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*Review Article*

# UV-MEDIATED STRESS AND ITS MITIGATION IN CYANOBACTERIA

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**ABSTRACT** : A substantial loss in the stratospheric ozone layer due to anthropogenically released chemicals has aroused concern about the effects of increased solar ultraviolet radiation (UVR), particularly UV-B (280-315 nm), on the Earth's surface. Solar UV-B radiation is detrimental for most sun-exposed organisms, including humans. They can penetrate deep into biologically significant depths in lakes, ponds, rivers and oceans. Cyanobacteria, the primitive $O_2$-evolving photosynthetic prokaryotes are solely dependent upon the solar radiation and thereby have to always face the UV-B stress. UVR affects cyanobacteria either directly or indirectly by inducing oxidative stress. In response, these organisms have developed a number of defense mechanisms such as avoidance, scavenging, screening, repair and programmed cell death to counteract its damaging effects. This review presents an overview on the effects of UVR on cyanobacteria and the defense mechanisms employed by these prokaryotes to withstand UVR stress.

**Keywords:** Cyanobacteria, DNA damage, Mycosporine-like amino acids (MAAs), Nitrogenase, Phycobilisomes, Scytonemin, Ultraviolet radiation.

## INTRODUCTION

The anthropogenically released atmospheric pollutants such as chlorofluorocarbons (CFCs), chlorocarbons (CCs) and organobromides (OBs) has resulted in the continued depletion of the stratospheric ozone layer and subsequent increase in the ultraviolet radiation (UVR; 280-400 nm) impinging onto the Earth's surface [1]. Natural production of considerable amounts of reactive nitrogen species (RNS) such as nitric oxide ($NO^-$), peroxynitrite ($ONOO^-$) and nitrous oxide ($N_2O$), from unpolluted terrestrial and aquatic ecosystems or from anthropogenic sources also contribute to the depletion of the ozone layer [2]. The processes of ozone depletion has been reported at mid latitudes but are more pronounced in the Antarctic region where ozone levels have been reported to decline by more than 70% during late winter and early spring in the polar vortex [3]. The harmful UVR reaching on the Earth's surface is absorbed by biomolecules such as nucleic acids and proteins ultimately resulting in lethal effects on the biological systems [4]. The negative impact of highly energetic UV-B (280-315 nm) radiation on aquatic and terrestrial life forms ranging from prokaryotic bacteria to eukaryotic lower and higher plants, animals as well as humans has aroused the scientific concern [5]. Cyanobacteria, the primitive group of Gram-negative, oxygenic photoautotrophic prokaryotes have cosmopolitan distribution ranging from hot springs to the Arctic and Antarctic regions and are important biomass producers in both aquatic and terrestrial ecosystems [4,6]. They are valuable sources of various natural products of medicinal and industrial importance [7]. In addition, these ecologically important organisms act as natural biofertilizers [8] by virtue of their ability to fix atmospheric nitrogen in the presence of the enzyme nitrogenase. Harvesting of solar energy to perform photosynthesis and nitrogen fixation exposes these cyanobacteria to lethal doses of UV-B and UV-A (315 - 400 nm) radiations in their natural brightly lit habitats. Both UV-B and UV-A causes chronic and physiological stress in cyanobacteria either by direct or indirect effects. It is evident that morphology, cell differentiation, growth, survival, pigmentation, motility and orientation, $N_2$ metabolism, phycobiliprotein composition, protein profile, DNA and $^{14}CO_2$ uptake are severely affected by UVR [9-11]. These changes could result from a number of primary UV-B mediated events such as direct photosynthetic damage, loss of permeability/membrane changes, pigment destruction, protein/enzyme inactivation, reduced DNA and protein synthesis, reduced uptake of nutrients, hormone inactivation and signal transduction through phycochrome or signal transduction via a specific UV-B photoreceptor [12,13].

Cyanobacteria have evolved a number of mitigation strategies to minimise the lethal effects of UVR. These defensive mechanisms includes avoidance, scavenging of ROS by antioxidants, accumulation of carotenoids, synthesis of UV-absorbing/screening compounds such as mycosporine-like amino acids (MAAs) and scytonemin [14], repair of UV-induced DNA damage by photoreactivation and excision repair [11,15] and resynthesis of proteins. This review provides an overview on the effects of UVR on cyanobacteria as well as mitigation strategies employed by them to cope with harmful radiations.

## Impacts of UVR on cyanobacteria

Some of the pronounced effects of UV on various metabolic processes of cyanobacteria are as follows:

**(A) Growth and survival**

The growth and survival of several cyanobacteria has been reported to be severely affected by UV-B radiation, thereby bringing about complete killing within 120-180 min of exposure [16,17]. Various species differ with respect to their tolerance to UV-B radiation and even closely related strains show differential sensitivity. The growth of Antarctica cyanobacterium *Oscillatoria priestleyi* was completely suppressed whereas it was 62% in case of *Phormidium murrayi* following similar dosage of UV exposure [18]. Mucilagenous covering of the filaments of *Nostoc commune* and *Scytonema* sp. provides them more tolerance to UVR as compared to the strains lacking the sheath [19].

**(B) Motility**

The avoidance mechanism in terms of motility and orientation employed by cyanobacteria to cope up with the harmful solar UV radiation has been reported to be adversely affected by UV-B radiation. A significant decrease in the number of motile filaments of *Anabaena variabilis, Oscillatoria tenuis* and *Phormidium uncinatum*, showing gliding motility was observed within 10-30 min of UVR exposure [20]. UV-B radiation also inhibits the phototactic orientation and photophopic responses in the cyanobacteria, thereby reducing the ability of these organisms to orient themselves in their photo-environment and ultimately leading to their death [21].

**(C) Cell differentiation**

UV-B radiation has a pronounced detrimental effect on cellular differentiation in certain cyanobacteria. Exposure of cyanobacterial cells to UV-B radiation has been found to delay the differentiation of vegetative cells into heterocyst and akinete especially in *Anabaena aqualis* [22]. Gao et al. [9] reported the suppression of heterocyst differentiation in *Anabaena* sp. PCC 7120. An alteration in C:N ratio following UV exposure was suggested to be responsible for the altered spacing pattern of heterocysts in the filament [19]. A decrease in the level of major heterocyst polypeptides (26, 54 and 55 kDa) following UV-B treatment has been noted which is presumed to be responsible for the disruption of multi-layered heterocyst wall, the essential component for maintaining the active form of enzyme nitrogenase [19]. Role of UV-B radiation in the opening of specific calcium channels in the heterocyst of *Anabaena* sp. has also been demonstrated [23]. Recently it has been reported that spiral filaments of *Arthrospira platensis* are broken and compressed under solar UVR [24,25].

**(D) Pigmentation**

Several studies have been conducted to show the photobleaching of photosynthetic pigments by UV-B radiation. The accessory light-harvesting pigment, phycobiliproteins (phycoerythrin; $\lambda_{max}$ 540-570 nm, phycocyanin; $\lambda_{max}$ 610-620 nm and allophycocyanin; $\lambda_{max}$ 650-655 nm) are arranged in a macromolecular complex called phycobilisomes [26]. Sinha et al. [27,28] reported a decrease in phycobiliprotein content and disassembly of phycobilisomal complex following UV-B irradiation in a number of cyanobacteria. The pattern of fluorescence emission spectra of phycobiliproteins observed after UV-B irradiation suggested an impairment of the energy transfer from the accessory pigments to the photosynthetic reaction center. Time-dependent bleaching and destruction of phycobiliproteins following increasing UV-B exposure time is evident from the absorption spectra (Figure1) in *Nostoc* sp. SDS-PAGE analysis of phycocyanin and associated linker polypeptides of *Anabaena* sp. revealed a loss of αβ monomers of phycocyanin, rod-core and core membrane linker polypeptides after UV-B irradiation [29]. Several studies by various workers suggests that chlorophyll and carotenoids are also negatively influenced by UV-B radiation [9, 10, 30, 31, 32].

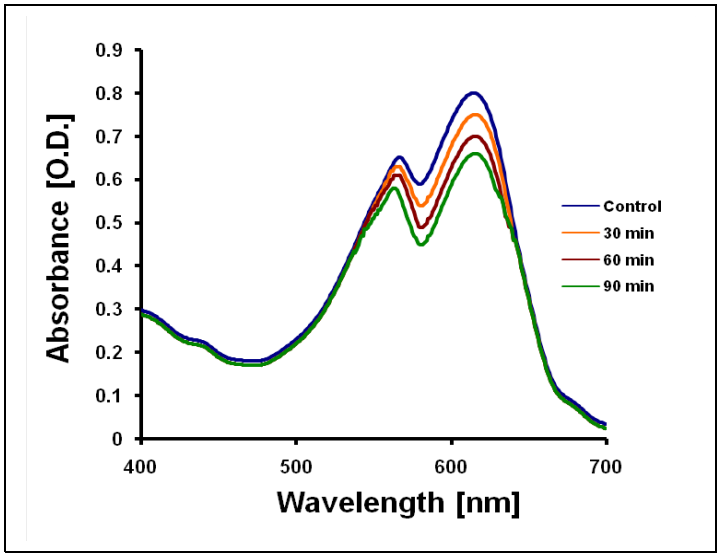



**Figure 1.** Effects of UV-B irradiation on absorption spectrum of the phycobiliproteins in *Nostoc* sp.

**(E)Photosynthesis**

Certain photosynthetic parameters such as $^{14}CO_2$ uptake, $O_2$ evolution and ribulose-1,5 bisphosphate carboxylase (RUBISCO) activity in cyanobacteria are inhibited by UVR [33]. Häder and Worrest [21] reported a decline in $O_2$ evolution in *Phormidium* strain within few minutes of UV exposure. It is suggecsted that RuBISCO may undergo a number of modifications such as photo-degradation, fragmentation and denaturation of polypeptide chain, change in active site and increased solubility of membrane proteins [34]. In addition, supply of ATP and $NADPH_2$ may also be impaired which may cause inhibition of $CO_2$-fixing ability. Exposure of cyanobacterial cultures to intermediate levels of UV-B radiation brings about the degradation of $D_1$ and $D_2$ polypeptides which are the major constituents of PS-II reaction centers [35,36]. Down regulation of several families of transcripts including mRNAs specifying proteins involved in light harvesting and photosynthesis by UV-B treatment was in accordance with the reduced photosynthetic activity [37]. UV-B radiation also induces the lipid peroxidation of polyunsaturated fatty acids (PUFA) via oxidative damage which subsequently affects the integrity of cellular and thylakoid membranes [38] thereby causing damage of photosynthetic components.

**(F) Nitrogen metabolism**

Nitrogenase, the key enzyme for nitrogen fixation is extremely sensitive to UV radiation [10,13,16]. Nitrogenase activity was found to be completely inactivated within 25-40 min of UV-B exposure in a number of rice-field cyanobacteria [13]. In a *Nostoc* sp., nitrogenase activity was lost within 45 min of UV-B exposure whereas nitrate reductase and glutamine synthetase activity remained more or less unaffected [16]. The loss of activity most probably occurs due to complete damage to the nitrogenase polypeptide. Various workers have suggested that the aromatic amino acids present or the native structure of nitrogenase is responsible for the extremely susceptible nature of nitrogenase to UV-B radiation.

**(G) Protein content**

Several studies have been conducted by various workers to show that UV-B radiation has detrimental effects on protein profile of several cyanobacteria [12, 30]. The number and quantity of protein bands in various cyanobacteria was found to decrease linearly with the increased duration of UV exposure. UV-B radiation severely affects the low molecular weight proteins, reported in *Nostoc* sp. where the αβ monomers of phycocyanin (approximately 20 kDa) were the most affected one. Kumar et al. [39] reported complete loss of protein bands between 14.2 and 45 kDa after 90 and 120 min of UV-B exposure in *Nostoc calcicola*. However, protein bands of relatively higher molecular weight viz., 55 and 66 kDa were unaffected even after 120 min of UV-B exposure. In *Nostoc commune*, out of 1350 protein spots, 493 were found to be changed by UV-B radiation while monitoring the proteome change following UV-B exposure using two-dimensional gel electrophoresis [40].

**(H) DNA damage**

DNA, the most prominent targets of solar UVR in all living organisms continuously incurs a myriad of types of damage that drastically attribute adverse effects on all living organisms including cyanobacteria. UVR induces DNA lesions either by directly absorbing the radiation by native DNA molecule or indirectly by oxidative stress. These UV-induced lesions consist of dimeric photoproducts such as cis-syn cyclobutane pyrimidine dimers (CPDs) and pyrimidine (6-4) pyrimidone photoproducts (6-4 PPs) and their Dewar isomers [12,41]. It has been reported by Britt [42] and Kumar et al. [43] that dimers inhibits the advancement of DNA polymerase, and brings about the stallation of mammalian RNA polymerase at both CPDs and 6-4 PPs. DNA synthesis and RNA transcription are adversely affected, ultimately leading to mutation or death of the organism following the formation of photoproducts. Contrary to this, oxidative stress commonly results in single- or double-strand breaks in the native DNA molecule [38].

The formation of UV-induced thymine dimers in three rice-field cyanobacteria, *Anabaena, Nostoc* (Figure 2) and *Scytonema* sp. has been demonstrated by a quantitative technique based on a blotting and chemiluminescence method developed by Sinha et al. [44]. It was also found that the frequency of thymine dimers increased with the increase in UVR exposure time, and after 120 min of exposure it reached 35-40 T^T/Mbp in all three cyanobacteria. A hypochromic effect in genomic DNA of the *Anabaena* strain BT2 after UVR exposure was studied by Kumar et al. [43] and they reported damaged template activity of DNA following UVR exposure using a PCR-based assay such as RAPD (random amplified polymorphic DNA), rDNA amplification and ARDA (restriction analysis of 16S rDNA). Quantification of the DNA strand breaks induced by ROS under UV-A and UV-B exposure in the cyanobacterium *Anabaena* sp. was done by He et al. [45] using fluorimetric analysis of DNA unwinding (FADU) assay. Recently, temperature and biomass dependent CPDs formation under UV stress in *A. platensis* has been reported Gao et al. [24].

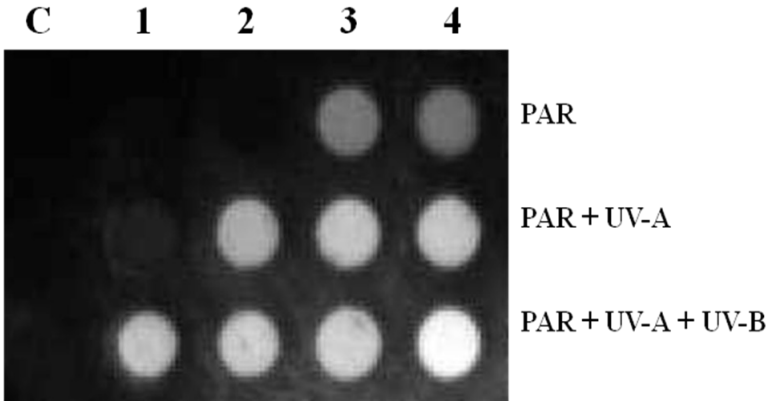



**Figure 2.** Thymine dimer formation in *Nostoc* sp. under various radiation conditions. C - control; lane 1 - 30 min; lane 2 - 60 min; lane 3 - 90 min and lane 4 - 120 min.

**Mitigation strategies in cyanobacteria**

As cyanobacteria are believed to have originated in the Precambrian era at a time when the ozone shield was absent, they presumably faced high fluxes of UV radiation, which must have acted as an evolutionary pressure leading to the selection for efficient UV radiation protecting mechanisms. Tolerance of cyanobacteria to intense sunlight as well as UV radiation might have contributed to their success during early stages of colonization. As a consequence they have developed effective mechanisms to counteract the damaging effects of UV-B radiation. Defense mechanisms employed by the cyanobacteria to cope up with the harmful radiation have been dealt below:

**(i) Avoidance**

Avoidance as a first line of defense mechanisms against high solar UVR includes migration from high to low UVR levels in the water column, formation of mats containing different cyanobacterial species or filaments enclosed in amorphous silica matrices, changes in morphology to increase self-shading, photokinetic and photophobic reactions and synthesis of extracellular polysaccharides. This mechanism depends on the motility and mat forming ability of cyanobacteria as well as on the turbidity and depth of the water column.

Motile cyanobacteria can escape from high solar radiation by downward migration into the mat communities [18] or by sinking deeper into the water column [46]. It has been reported that *Oscillatoria cf. laetevirens* and *Spirulina cf. subsalsa* from hypersaline ponds near Mexico, protect their photosynthetic apparatus from UV-A and UV-B radiation by downward migration [47]. Similarly, *Microcoleus chthonoplastes* in the microbial mats of lake Sinai, Egypt showed migration in response to UV-B [48]. Self-shading as an effective protective mechanism against photo inhibition has been reported in *Arthrospira platensis* resulting from decreased helix pitch in presence of UV-B [25]. The synthesis of extracellular glycan in *Nostoc commune* was stimulated by UV-B and anticipated to provide UV resistance by increasing the effective path length for the absorption of radiation [49]. Phoenix et al. [50] found that the deposits of amorphous silica matrices over the cyanobacterial mats provide protection against lethal UVR.

**(ii) Antioxidative systems**

The interaction of UVR with oxygen and other organic compounds within the cell produces toxic ROS such as superoxide ($O_2^-$), hydroxyl radical ($OH^-$) or hydrogen peroxide ($H_2O_2$) finally resulting in oxidative stress. To overcome this stress an antioxidant system consisting of enzymatic and non-enzymatic antioxidants has been developed by cyanobacteria as a second line of defence. Non-enzymatic antioxidants comprise ascorbate (vitamin C), α-tocopherol (vitamin E), carotenoids and reduced glutathione. The enzymatic antioxidants include superoxide dismutase (SOD), catalase, glutathione peroxidase (GSH-Px) and the enzymes involved in the ascorbate-glutathione cycle to detoxify ROS, such as ascorbate peroxidase (APX), monodehydroascorbate reductase (MDHAR), dehydroascorbate reductase (DHAR) and glutathione reductase (GR) [38]. Carotenoids protect cells against photooxidative damage by absorbing triplet state energy from chlorophyll and quenching singlet state oxygen while α-tocopherol prevents lipid peroxidation by scavenging ROS [51]. However, ascorbate performs direct quenching of ROS, regenerate α-tocopherol and acts as a substrate in both violaxanthin de-epoxidase and APX reactions. Glutathione protects thiol groups in various enzymes and is also involved in α-tocopherol and ascorbate regeneration through the glutathione-ascorbate cycle [51]. SOD scavenges superoxide radicals and converts them to hydrogen peroxide which is further converted to water and $O_2$ via a combined catalase-peroxide system [52].

Latifi et al. [53] has recently reported the presence of three types of catalase-orthologues i.e., monofunctional haem-containing catalases, bifunctional haem-containing catalase-peroxidases and nonhaem-manganese catalases in 20 cyanobacterial genome. Genomic analysis revealed that NiSOD is the only SOD found in primitive cyanobacteria, Fe and Mn occupy the higher orders of cyanobacteria and Cu/ZnSOD is rare in cyanobacteria. The most evolved filamentous, heterotrichous and heterocystous forms predominantly have only Fe and Mn metalloforms [54]. UV-B irradiation was reported to increase the carotenoid content in *N. Commune* and myxoxanthophyll and echinenone were suggested to act as outer membrane-bound UV-B photoprotectors [49] and their induction by UV-B radiation is a fast active SOS response.

The results from an mRNA profile study revealed an increase in the transcripts for photoprotective molecules such as carotenoids, glutathione peroxidase and superoxide dismutase in *Synechocystis* sp. PCC 6803 [37]. ROS regulates the photosynthetic genes and induces the antioxidants, and thus acts as a signal molecule. The exogenous addition of antioxidants such as ascorbic acid and N-acetylcysteine (NAC) was found to reduce chlorophyll bleaching, damage to the photosynthetic apparatus, lipid peroxidation and DNA strand breaks under UVR-induced oxidative damage ultimately resulting in higher survival rate of *Anabaena* sp. [32]. An accumulation of active iron superoxide dismutase (FeSOD) in desiccated field cyanobacterium *N. commune* was found to reverse the effects of oxidative stress imposed by multiple cycles of desiccation and rehydration during the UV-A or UV-B irradiation in situ [55]. Two NADPH-dependent glutathione peroxidase-like proteins have been characterized in *Synechocystis* PCC 6803 and were found to provide protection to the membranes against lipid peroxidation [56]. Besides these, Mycosporine-glycine, a biological antioxidant was found to effectively stifle various detrimental effects of the type-II photosensitization and decreases the level of singlet oxygen generated by certain basic dyes [57].

**(iii) Screening**

The synthesis of UV-absorbing compounds in certain cyanobacteria has evolved as a third line of defence against UV-induced photodamage [58]. MAAs and scytonemin are well known UV-absorbing/screening compounds that provide photoprotection against UV-B and/or UV-A radiations [14]. Photoprotective role of these UV-screening compounds has been briefly described below:

**(a) Mycosporine-like amino acids (MAAs):**

MAAs are small (<400 Da), colorless, water-soluble compounds composed of a cyclohexenone or cyclohexenimine chromophore conjugated with the nitrogen substituent of an amino acid or its imino alcohol [59, 60]. These natural products are attached to the core through imine linkages, leading to a combination of resonanting tautomers, which facilitates UV- absorption [61]. The biosynthesis of MAAs and mycosporines has been suggested to occur via the first part of the shikimate pathway, details of which has been recently published by Singh et al. [62]. Absorption spectra of MAAs range from 310-362 nm due to variations in the attached side groups and nitrogen substituents. Characteristic feature of some MAAs has been elucidated in Table1. In addition to photostabilization, their ability to resist certain physico-chemical stressors like temperature, strong UVR, various solvents as well as pH make them successful photoprotectants in various habitats and organisms [63]. MAAs protect the cells by absorbing highly energetic UVR and then dissipating this energy in the form of harmless heat radiation to their surroundings [64]. They can also act as antioxidants to prevent damage from ROS resulting from UVR [65]. Recent report suggests that the MAA shinorine in *Anabaena variabilis* PCC 7937 is synthesized under various abiotic stressors with or without UVR [66], indicating their additional role. Singh et al. [66] reported the dependency of MAAs synthesis on available nitrogen and highest MAAs synthesis was supported in the growth media having nitrogen source [67]. Recently, in the rice-field cyanobacterium *A. doliolum* three MAAs (mycosporine-glycine, porphyra-334 and shinorine) has been found to be biosynthesized. The protection efficiency of MAAs against UVR depends on the location of these compounds in the cell. A significant, but limited, protection by MAAs located in the cytoplasm has been reported for various cyanobacteria [68], however, in *N. commune*, MAAs are actively excreted and accumulated extracellularly; therefore, they are more effective and act as true screening compounds [49].

**(b) Scytonemin**

Scytonemin is a yellow-brown lipid soluble pigment located in the extracellular polysaccharide sheath of some terrestrial cyanobacterial species [14]. Scytonemin is a dimer composed of indolic and phenolic subunits (Figure 3) having a molecular mass of 544 Da and has an *in vivo* absorption maximum at 370 (Figure 4) nm in the UV-A region but shows considerable absorption in the UV-B region also. Purified scytonemin shows absorption maximum at 386 nm although there being substantial absorbance at 252, 278 and 300 nm. UV-A in combination with increased temperature and oxidative stress have a synergistic effect on scytonemin synthesis [69]. Microspectrophotometric measurements of the transmittance of pigmented sheath and the quenching of ultraviolet excitation of phycocyanin fluorescence suggest that the pigment is effective in shielding the cells from incoming UV radiation and is able to reduce the entry of UV-A radiation into the cells to around 90% [68,70].

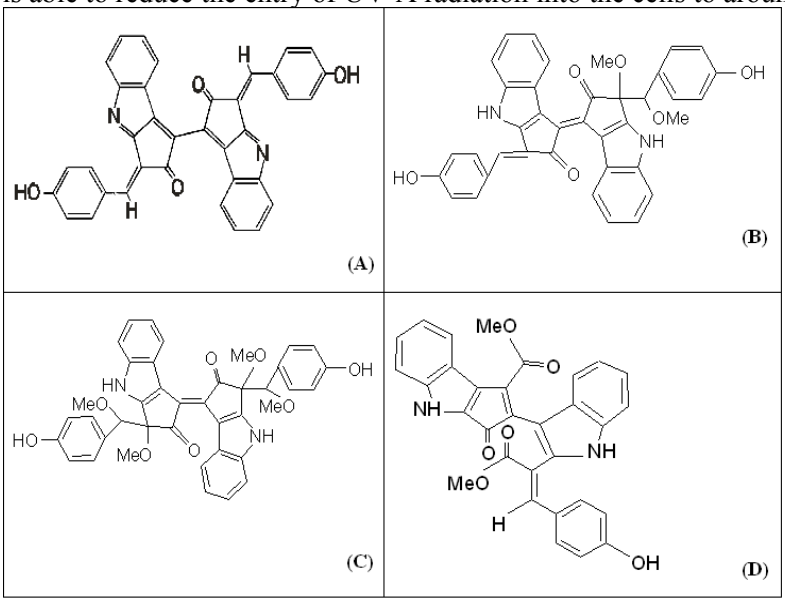
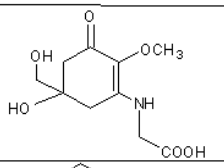
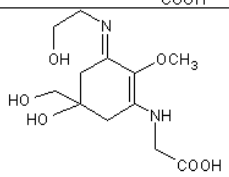
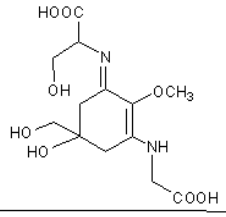
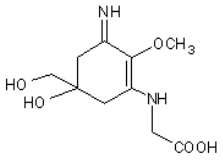
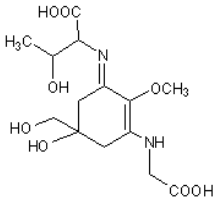
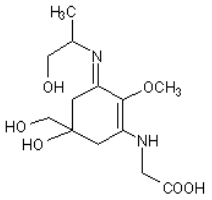
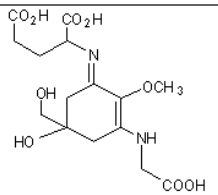
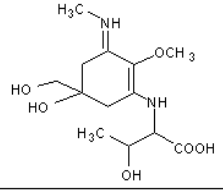



**Figure 3:** Chemical structure of scytonemin (A), dimethoxyscytonemin (B), tetramethoxyscytonemin (C) and scytonin (D).

**Table 1: Cyanobacterial mycosporine-like amino acids (MAAs) with their corresponding molecular structure, absorption maxima and extinction coefficient.**

| MAAs | Molecular Structure | $\lambda_{max}$ (nm) 310 | ε ($M^{-1}$ $cm^{-1}$) 28100 |
|---|---|---|---|
| Mycosporine-glycine |  | | |
| Asterina-330 |  | 330 | 43500 |
| Shinorine |  | 334 | 44700 |
| Palythene |  | 360 | 50000 |
| Porphyra-334 |  | 334 | 42300 |
| Palythinol |  | 332 | 43500 |
| Mycosporine-glutamic acid-glycine |  | 330 | - |
| Mycosporine-methylamine-threonine |  | 327 | - |

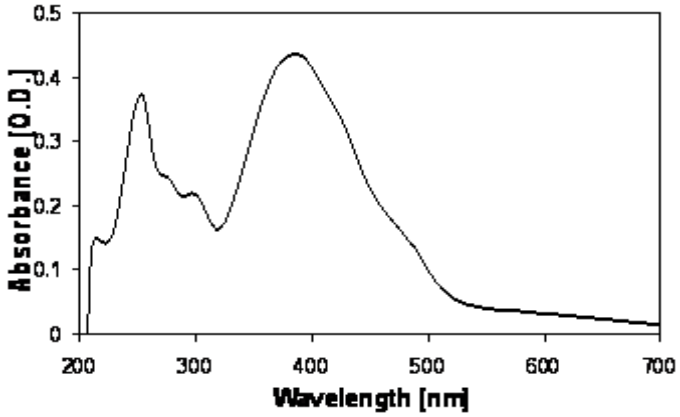



**Figure 4:** Absorption spectrum of scytonemin showing maximum absorbance at 386 nm.

Scytonemin may constitute as much as 5% of the total cellular dry weight or even higher in some naturally occurring cyanobacteria [71]. It is highly stable and able to persist very long in terrestrial cyanobacterial crusts or dried mats [70,72] and performs its screening activity without any further metabolic investment even under prolonged physiological inactivity (e.g. desiccation) [62]. Three new pigments such as tetramethoxyscytonemin, dimethoxyscytonemin, and scytonine from the organic extracts of *Scytonema* sp. (Figure 3) has been reported to be derived from the scytonemin skeleton of the scytonemin [73].

The scytonemin was proposed to be synthesized from metabolites of aromatic amino acid biosynthesis by condensation of tryptophan and phenylpropanoid-derived subunits [74]. Details of the pathway has been discussed by Singh et al. [62]. Besides having a role as UV protectant, scytonemin may have additional roles such as protection against pathogenic attack, bacterial decomposition or herbivore grazing [12].

**(iv) Repair**

The damaging effects of UV-B radiation can be alleviated by UV-A or visible radiation suggesting that in nature severity of the damaging effects of UV-B can be reduced by PAR and UV-A present in the solar radiation. Cyanobacteria have developed certain repair mechanisms and resynthesis of sensitive targets as fourth line of defence. The existence of polyploidy in cyanobacteria may be of additional importance since this may mask the effect of single mutations of a DNA molecule. These mechanisms include photoreactivation by photolyase which converts UV induced dimers into monomers, the dark or excision repair and the recombinational repair [12,75]. Levine and Thiel [15] has reported the presence of a UV-inducible photoreactivation system in strains of *Anabaena* sp. such as *Anabaena* sp. PCC 7120, *A. variabilis* PCC 7937, *Anabaena* sp. M-131 and *A. variabilis* sp. PCC 7118. Homologus gene for photolyase has been identified and functionally characterized in *Synechocystis* sp. PCC 6803 [76]. The gene for the DNA repair enzyme Fpg (formamydopyrimidine-DNA glycosylase) has been reported from *Synechococcus elongatus* and was suggested to be involved in photoprotection against oxidative damage [77]. Han et al. [78] have also reported photoreactivation of UV-B induced inhibition of photosynthesis in *Anabaena* sp. Following UV exposure cyanobacteria also synthesizes new proteins to replace damaged copies. The UV-B shock and acclimation response of *N. commune* under UV-B stress was found to be a completely different and complex phenomenon influencing a total of 493 proteins [40]. D1 and D2 proteins of the photosystem II reaction center was found to provide protection and acclimatization against UV-B in *Synechocystis* sp. [36]. Recently, an autocatalytic Programmed Cell Death (PCD) has been shown to operate in the nitrogen-fixing cyanobacterium *Trichodesmium* sp. and was found to be induced by high irradiance, iron starvation and oxidative stress [79]. But the advantageous role of this mechanism under UV stress or UV-mediated oxidative stress is still not very clear.

## CONCLUSION

Intense UV-B radiation impinging onto the Earth's surface has detrimental effect on a number of vital physiological and biochemical processes of cyanobacteria leading to reduction in growth and survival. In addition, photosynthesis, nitrogen fixation, pigmentation, proteins and DNA are also adversely affected. Formation of thymine dimers, *cis-syn* cyclobutane dimers and pyrimidine (6-4) pyrimidone products following UV-B exposure in certain cyanobacteria has also been demonstrated. However, these organisms have developed several lines of defence mechanisms that sustain their successful growth and survival in various habitats receiving high solar UVR. The balance between damage and defence mechanisms also has the ecological importance as it maintains the productivity and nitrogen economy of an ecosystem and, thereby, regulating other climate change problems. Information related to the ecological significance of sun-screening substances such as scytonemin and MAAs and spatial distribution of MAAs within the cyanobacterial cells is still in its infancy stage. Since UV-B damage is ameliorated by PAR and UV-A, UV-B/UV-A ratio has a major influence on the extent of UV-B damage. Several reports suggests damage by UVR in cyanobacteria, however, all are seldomly distinguishing between direct damage by UVR and UVR-induced oxidative damage. Hence, the existence and significance of enzymatic defense mechanisms and repair of UV-B-induced DNA damage in cyanobacterial populations needs to be critically studied.

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