3. Chemical structures of 8-methoxypsoralen, 3-carbethoxypsoralen, angelicin and 5,7-dimethoxycoumarin.

By comparing the relative survival of several DNA repair deficient strains of *Escherichia coli* K-12 after treatment with the monoadduct and cross-link forming furocoumarins (and using light sources that should alter the extent of cross-link formation), we should gain a better understanding of the relative lethality of DNA monoadducts and cross-links and of the genetic control of the repair of these lesions.

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

Our stock number	Source number	Relevant genotype	Other markers	Source
SR176	MM450	*recA56*	F⁻*rha lacZ rpsL deo*	M. Monk
SR281	DY178	*uvrB5*	F⁻*rha-5 lacZ53 rpsL151 leuB19 thyA36 deo(C2?)*	D. A. Youngs
SR385	JG139	wild type	F⁻*rha-5 lacZ53 rpsL151 thyA36 deo(C2?)*	E. C. Friedberg
SR760*	—	*polA1*	F⁻*rha-5 lacZ53 rpsL151 deo(C2?)*	M. Tang

* Spontaneous Thy⁺ revertant of JG138, which is a cotransductant with SR385.

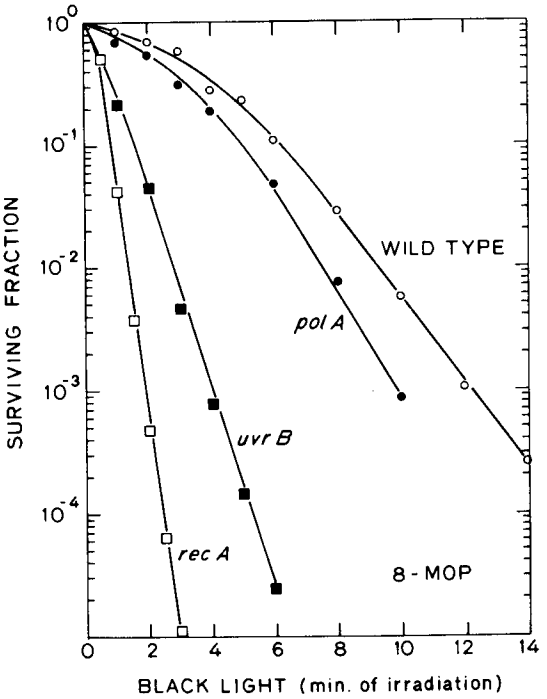

Figure 4. Survival of *E. coli* K-12 strains after exposure to black light (5.7 W/m^2) in the presence of 10 μg/mℓ 8-methoxypsoralen.

MATERIALS AND METHODS

Bacterial strains and culture conditions. The strains of *E. coli* K-12 used are listed in Table 1. A minimal salts glucose medium (Ganesan and Smith, 1968) supplemented with thiamine–HCl (at 0.5 μg/mℓ), thymine (at 10 μg/mℓ) and 1 m*M* L-leucine was used for overnight and log phase cultures. The cells were grown at 37°C for several generations to about 2 × 10^8 cells/mℓ, harvested in log phase on membrane filters (0.45 μm pore size, Millipore Corp.) and resuspended in 0.067 *M* phosphate buffer (pH 7.0) at ~2 × 10^8 cells/mℓ.

Irradiation conditions. The cells were irradiated in phosphate buffer at room temperature in covered Kimax petri dishes on a rotary shaker located 10 cm below two, 15-W fluorescent lamps. General Electric (G.E.) F15T8-BL lamps were used for 'black light' (BL) (280–475 nm; peak ~365 nm) irradiations, and G.E. F15T8-B lamps were used for 'blue light' (BluL) (340–650 nm; peak ~440 nm) irradiations. The incident radiation fluence was measured with a calibrated Eppley Laboratory thermopile. The average fluence rate transmitted by the Kimax dish top was 5.7 W/m^2 for BL, and 5.8 W/m^2 for BluL. The experiments were carried out under G.E. 'gold' fluorescent lamps to minimize photosensitization by the ambient light.

Survival curves. In the photosensitization experiments, nine parts of the cell suspension in buffer were added to one part of the photosensitizer in absolute ethanol. The initial mixing led to about 10–15% lethality, attributed to the presence of the ethanol, after which all strains remained viable in the dark over the time span of the experiments.

After adding the drug, the cells were held in the dark for 30 min prior to irradiation to allow the drug to bind with the DNA. The irradiated cells were diluted with phosphate buffer and plated on supplemented minimal medium solidified with 1.6% Difco Noble agar to prevent the inhibition of the *recA* gene-dependent pathway of excision repair by impurities in less pure agar (Van der Schueren *et al.*, 1974). The plates were incubated 48–72 h at 37°C in the dark. The survival curves shown represent the average of two or more experiments.

Chemicals. 8-Methoxypsoralen was obtained from Sigma Chemical Co.; 3-carbethoxypsoralen was provided by Dr. E. Bisagni of the Fondation Curie, Institut du Radium, Orsay, France; angelicin was provided by Dr. M. J. Ashwood-Smith of the University of Victoria, British Columbia, Canada; 5,7-dimethoxycoumarin was provided by Dr. P.-S. Song of Texas Tech University, Lubbock, TX. All stock solutions of furocoumarins were prepared in absolute ethanol. The chemical structure of the photosensitizers are shown in Fig. 3.

RESULTS

Photosensitization by 8-methoxypsoralen

Several strains of *E. coli* K-12 were irradiated with BL in the presence of 8-MOP (Fig. 4). The *polA* strain was only slightly more sensitive than the wild-type strain (see Table 2), while the *uvrB* and *recA* strains were much more sensitive. In control experiments (i.e. without 8-MOP) the wild-type and *uvrB* strains were unaffected by 60 min of BL irradiation alone. A 50% killing of the *recA* and *polA* strains was achieved by 60 min and 45 min, respectively, of BL irradiation alone, but there was negligible killing with the 3 and 10 min, respectively, of BL irradiation used for these strains (data not shown).

Table 2. Ratios of times of irradiation to produce 50% survival in *E. coli* K-12 (Data taken from Figs. 4–9)

Strain of *E. coli* K-12	BL*				BluL		BluL/BL	
	8-MOP	A	DMC	3-CPs	8-MOP	3-CPs	8-MOP	3-CPs
WT	(3.25)†	(22.0)	(64.5)	(57.0$.	(49.0)	(103.0)	—	—
	1	1	1	1	1	1	1	1
polA	0.71	0.23	0.42	0.54	0.61	0.31	0.86	0.57
uvrB	0.23	0.16	0.29	0.41	0.24	0.23	1.04	0.56
recA	0.17	0.10	0.04	0.04	0.12	0.14	0.71	3.50

* BL, black light; BluL, blue light; 8-MOP, 8-methoxypsoralen; A, angelicin; DMC, 5,7-dimethoxycoumarin; 3-CPs, 3-carbethoxypsoralen; WT, wild type.

† The values in parentheses are the min of irradiation required to produce 50% survival in the wild-type strain. These values were then used to divide the irradiation times required to achieve 50% survival in all the strains under each experimental condition. These ratios are what are listed in this Table.

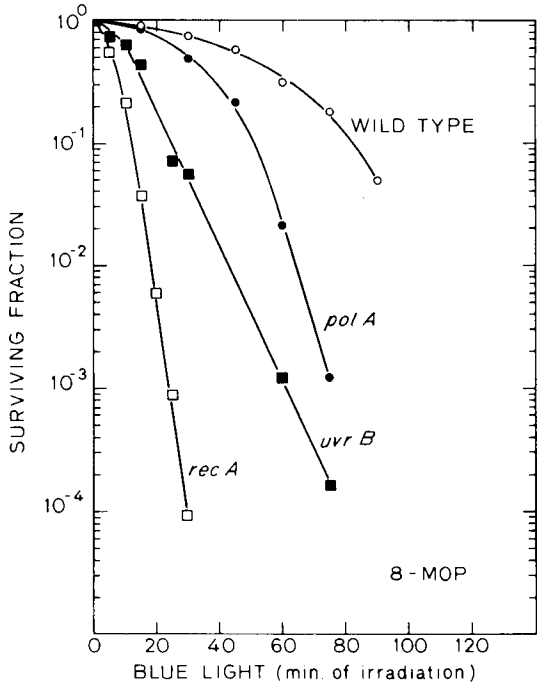

Figure 5. Survival of *E. coli* K-12 strains after exposure to blue light (5.8 W/m^2) in the presence of 10 μg/mℓ 8-methoxypsoralen.

The same strains were also exposed to BluL irradiation in the presence of 8-MOP (Fig. 5). Although the incident fluence of BluL required to achieve the same lethality with 8-MOP (Fig. 5) was much higher than with BL (Fig. 4), the relative sensitivities of the different strains was qualitatively similar in the two cases, however, the *polA* and *recA* strains were relatively more sensitive to 8-MOP in the presence of BluL (Table 2). In control experiments, 120 min of BluL irradiation alone had no effect on the viability of any of the strains (data not shown).

Photosensitization by monoadduct-forming furocoumarins

Angelicin. The wild-type strain was much less sensitive to BL irradiation in the presence of angelicin (Fig. 6) than in the presence of 8-MOP (Fig. 4). However, compared to the wild-type strain, the *polA* strain was relatively more sensitive to BL plus angelicin than to BL plus 8-MOP, or to BluL plus 8-MOP (Table 2). Similar to the results for BL plus 8-MOP, the *uvrB* and *recA* strains were quite sensitive to BL plus angelicin (Table 2). Angelicin does not absorb BluL sufficiently (Fig. 2) to obtain survival data under these conditions.

5,7-Dimethoxycoumarin. The survival curves using DMC and BL are shown in Fig. 7. Compared to the wild-type strain, the *polA* strain was less sensitive to DMC than to angelicin, but was more sensitive to DMC than to 8-MOP plus BL (Table 2). The *uvrB* strain was quite sensitive to BL plus DMC, but the *recA* strain was especially sensitive (Table 2). DMC does not absorb BluL sufficiently (Fig. 2) to obtain survival data under these conditions.

3-Carbethoxypsoralen. The survival curves for the photosensitization of *E. coli* by 3-CPs show two unexpected anomalies. The *polA* and *uvrB* strains were much more resistant to BL (Fig. 8) than to BluL

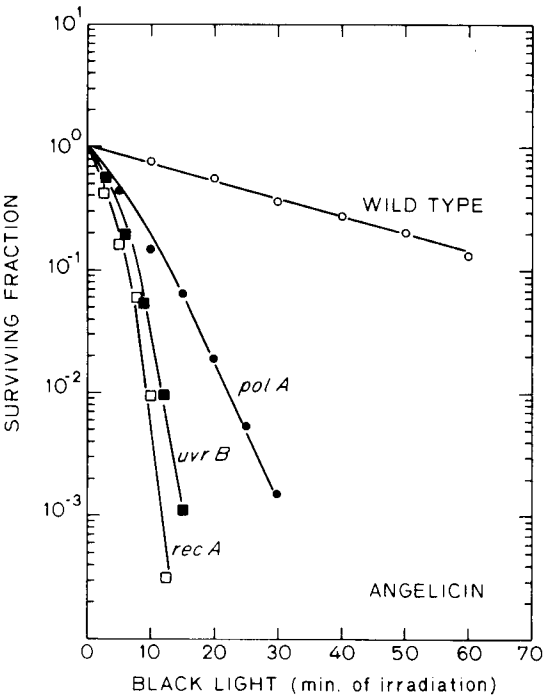

Figure 6. Survival of *E. coli* K-12 strains after exposure to black light (5.7 W/m^2) in the presence of 20 μg/mℓ angelicin.

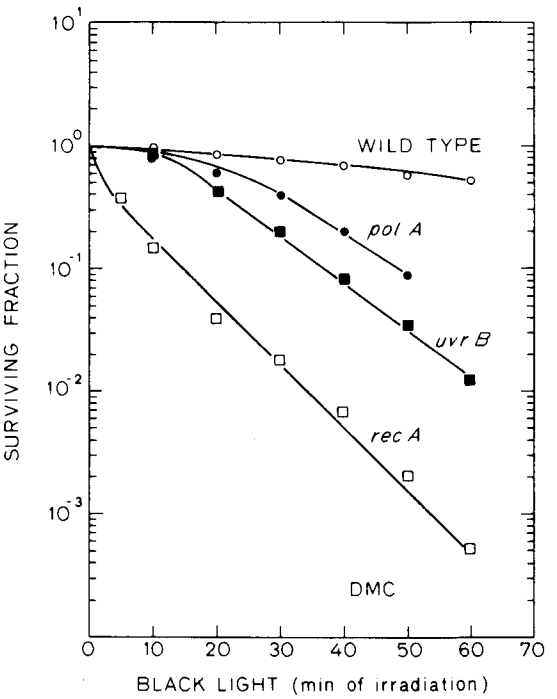

Figure 7. Survival of *E. coli* K-12 strains after exposure to black light (5.7 W/m^2) in the presence of 50 μg/mℓ 5,7-dimethoxycoumarin.

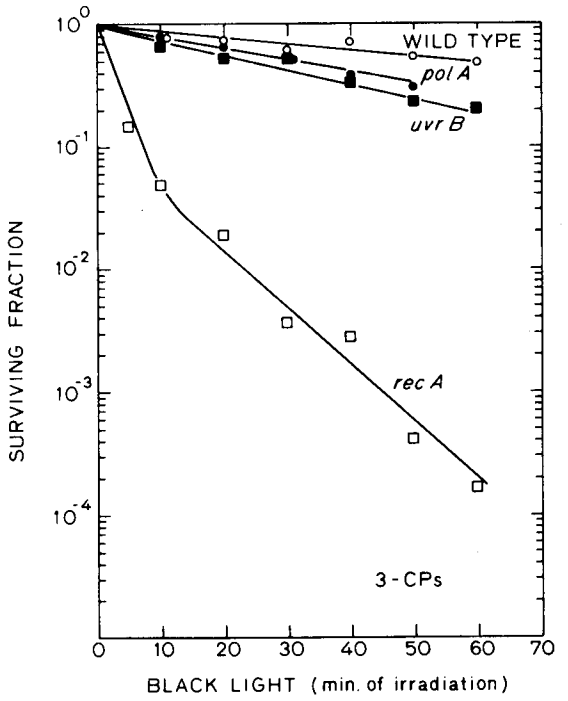

Figure 8. Survival of *E. coli* K-12 strains after exposure to black light (5.7 W/m^2) in the presence of 50 μg/mℓ 3-carbethoxypsoralen.

(Fig. 9), but the reverse was true for the *recA* strain (Table 2). Furthermore, the *uvrB* strain was unusually resistant to BL plus 3-CPs (relative to the wild-type strain) (Fig. 8), compared with the other photosensitizers and irradiation conditions (Table 2). These results are not consistent with the formation of a single type of monoadduct under the two irradiation conditions.

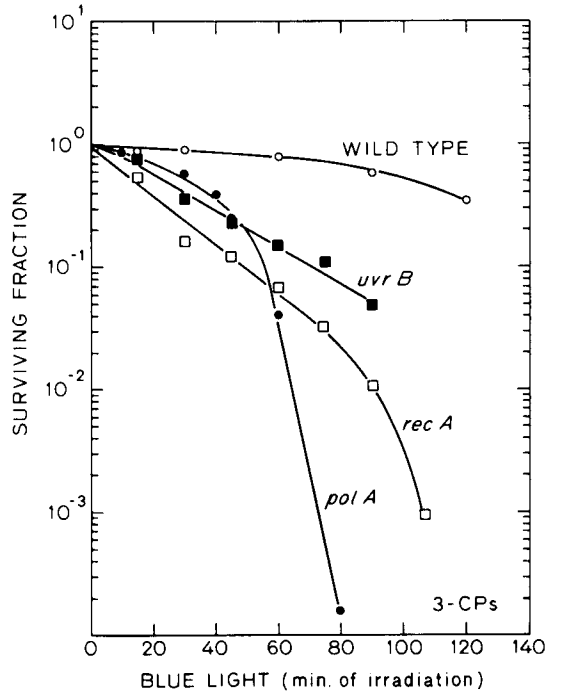

Figure 9. Survival of *E. coli* K-12 strains after exposure to blue light (5.8 W/m^2) in the presence of 50 μg/mℓ 3-carbethoxypsoralen.

DISCUSSION

The lethality of furocoumarin DNA cross-links has been demonstrated in *E. coli* with the sensitizer 4,5',8-trimethylpsoralen (TMP) (Cole, 1971, 1973; Sinden and Cole, 1978), in *Saccharomyces cerevisiae* with 8-MOP (Averbeck *et al.*, 1978), in mammalian cells with 8-MOP (Chandra *et al.*, 1973) and with TMP (Ben-Hur and Elkind, 1973) and may be inferred for many other furocoumarin photosensitized cellular systems (see, e.g. the review of Scott *et al.*, 1976). Studies with photosensitizers that produce few if any cross-links are taken as evidence of monoadduct lethality, e.g. angelicin (Bordin *et al.*, 1975; Ashwood-Smith and Grant, 1976; Ashwood-Smith *et al.*, 1977). 4,5-dimethylangelicin (Bordin *et al.*, 1978, 1979). 3-CPs at wavelengths above 340 nm (Averbeck *et al.*, 1978), 5-hydroxypsoralen and 8-hydroxypsoralen (Song *et al.*, 1975), and DMC (Harter *et al.*, 1976). Based on comparisons of relative lethalities for photosensitization by cross-link formers and monoadduct formers, it has generally been assumed that cross-links are the dominant lethal lesions (Scott *et al.*, 1976; Song and Tapley, 1979). One objective of the present work was to test this assumption. To do this we have investigated the photosensitization of several strains of *E. coli* K-12 (having different DNA repair capacities) by 8-MOP and several monoadduct-forming photosensitizers in the presence of 'black light' and 'blue light' (irradiation conditions that should alter the number of 8-MOP DNA cross-links).

Survival results

The rationale outlined in the Introduction suggests that BL irradiation should convert both types of 8-MOP monoadducts to cross-links. Therefore, a comparison of the results for the relative survival of the *polA* and wild-type strains after irradiation with BL plus 8-MOP (Fig. 4) and with BL plus angelicin (Fig. 6), which forms only monoadducts, leads to the conclusion that the *polA* gene product (i.e. DNA polymerase I) is involved in the repair of DNA monoadducts, but plays little or no role in the repair of 8-MOP cross-links.

Sinden and Cole (1978) reported that a *polA* strain was quite sensitive to TMP plus near-UV radiation relative to the *polA*$^+$ strain. In view of our results with 8-MOP, these data of Sinden and Cole (1978) suggest to us that a considerable number of TMP monoadducts remained unconverted to cross-links under their irradiation conditions, which excluded wavelengths below about 320 nm, and that the reduced rate of repair of DNA strand breaks that these authors observed for a *polA* strain may have been more related to the repair of monoadducts than to cross-links.

In the presence of 8-MOP, the *polA* strain is somewhat more sensitive to irradiation with BluL than with BL (Table 2). This differential sensitivity may result from the accumulation of 3,4-monoadducts of 8-MOP with BluL irradiation, where wavelengths below about 340 nm were not present, as suggested by the scheme in Fig. 1.

The involvement of DNA polymerase I (Pol I) in the repair of DNA monoadducts is also suggested by the sensitivity of the *polA* strain to BL plus DMC (Fig. 7). However, 3-CPs appears to be anomalous; there is a lower sensitivity of *polA* to BL plus 3-CPs (Fig. 8) than to BluL plus 3-CPs (Fig. 9). A possible explanation, consistent with the higher sensitivity of the *polA* strain to angelicin (Table 2), is that 3-CPs forms monoadducts when irradiated with BluL [which is consistent with the report by Averbeck *et al.* (1978) for wavelengths above 340 nm], but cross-links (or some other lesion that does not require Pol I for its repair) are generated by BL (i.e. containing wavelengths shorter than 340 nm). 3-CPs has been reported to be photochemically labile (Vigny *et al.*, 1979).

The *uvrA* gene has been implicated in the repair of TMP cross-links (Cole, 1973) and our results (Fig. 4) suggest that the *uvrB* gene may also play a role in the repair of 8-MOP cross-links. Similarly, the high sensitivity of the *uvrB* strain to BL plus angelicin (Fig. 6) implicates the *uvrB* gene in the repair of monoadducts. However, the somewhat reduced importance of the *uvrB* gene product in the repair of DMC monoadducts and its greatly reduced importance in the repair of the putative 3-CPs monoadducts relative to angelicin (Table 2) suggest that either the DMC, 3-CPs and angelicin monoadducts may not be repaired by identical mechanisms, or that DMC and 3-CPs may cause more complicated photochemistry than the formation of simple monoadducts to DNA.

Cole (1973) demonstrated that *recA* strains of *E. coli* K-12 are deficient in the repair of TMP cross-links. The sensitivity of the *recA* strain to all of the photosensitizers that we have studied (Table 2) indicates that the *recA* gene product plays a key role in the repair of DNA monoadducts as well as cross-links. The fact that the *recA* strain was more sensitive to BL plus DMC or 3-CPs than to any of the other experimental conditions (Table 2) also suggests a greater complexity of the photochemistry or of the repair of the lesions produced by DMC and 3-CPs relative to angelicin.

GENERAL CONCLUSIONS

(1) As discussed above, the *polA* gene product (DNA polymerase I) appears to play a major role in the repair of furocoumarin monoadducts to DNA, but appears to play little or no role in the repair of DNA cross-links.

(2) Compared to the wild-type strain, the *polA* strain was much more resistant to BL plus DMC or 3-CPs than to angelicin, yet all these compounds are supposed to form only monoadducts. This suggests either that the DMC and 3-CPs monoadducts are repaired by a different mechanism than are angelicin monoadducts, or that BL causes photochemical side reactions with DMC and 3-CPs that produces DNA lesions other than the expected monoadducts. Consistent with this conclusion, the *recA* strain was much more sensitive to BL plus DMC or 3-CPs than to 8-MOP or angelicin.

(3) Irradiation with wavelengths longer than 340 nm (i.e. with BluL) did not produce as large an effect on the survival of the *polA* strain (relative to the wild-type strain) as we predicted from the scheme in Fig. 1, but was consistent with this scheme. This may suggest that the 4',5'- and 3,4-monoadducts of 8-MOP are not formed in equal amounts. BluL plus 8-MOP also had a large sensitizing effect on the *recA* strain, but not on the *uvrB* strain (relative to the wild-type strain).

(4) For cells in the presence of 3-CPs, irradiation with BluL yielded markedly different results than when BL was used; the *polA* and *uvrB* strains were much more sensitive to BluL, while the *recA* strain was more resistant. The original studies on 3-CPs were performed at wavelengths longer than 340 nm (Averbeck *et al.*, 1978), which is similar to our BluL irradiation conditions. However, when 3-CPs was employed in the phototherapy of psoriasis (Dubertret *et al.*, 1979), lamps (described in Parrish *et al.*, 1974) were used that were more equivalent to our BL irradiation conditions. Since our results with 3-CPs were dramatically different depending upon which lamp we used, it cautions against carrying laboratory experiments with furocoumarins over to the clinic when the laboratory experiments are performed under different irradiation conditions than those used in the clinic.

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