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UV-B Induced Effects and Repair Mechanism in Aquatic Cyanobacteria

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Mini Review

Stratospheric depletion of ozone layer has resulted in an increase in the solar UV-B radiation (280 to 320 nm) reaching the Earth's surface. UV-B radiation is potentially harmful to all forms of life but is more detrimental to photosynthetic organisms, including cyanobacteria. Cyanobacteria are the largest and most widely distributed group of photosynthetic prokaryotes on the Earth, and their contribution to global CO_2 and N_2 fixation is remarkable. These organisms are proficient in fixing atmospheric nitrogen using enzyme nitrogenase hence potentially recognized as a biofertilizer in rice paddies [1] and other crops. They are important constituent of aquatic ecosystem which is one of the most productive and diverse ecosystem thus any alteration in the size and composition of phytoplankton communities will directly affect its productivity. Hence, it is quite relevant to study the effects of ultraviolet (UV-B) radiation on them. The aquatic ecosystem has shown a large sensitivity towards increased solar ultraviolet radiation resulting in decreased biomass productivity, reduced food production for humans [2], reduced sink capacity for atmospheric carbon dioxide [3,5,6] as well as changes in species composition and ecosystem integrity. In cyanobacteria, UV-B radiation has been found to affect a number of physiological and biochemical processes such as growth, survival, cell differentiation, motility, pigmentation, photosynthesis, nitrogen metabolism, and protein profile [7,8,9]. It also affects membrane permeability, pigment stability, nutrient uptake mechanisms and signal transduction through phytochrome or specific UVB photoreceptors [10] Portwich [11]; Kumar et al. [12]; Cadoret et al. [13]. However, enzymes such as nitrate reductase, glutamine synthetase and glutamate synthase are less sensitive to elevated level of UV-B intensity [14] and the response of cyanobacteria towards UV-B radiation differs in various species. DNA and photosynthesis are recognized as the most predominant targets of UV-B [15]. It has been shown that UV-B affects the photosynthetic electron transport and pigment-protein complexes in cyanobacteria by photobleaching of photosynthetic pigments, reduction in phycobili protein content and disassembly of phycobilisome complex [16]. Additionally, several studies have demonstrated that UV-B radiation affects spectral properties of pigments specifically chlorophyll a and phycobiliproteins of cyanobacteria [8] and also influences the chlorophyll and carotenoids contents in cyanobacteria [8,17,18]. Observed a down regulation of transcripts including mRNAs specifying proteins involved in light harvesting and photosynthesis after UV-B exposure. The photosynthetic parameters such as CO_2 uptake, O_2 evolution and ribuolose-1, 5 bisphosphate carboxylase/oxygenase (RUBISCO) activities are also down regulated [9].

The D1 and D2 proteins that are major constituent of PSII reaction center are degraded by exposure of UV-B. Exposure of UV-B radiation also results in significant alterations of total protein profile of cyanobacteria [16]. Total proteome analysis of *Synechocystis* sp. PCC 6803 by 2-dimensional (2-D) gel electrophoresis showed different level of proteins expression in the cytoplasm under short and long-term UV-B stress [18]. Cyanobacteria exposed to UVR have evolved a number of mitigation strategies to reduce its direct and indirect damaging effects. The first line of protective strategies include migration from high level to low level of UV intensity in water column, formation of mats/crust, changes in morphology or synthesis of extracellular polysaccharides i.e. gypsum crystals. To escape from high solar radiation, motile cyanobacteria in mats often migrate up and down-wards depending on the spectral waveband [19]. In planktonic cyanobacteria, sinking and floating regulated by gas vacuoles are also protective strategies against UVR [20]. Another mode of defense is the generation of antioxidants in response to reactive oxygen species generated during UV-B stress. The enzymatic antioxidants are superoxide dismutase (SOD), catalase, glutathione peroxidase (GSH-Px) and the enzymes involved in the ascorbate-glutathione cycle to detoxify the ROS such as ascorbate peroxidase (APX), monodehydroascorbate

reductase (MDHAR), dehydroascorbate reductase (DHAR) and glutathione reductase (GR) [21]. Exposure of DNA to UV-B causes several types of DNA lesions, which are mainly repaired by photoreactivation (light-dependent) and excision repair (light-independent) mechanism. Photoreactivation occurs with the help of the photolyase enzyme that specifically binds to cyclobutane-pyrimidine dimers (CPDs) or 6-4 photo-lyase (6-4PPs) and reverses the damage after absorption of light energy at 400nm [22]. The major photo reactivating factor phrA in the *Cyanobacterium Synechocystis* sp. PCC6803 codes for a cyclobutane-pyrimidine dimer-specific DNA photolyase. In the excision repair process, various enzymes (e.g. glycosylases or polymerases) are involved. First, the damaged DNA is nicked and then the short single strand segments are important role in photoprotection as they are located in the extracellular glycan layer covalently linked to oligosaccharides [23,24]. In most cyanobacteria, however, MAAs is located in the cytoplasm, where only 10-26% of harmful radiation is absorbed by this compound [25]. Besides acting as sunscreens, MAAs may provide additional protection as antioxidants [26]. Another UV-absorbing component known for UV-screening properties in cyanobacteria is scytonemin which is formed by condensation of tryptophan and phenyl-propanoid derived subunits [27]. Cyanobacteria may also undergo apoptosis or programmed cell death (PCD) when a cell is damaged beyond repair. An autocatalytic PCD induced by high irradiance was found to operate in the nitrogen-fixing *Cyanobacterium Trichodesmium* sp. [28]. The caspase activity involved in PCD was observed in *Microcystis aeruginosa* [29] as well in *Trichodesmium* sp. [28] implicating the role of PCD under oxidative stress [30-35].

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