JOURNAL OF BACTERIOLOGY, Sept. 1987, p. 3988–3993
0021-9193/87/093988-06$02.00/0

Vol. 169, No. 9

UV-Inducible DNA Repair in the Cyanobacteria *Anabaena* spp.

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Received 13 April 1987/Accepted 8 June 1987

Strains of the filamentous cyanobacteria *Anabaena* spp. were capable of very efficient photoreactivation of UV irradiation-induced damage to DNA. Cells were resistant to several hundred joules of UV irradiation per square meter under conditions that allowed photoreactivation, and they also photoreactivated UV-damaged cyanophage efficiently. Reactivation of UV-irradiated cyanophage (Weigle reactivation) also occurred; UV irradiation of host cells greatly enhanced the plaque-forming ability of irradiated phage under nonphotoreactivating conditions. Postirradiation incubation of the host cells under conditions that allowed photoreactivation abolished the ability of the cells to perform Weigle reactivation of cyanophage N-1. Mitomycin C also induced Weigle reactivation of cyanophage N-1, but nalidixic acid did not. The inducible repair system (defined as the ability to perform Weigle reactivation of cyanophages) was relatively slow and inefficient compared with photoreactivation.

The ability to repair damage to DNA appears to be almost universal among living organisms. In most organisms that have been studied in detail, there are multiple pathways that are involved in the repair of DNA lesions; some pathways are specific for certain types of lesions, whereas other pathways are more general and may involve DNA replication and recombination. Virtually all organisms studied have some type of excision repair, and many organisms, including man, can photoreactivate UV light-induced pyrimidine dimers (13, 32). Inducible SOS repair, which is error-prone, has been described in many gram-negative bacteria (31); in a few gram-positive organisms, including *Bacillus subtilis* (40, 41); and in some eucaryotes (8, 13). DNA repair has been studied most extensively in *Escherichia coli*, which has several repair pathways, including photoreactivation, excision repair, recombination-dependent repair, and SOS repair (31, 32). The SOS response begins with activation of the RecA protein to a protease that cleaves the LexA repressor, which normally prevents transcription of the many genes under the control of the SOS pathway (18, 32). The SOS pathway is induced in response to various DNA-damaging agents, including UV light and mitomycin C, and is also induced by inhibition of DNA replication by agents such as nalidixic acid (32). Some of the responses of the cell to induction of the SOS pathway include reactivation of UV-irradiated bacteriophage (Weigle reactivation), mutagenesis of UV-irradiated cellular or phage DNA, repair of double-strand breaks in the DNA, alleviation of restriction, and induction of repair pathways that allow long patch excision repair (19, 31, 32, 37).

DNA repair has been studied in several strains of cyanobacteria. Photoreactivation of UV light-damaged DNA occurs in *Agmenellum quadruplicatum* (30), *Anacystis nidulans* (3, 29). *Gleocapsa alpicola* (*Synechocystis* sp. strain PCC 6308) (21, 22). *Plectonema boryanum* (24, 35), and *Anabaena doliolum* (27). Photoreactivation of UV-irradiated infecting cyanophage has also been demonstrated (4, 26, 39). There is evidence for excision repair in cyanobacteria. Repair of *Synechocystis* sp. strain PCC 6308 occurs under conditions that do not allow photoreactivation (36), and dimer excision occurs under both photoreactivating and nonphotoreactivating conditions (21). Under conditions that prevent photoreactivation dimer excision is accompanied by DNA degradation, and that degradation is inhibited by excision repair inhibitors (22). In several strains of cyanobacteria, sensitivity to UV irradiation increases in the presence of caffeine or acriflavine, both of which are known to prevent excision repair in other bacteria (3, 27, 36). The evidence for UV-inducible DNA repair in cyanobacteria is indirect. The sensitivity of *A. nidulans* to UV irradiation increases when the cells are treated with chloramphenicol prior to irradiation (5). Exposure of *Synechocystis* sp. strain PCC 6308 to a sublethal dose of UV light results in decreased DNA degradation after a subsequent challenge dose, and this decrease is abolished by protein synthesis inhibitors administered between the exposures to UV light (22). If the DNA degradation is attributable to excision repair, then the decrease in degradation implies that another repair pathway is operational and that it requires protein synthesis (22). In *E. coli* decreased DNA degradation is seen under similar conditions, and that decrease in degradation requires the RecA protein (20, 25). Geoghegan and Houghton (11) have recently reported the isolation of a gene that complements the UV-sensitive phenotype of an *E. coli recA* mutant.

Little is known about DNA repair in the heterocyst-forming filamentous cyanobacteria. Cyanophages have been isolated that infect several strains of *Anabaena* spp. (15); therefore, we attempted to quantitate DNA repair in several strains of *Anabaena* spp. by measuring the reactivation of UV-damaged cyanophage. We report here that these strains of *Anabaena* spp. are capable of very efficient photoreactivation and Weigle reactivation of cyanophages. The latter ability provided evidence for a DNA repair pathway that could be induced by either UV light or mitomycin C.

MATERIALS AND METHODS

Strains and growth conditions. *Anabaena* sp. strain PCC 7120 (ATCC 27893), *Anabaena variabilis* ATCC 29413, *Anabaena* sp. strain M-131 (University of Tokyo), and *A. variabilis* sp. strain PCC 7118 (ATCC 27892) were grown either in an eightfold dilution of the medium of Allen and Arnon (2) supplemented with 2.5 mM KNO_3 and 2.5 mM $NaNO_3$ (AA/8 plus nitrate) or on BG-11 medium (28) solidified with 1% Bacto-Agar (Difco Laboratories, Detroit, Mich.) purified by the method of Braun and Wood (6).

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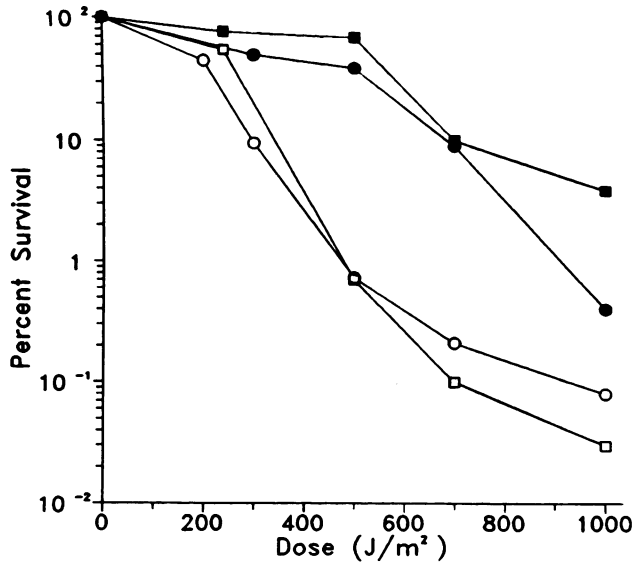

FIG. 1. UV light-induced inactivation and photoreactivation of cyanobacteria. Very short filaments of cyanobacterial cells were exposed to the indicated doses of UV light, diluted, and plated prior to exposure to photoreactivating (white light) or nonphotoreactivating (yellow light) conditions. *Anabaena* sp. strain PCC 7120 was exposed to white (■) or yellow (□) light; *A. variabilis* ATCC 29413 was also exposed to white (●) or yellow (○) light.

Cultures were grown in 50-ml volumes in 125-ml flasks with shaking at 100 rpm at 32°C under white lights at approximately 50 microeinsteins m^{-2} s^{-1}. In experiments in which it was necessary to prevent photoreactivation, all manipulations and incubations were done under yellow lights (Sylvania model F20T12/GO). The generation time of these strains was about 24 h under the conditions described here and was the same for equal fluences of white or yellow light.

Cyanophages N-1 (1), A-1(L) (16), and AN-10 (15) were propagated separately on each cyanobacterial strain by the plate lysate technique by using cyanophage buffer (15) to elute phage.

Survival after UV-irradiation. Two-week-old cultures of cyanobacteria were fragmented by bath cavitation (38) into filaments of two to four cells and then were washed and suspended in AA/8 plus nitrate at a concentration of 4×10^6 to 6×10^6 CFU ml^{-1}. Cells (5 ml) or phage (1 ml) in a 100-mm-diameter sterile plastic petri dish were exposed, with constant stirring, to shortwave UV irradiation (Sylvania germicidal lamp model G30T8) for times ranging from 10 s to 20 min (25 to 3,000 J m^{-2}). Appropriately diluted samples of UV-irradiated cells were spread on BG-11 agar plates. Irradiated phage were diluted such that 100 to 200 PFU were mixed with 5 ml of the appropriate host cells (at 2×10^7 to 3×10^7 cells ml^{-1}) and 5 ml of molten BG-11 agar and poured into sterile petri dishes. Plates were incubated at 32°C under white (photoreactivating) or yellow (nonphotoreactivating) lights. Colonies were counted after 7 to 10 days, and plaques were counted after 3 to 5 days. Data are the average of at least three experiments.

Weigle reactivation. Phage exposed to various doses of UV irradiation as described above were used to infect unirradiated and UV-irradiated host cells at the doses indicated in the figures. Conditions for plating and incubation were as described above, except that all plates were incubated under yellow lights. Weigle reactivation was measured as the ratio of the titer of irradiated phage on irradiated host cells to the titer of the same phage on unirradiated host cells. Data are the average of at least three experiments.

Nalidixic acid or mitomycin C treatments. Nalidixic acid was added to washed cells at a final concentration of 50 μg ml^{-1}; mitomycin C was added at a final concentration of 0.5 or 1.0 μg ml^{-1}. After 6 h at 32°C (under yellow light for nalidixic acid-treated cells, and in darkness for mitomycin C-treated cells), cells were washed three times in AA/8 plus nitrate and resuspended in the same medium. Cells were infected with N-1 that had been exposed to various doses of UV light and were plated and incubated as described for Weigle reactivation. Data are the average of at least three experiments.

RESULTS

Photoreactivation of UV-treated cyanobacteria. All cyanobacterial strains tested were capable of photoreactivation. *A. variabilis* ATCC 29413 and *Anabaena* sp. strain PCC 7120 cells irradiated with UV light at a dose of 500 J m^{-2} produced about 100 times as many colonies under conditions that allowed photoreactivation as they did under conditions that did not (Fig. 1). Unirradiated cells grew equally well under either condition. Similar results were obtained with *A. variabilis* PCC 7118 (data not shown). *Anabaena* sp. strain M-131 was more sensitive to UV light; under nonphotoreactivating conditions a dose of UV light of about 200 J m^{-2} was required to kill 50% of the cells of the aforementioned strains of cyanobacteria, whereas 50% of the *Anabaena* sp. strain M-131 cells were killed by a dose of UV light of less than 100 J m^{-2} (data not shown).

Photoreactivation of cyanophage. Cyanophage N-1 was inactivated by UV light. Survival of UV-treated phage was stimulated 10- to 1,000-fold by incubation of infected *A. variabilis* ATCC 29413 or *Anabaena* sp. strain PCC 7120 cells under conditions that allowed photoreactivation (Fig. 2). Similar inactivation and photoreactivation of N-1 was seen in infected *A. variabilis* PCC 7118 and *Anabaena* sp. strain M-131 cells (data not shown). The results were also very similar for inactivation and photoreactivation of cyanophages AN-10 and A-1(L) in three of the cyanobacterial hosts (*A. variabilis* ATCC 29413 is not a host for these cyanophages [15]) (data not shown).

Weigle reactivation of cyanophage N-1. Weigle reactivation is the enhancement of the plaque-forming ability of irradiated phage by infection of an irradiated host versus that of an unirradiated host (34). This enhancement is attributable to the induction of a DNA repair pathway in the irradiated host that can repair the UV damage present in infecting phage and, hence, increase plaque-forming ability. In these experiments, reactivation was measured as the titer of a UV-irradiated suspension of cyanophage N-1 on irradiated host cells divided by the titer of the same UV-irradiated phage on unirradiated host cells. Reactivation of N-1 was maximal at doses of UV light to the host cells of 360 J m^{-2} and was evident at doses to *Anabaena* sp. strain PCC 7120 as low as 120 J m^{-2} (Fig. 3). Similar levels of reactivation of cyanophage N-1 in *Anabaena* sp. strain M-131 and of cyanophages AN-10 and A-1(L) in *Anabaena* sp. strain PCC 7120 were also observed (data not shown). The apparent greater reactivation of N-1 in *A. variabilis* ATCC 29413 than in *Anabaena* sp. strain PCC 7120 was due to the fact that about a fourfold-greater efficiency of plating of unirradiated N-1 occurred on irradiated *A. variabilis* ATCC 29413 cells than occurred on unirradiated host cells (Table 1). This greater

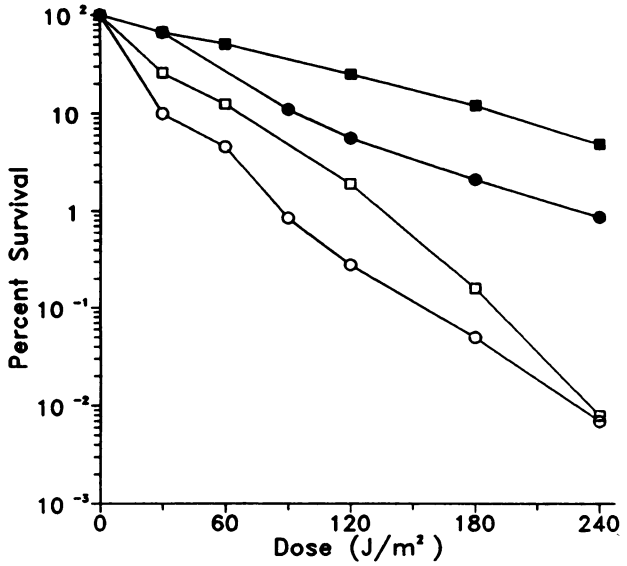

FIG. 2. UV light-induced inactivation and photoreactivation of cyanophage N-1. Cyanophage N-1 was exposed to the indicated doses of UV light, diluted, and plated on the indicated host strains prior to exposure to photoreactivating (white light) or nonphotoreactivating (yellow light) conditions. Infected host strain *Anabaena* sp. strain PCC 7120 was exposed to white (●) or yellow (○) light; infected host strain *A. variabilis* ATCC 29413 was also exposed to white (■) or yellow (□) light.

efficiency did not occur in two other strains of *Anabaena* spp. (Table 1). The mechanism for this enhanced plating efficiency on irradiated host cells is not known. As has been reported previously (9) the efficiency of adsorption of N-1 to *A. variabilis* ATCC 29413 cells compared with that to *Anabaena* sp. strain PCC 7120 cells was very low; however, irradiation of the cells had no effect on adsorption of N-1 to either strain (data not shown). Thus, the greatly enhanced reactivation of irradiated N-1 on irradiated *A. variabilis* ATCC 29413 cells represented the combined effects of Weigle reactivation and increased plating efficiency of N-1 on irradiated versus on unirradiated cells of this strain.

In *E. coli*, the pyrimidine dimers that result from UV irradiation are both the substrate for photoreactivation and the inducer of SOS repair (31). Therefore, repair of the dimers by photoreactivation should prevent Weigle reactivation. Photoreactivation of irradiated *Anabaena* sp. strain PCC 7120 cells for 24 h prior to infection with irradiated N-1 inhibited Weigle reactivation almost completely (Fig. 4), suggesting that pyrimidine dimers may induce Weigle reactivation of N-1.

Effects of mitomycin C and nalidixic acid on reactivation. Mitomycin C and nalidixic acid induce the SOS response in *E. coli* (31). Mitomycin C induced reactivation of N-1 in both *A. variabilis* ATCC 29413 and *Anabaena* sp. strain PCC 7120 to about the same extent as did UV light (Table 2). However, nalidixic acid had virtually no effect on reactivation of N-1 in either strain (Table 2).

Rate of repair of UV damage to cyanobacteria. Weigle reactivation provided a convenient measure of the amount of damage in UV- or mitomycin C-treated cells. *Anabaena* sp. strain PCC 7120 cells, treated with either UV light or mitomycin C, were incubated under either white or yellow light, and at daily intervals after treatment the cells were infected with unirradiated or irradiated N-1. This treatment

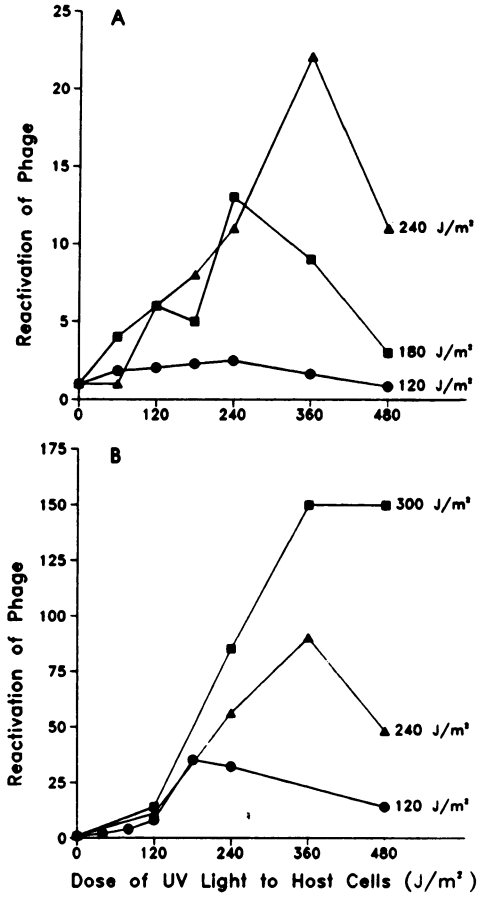

FIG. 3. Weigle reactivation of cyanophage N-1. N-1, treated with UV light at the doses indicated within the graph, was used to infect cells irradiated at the doses indicated on the *x* axis of the graph. Reactivation was measured as the titer of irradiated N-1 on irradiated cells divided by the titer of the same phage on unirradiated cells. (A) Host strain *Anabaena* sp. strain PCC 7120. (B) Host strain *A. variabilis* ATCC 29413.

had little or no effect on unirradiated N-1 at any time (data not shown). Both irradiated and mitomycin C-treated cells showed Weigle reactivation shortly after the treatment, and that reactivation decreased with time (Fig. 5). UV-irradiated cells incubated under white light had little reactivating activity by 24 h posttreatment; however, the same cells, incubated under yellow light, required 72 h to repair the UV-induced lesions that allowed Weigle reactivation. The rate of loss of reactivation of N-1 in mitomycin C-treated

TABLE 1. Efficiency of plating of cyanophage N-1 on strains of *Anabaena* spp.

Strain	Titer[a] of phage on:	
	Unirradiated cells	Irradiated cells[b]
A. variabilis ATCC 29413	3.5×10^6	1.2×10^7
Anabaena sp. strain PCC 7120	3.1×10^8	2.1×10^8
Anabaena sp. strain M-131	4.4×10^8	2.3×10^8

[a] Cells of the indicated strain were infected with diluted samples from a single stock of cyanophage N-1 which had been propagated on *A. variabilis* ATCC 29413.

[b] Cells were irradiated with UV light at a dose of 180 J m^{-2} as described in Materials and Methods.

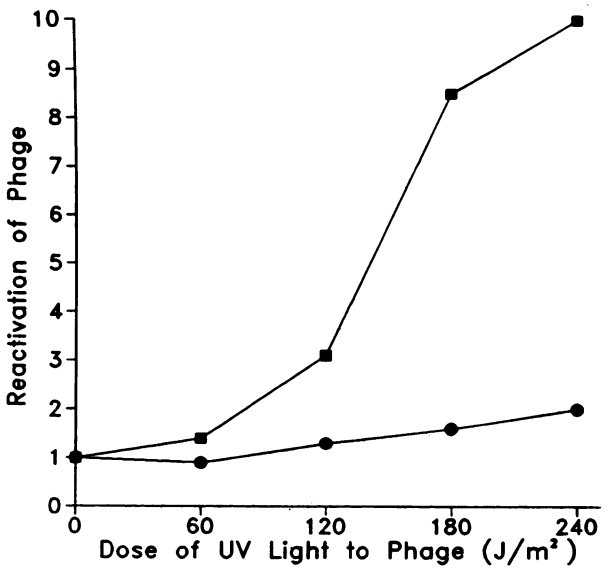

FIG. 4. Reversal of Weigle reactivation of N-1 by photoreactivation of host cells. *Anabaena* sp. strain PCC 7120 cells were irradiated at a dose of 240 J m^{-2} and then either immediately infected with N-1 irradiated at the doses indicated on the graph (■) or allowed to incubate under white light for 24 h prior to infection (●). All infected cells were subsequently incubated under yellow light prior to quantitation of plaques. Reactivation was measured as described in the legend to Fig. 3.

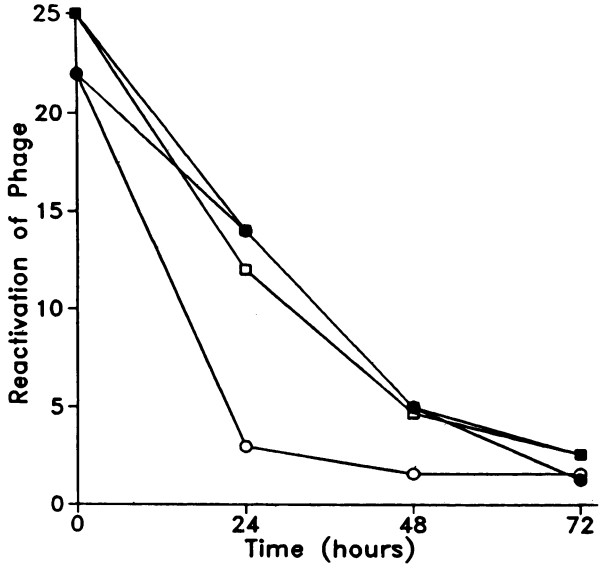

FIG. 5. Rate of repair of UV-induced damage to *Anabaena* sp. strain PCC 7120. *Anabaena* sp. strain PCC 7120 cells were irradiated at a dose of 360 J m^{-2} and were incubated under either white (○) or yellow (●) light for the time indicated prior to infection with N-1 irradiated at a dose of 240 J m^{-2}. In a similar experiment, cells of the same strain were treated with mitomycin C (0.5 μg ml^{-1}) and were incubated under either white (□) or yellow (■) light for the time indicated prior to infection with N-1 irradiated as described above. After infection, all cells were incubated under yellow light prior to quantitation of plaques. Reactivation was measured as described in the legend to Fig. 3.

cells was very similar for cells incubated under either white or yellow light and was essentially the same as for irradiated cells incubated under yellow light (Fig. 5). It appeared that DNA damage induced by UV light was repaired with much greater efficiency by photoreactivation than by the other repair systems (including UV-inducible repair) of this organism. The rate of repair of mitomycin C-induced lesions suggested that they were also repaired slowly by relatively inefficient pathways.

DISCUSSION

All the *Anabaena* spp. strains tested could photoreactivate UV-induced damage efficiently, restoring viability to nearly 100% at doses of UV light up to about 150 J m^{-2}. They were much more sensitive to UV light inactivation under conditions that allowed growth but that prevented photoreactivation (i.e., yellow light); however, in most of the strains tested, survival was greater than 80% at a dose of 80 J m^{-2}, which kills 99.9% of *E. coli* B/r cells (14). These strains were also more resistant to UV damage than is the unicellular cyanobacterium *Synechocystis* sp. strain PCC 6308; only about 1% of the latter cells survive a dose of 120 J m^{-2} (17). In contrast, *A. nidulans* (29) and *A. doliolum* (27) are more resistant to UV light than are the strains used in the present study.

TABLE 2. Effect of mitomycin C, nalidixic acid, and UV light on reactivation of cyanophage N-1

Host	Treatment	Reactivation[a]
A. variabilis ATCC 29413	None	1
	UV light (240 J m^{-2})	37
	Mitomycin C (1.0 μg ml^{-1})	23
	Nalidixic acid (50 μg ml^{-1})	1
Anabaena sp. strain PCC 7120	None	1
	UV light (360 J m^{-2})	22
	Mitomycin C (0.5 μg ml^{-1})	25
	Nalidixic acid (50 μg ml^{-1})	1

[a] Reactivation was measured as the titer of UV-irradiated N-1 (240 J m^{-2}) on treated cells divided by the titer of the same irradiated phage on untreated cells.

The cyanophage used in this study were sensitive to UV irradiation; however, the host cells could photoreactivate these phage very efficiently. At doses of 180 to 240 J m^{-2} to cyanophage N-1, photoreactivation increased the number of PFU by 100- to 1,000-fold. In addition to photoreactivation, strains of *Anabaena* spp. could induce Weigle reactivation of N-1 and of other cyanophages. In *E. coli*, Weigle reactivation is the result of induction, by UV light and by a variety of other agents, of a repair pathway called SOS (10, 23). The ability of these strains of *Anabaena* spp. to induce Weigle reactivation of cyanophages is evidence that a UV-inducible repair pathway also exists in these organisms. The levels of Weigle reactivation of N-1 in *Anabaena* sp. strain PCC 7120 were significant; however, they were at least an order of magnitude lower than the levels of photoreactivation of phage treated with similar doses of UV light. The fact that incubation of UV-irradiated cells under white light abolished their ability to induce Weigle reactivation of N-1 suggests that pyrimidine dimers were the inducing signal in UV-irradiated cells. However, pyrimidine dimers were not necessary for Weigle reactivation of N-1. Mitomycin C was equally effective in causing Weigle reactivation, and incubation of cells under conditions that allowed photoreactivation did not reverse the Weigle reactivation induced by mitomycin C. Thus it appeared that, in *Anabaena* spp., as in *E. coli* and in many other microorganisms, damage to DNA induced

a repair pathway directly or indirectly. Nalidixic acid, which induces the SOS pathway in *recBC*$^+$ *E. coli* strains (7, 12), did not cause Weigle reactivation of N-1 in two strains of *Anabaena* spp.

Weigle reactivation of N-1 in *A. variabilis* ATCC 29413 was severalfold higher than in the other strains of *Anabaena* spp.; however, this increase was probably attributable to the increased plating efficiency of N-1 (unirradiated) on irradiated *Anabaena* spp. cells versus that on unirradiated cells of the former strain. Cyanophage N-1 plated poorly on *A. variabilis* ATCC 29413 compared with the other strains of *Anabaena* spp., and we (unpublished data) and others (9) have observed that N-1 adsorbs poorly to this strain. The enhancement of plating efficiency by irradiation of *A. variabilis* ATCC 29413 with UV light could not be explained by better adsorption, and we have been unable to identify the step in the infection process that was enhanced by UV irradiation of the host. A similar phenomenon has been reported in UV-irradiated monkey cells infected with simian virus 40 and has been called "enhanced capacity" (8). If we assume in the case of N-1 that the level of enhanced capacity is similar for both irradiated and unirradiated phage, then the Weigle reactivation of N-1 (total reactivation divided by enhanced capacity) is similar in all the strains of *Anabaena* spp. tested.

The repair pathway that was induced by UV light or mitomycin C remained active for up to 72 h. UV-irradiated cells that were incubated under conditions that allowed photoreactivation lost inducible repair activity by about 24 h after the initiation of photoreactivation. This loss suggests that lesions caused by UV light or by mitomycin C were repaired slowly by the inducible repair and "dark" repair pathways of these organisms. Since removal of the dimers in UV-irradiated cells by photoreactivation resulted in loss of Weigle reactivation by 24 h, it is unlikely that in nonphotoreactivating cells an inducible, but relatively stable, repair pathway persisted, allowing Weigle reactivation for up to 3 days. It appears more likely that the lesions themselves remained. This suggests that, in the absence of photoreactivation, even with a UV-inducible repair pathway, repair of DNA damage was relatively inefficient in these strains. The much greater efficiency of photoreactivation, as compared with Weigle reactivation, of UV-damaged N-1 also supports the idea that photoreactivation was the more active pathway for repair of pyrimidine dimers. The function of the inducible repair pathway is not known; however, it presumably aids in repair of pyrimidine dimers and functions in the repair of other lesions that are not a substrate for the photoreactivation enzyme(s).

ACKNOWLEDGMENTS

We thank Sheri Bonar and Denise Jadali for excellent technical assistance.

This research was supported by National Science Foundation Grant DCB-8508542.

LITERATURE CITED

1. **Adolph, K. W., and R. Haselkorn.** 1971. Isolation and characterization of a virus infecting the blue-green alga *Nostoc muscorum*. Virology **446**:200–208.
2. **Allen, M. B., and D. I. Arnon.** 1955. Studies on nitrogen fixing blue-green algae. I. Growth and nitrogen fixation by *Anabaena cylindrica* Lemm. Plant Physiol. **30**:366–372.
3. **Asato, Y.** 1972. Isolation and characterization of ultraviolet light-sensitive mutants of the blue-green alga *Anacystis nidulans*. J. Bacteriol. **110**:1058–1064.
4. **Asato, Y.** 1976. Ultraviolet light inactivation and photoreactivation of AS-1 cyanophage in *Anacystis nidulans*. J. Bacteriol. **126**:550–552.
5. **Bhattacharjee, S. K.** 1977. Unstable protein mediated ultraviolet light resistance in *Anacystis nidulans*. Nature (London) **269**: 82–83.
6. **Braun, A. C., and H. N. Wood.** 1962. On the activation of certain essential biosynthetic systems in cells of *Vinca rosea* L. Proc. Natl. Acad. Sci. USA **48**:1776–1782.
7. **Chaudbury, A. M., and G. R. Smith.** 1985. Role of *Escherichia coli* RecBC enzyme in SOS induction. Mol. Gen. Genet. **201**: 525–528.
8. **Cornelis, J. J., B. Klein, J. H. Lupker, P. J. Abrahams, R. A. M. Hooft van Huysduynen, and A. J. van der Eb.** 1982. The use of viruses to study DNA repair and induced mutagenesis in mammalian cells, p. 337–350. *In* A. T. Natarajan, G. Obe, and H. Altman (ed.), Progress in mutation research, vol. 4. DNA repair, chromosome alteration and chromatin structure. Elsevier Biomedical Press, Amsterdam.
9. **Currier, T. C., and C. P. Wolk.** 1979. Characteristics of *Anabaena variabilis* influencing plaque formation by cyanophage N-1. J. Bacteriol. **139**:88–92.
10. **Defais, M., P. Fauquet, M. Radman, and M. Errera.** 1971. Ultraviolet reactivation and ultraviolet mutagenesis of lambda in different genetic systems. Virology **43**:495–503.
11. **Geoghegan, C. M., and J. A. Houghton.** 1987. Molecular cloning and isolation of a cyanobacterial gene which increases the UV and methyl methanesulfonate survival of *recA* strains of *Escherichia coli* K12. J. Gen. Microbiol. **133**:119–126.
12. **Gudas, L. J., and A. B. Pardee.** 1975. Model for regulation of *Escherichia coli* DNA repair functions. Proc. Natl. Acad. Sci. USA **72**:2330–2334.
13. **Hanawalt, P. C., P. K. Cooper, A. K. Ganesan, and C. A. Smith.** 1979. DNA repair in bacteria and mammalian cells. Annu. Rev. Biochem. **48**:783–836.
14. **Harm, W.** 1968. Dark repair of photorepairable UV lesions in *Escherichia coli*. Mutat. Res. **6**:25–35.
15. **Hu, H. -T., T. Thiel, T. H. Giddings, Jr., and C. P. Wolk.** 1981. New *Anabaena* and *Nostoc* cyanophages from sewage settling ponds. Virology **114**:236–246.
16. **Koz'yakov, S. Y., B. V. Gromov, and I. Y. Khudyakov.** 1972. A-1(L) cyanophage of the blue-green alga *Anabaena variabilis*. Mikrobiologiya **41**:555–559.
17. **Lambert, J. A. M., E. Williams, P. A. O'Brien, and J. A. Houghton.** 1980. Mutation induction in the cyanobacterium *Gleocapsa alpicola*. J. Gen. Microbiol. **121**:213–219.
18. **Little, J. W.** 1983. The SOS regulatory system: control of its state by the level of RecA protease. J. Mol. Biol. **167**:791–808.
19. **Little, J. W., and D. W. Mount.** 1982. The SOS regulatory system of *Escherichia coli*. Cell **29**:11–22.
20. **Marsden, H. S., E. C. Pollard, W. Ginoza, and E. P. Randall.** 1974. Involvement of *recA* and *exr* genes in the in vivo inhibition of the *recBC* nuclease. J. Bacteriol. **118**:465–470.
21. **O'Brien, P. A., and J. A. Houghton.** 1982. Photoreactivation and excision repair on UV induced pyrimidine dimers in the unicellular cyanobacterium *Gleocapsa alpicola* (*Synechocystis* PCC 6803). Photochem. Photobiol. **35**:359–364.
22. **O'Brien, P. A., and J. A. Houghton.** 1982. UV induced DNA degradation in the cyanobacterium *Synechocystis* PCC 6803. Photochem. Photobiol. **36**:417–422.
23. **Radman, M.** 1975. SOS repair hypothesis: phenomenology of an inducible DNA repair which is accompanied by mutagenesis, p. 355–367. *In* P. Hanawalt and R. B. Setlow (ed.), Molecular mechanisms for repair of DNA, part A. Plenum Publishing Corp., New York.
24. **Saito, N., and H. Werbin.** 1970. Purification of a blue-green algal deoxyribonucleic acid photoreactivating enzyme. An enzyme requiring light as a physical cofactor to perform its catalytic function. Biochemistry **3**:2610–2620.
25. **Satta, G., L. Gudas, and A. B. Pardee.** 1979. Degradation of *E. coli* DNA: evidence for limitation *in vitro* by protein X, the *recA* gene product. Mol. Gen. Genet. **168**:69–80.
26. **Singh, P. K.** 1975. Photoreactivation of UV-irradiated blue-

green algae and algal virus LPP-1. Arch. Microbiol. **103**:297–302.
27. **Srivastava, B. S., H. D. Kumar, and H. N. Singh.** 1971. The effect of caffeine and light on killing of the blue-green alga *Anabaena doliolum* by ultraviolet radiation. Arch. Microbiol. **78**:139–144.
28. **Stanier, R. Y., R. Kunisawa, M. Mandel, and G. Cohen-Bazire.** 1971. Purification and properties of unicellular blue-green algae (order *Chroococcales*). Bacteriol. Rev. **35**:171–205.
29. **Tang, T., and Y. Asato.** 1978. Ultraviolet light induction and photoreactivation of thymine dimers in the cyanobacterium *Anacystis nidulans*. Arch. Microbiol. **118**:193–197.
30. **Van Baalen, C.** 1968. The effects of ultraviolet irradiation on a coccoid blue-green alga: survival, photosynthesis, and photoreactivation. Plant Physiol. **43**:1689–1695.
31. **Walker, G. C.** 1984. Mutagenesis and inducible responses to deoxyribonucleic acid damage in *Escherichia coli*. Microbiol. Rev. **48**:60–93.
32. **Walker, G. C.** 1985. Inducible DNA repair systems. Annu. Rev. Biochem. **54**:425–457.
33. **Walker, G. C., L. Marsh, and L. A. Dodson.** 1985. Genetic analyses of DNA repair: interference and extrapolation. Annu. Rev. Genet. **19**:103–126.
34. **Weigle, J. J.** 1953. Induction of mutations in a bacterial virus. Proc. Natl. Acad. Sci. USA **39**:628–636.
35. **Werbin, H., and C. S. Rupert.** 1968. Presence of photoreactivating enzyme in blue-green algal cells. Photochem. Photobiol. **7**:225–230.
36. **Williams, E., J. Lambert, P. O'Brien, and J. A. Houghton.** 1979. Evidence for dark repair of ultraviolet light damage on the blue-green alga *Gleocapsa alpicola*. Photochem. Photobiol. **29**:543–547.
37. **Witkin, E. M.** 1976. Ultraviolet mutagenesis and inducible DNA repair in *Escherichia coli*. Bacteriol. Rev. **40**:869–907.
38. **Wolk, C. P., and E. Wojciuch.** 1973. Simple methods for plating single vegetative cells of, and for replica plating, filamentous blue-green algae. Arch. Mikrobiol. **91**:91–95.
39. **Wu, J. H., R. A. Lewin, and H. Werbin.** 1967. Photoreactivation of UV-irradiated blue-green algal virus LPP-1. Virology **31**: 657–664.
40. **Yasbin, R. E.** 1977. DNA repair in *Bacillus subtilis*. I. The presence of an inducible system. Mol. Gen. Genet. **153**:211–218.
41. **Yasbin, R. E.** 1977. DNA repair in *Bacillus subtilis*. II. Activation of the inducible system in competent bacteria. Mol. Gen. Genet. **153**:219–225.