International Journal of Health Sciences and Research
Volume 14; Issue: 8; August 2024
Website: www.ijhsr.org
Review Article
ISSN: 2249-9571

Green Alchemy: Mechanisms of Cyanobacterial Photoprotection in the Spotlight

Prathima R[1], Mutthuraj D[1], Kshitija Aherkar[1], Kanthesh M Basalingappa[1]

[1]Division of Molecular Biology, School of Life Sciences, JSS academy of Higher Education & Research, Mysuru.

Corresponding Author: Dr. Kanthesh M Basalingappa

DOI: https://doi.org/10.52403/ijhsr.20240840

ABSTRACT

Cyanobacteria, ancient photosynthetic microorganisms, have perfected an intricate defense system against harsh ultraviolet radiation (UVR) and environmental stressors. Their primary defense deploys pigments like chlorophyll-a and carotenoids to absorb and dissipate excess energy, shielding vital cellular structures. Secondary defense strategies introduce non-enzymatic antioxidants like ascorbate and carotenoids, fortifying cyanobacteria against reactive oxygen species triggered by UVR exposure. Moving further, tertiary photoprotection utilizes UV-absorbing compounds, such as scytonemin and mycosporine-like amino acids (MAAs), creating an extra shield against UVR's relentless onslaught during prolonged exposure. Beyond that, quaternary photoprotection engages DNA repair mechanisms through photolyases (PLs), which, activated by blue light, mend DNA lesions caused by UVR. The story takes an intriguing twist with cyanobacteria's responses to UVR-induced stress, including programmed cell death (PCD). Caspase-like protease activity and metacaspases (MCAs) suggest a fine balance between cell death and survival. In conclusion, this comprehensive odyssey through cyanobacterial photoprotection mechanisms unveils the adaptability and resilience that cyanobacteria have meticulously honed over millions of years. These ancient microorganisms continue to inspire awe as they thrive in a diverse range of ecosystems, shedding light on the enduring marvels of life's adaptability and ingenuity. “Green Alchemy: Mechanisms of Cyanobacterial Photoprotection in the Spotlight” illuminates the intricate strategies that cyanobacteria have honed over millions of years, offering a glimpse into the resilience and adaptability of these remarkable microorganisms.

Keywords: Photoprotection, Mechanisms, Cyanobacteria & Ultraviolet radiation (UVR)

INTRODUCTION

Cyanobacteria, often regarded as the most primordial among Gram-negative bacterial groups, constitute a diverse collective of photosynthetic prokaryotes that engage in oxygen-evolving processes. These resilient microorganisms have colonized a wide spectrum of environments, spanning from scorching hot springs to the frigid reaches of the Arctic and Antarctic regions. Their remarkable endurance has enabled them to thrive throughout Earth's ecosystems, dating back to their emergence during the ancient Precambrian era, approximately 2.8 to 3.5 billion years ago. In doing so, cyanobacteria laid the groundwork for the eventual evolution of contemporary aerobic life forms. These diminutive life forms, often colloquially referred to as “blue-green algae,” occupy a pivotal niche within both

aquatic and terrestrial ecosystems [1]. They serve as the primary architects of biomass, driving the productivity of ecosystems by harnessing the boundless energy of the sun through the intricate process of photosynthesis. Beyond this, their unique ability to fix atmospheric nitrogen endows them with an indispensable ecological role. In tropical regions where rice cultivation is vital, cyanobacteria serve as nature's own biofertilizers, enriching rice fields with essential nutrients and thus enhancing agricultural yields. Cyanobacteria's role in the global nutrient cycle is undeniably significant. Annually, they contribute substantially by fixing over 35 million tons of atmospheric nitrogen. Their ecological importance, however, extends beyond this realm, as they serve as prolific producers of natural compounds with profound medicinal and industrial applications, underscoring their multifaceted significance. Yet, within the intricate tapestry of existence, cyanobacteria, like all life forms, confront challenges [2]. Foremost among these is the relentless barrage of ultraviolet radiation (UVR). Solar UVR encompasses UV-A (315–400 nm), UV-C (100–280 nm) and UV-B (280–315 nm) constituting a segment of electromagnetic radiation emanating from the sun. Recent decades have borne witness to the depletion of the protective ozone layer, driven by anthropogenic emissions of compounds such as chlorofluorocarbons (CFCs) and volatile organic compounds (VOCs), intensifying the influx of solar UVR onto the Earth's surface. UVR, particularly the pernicious UV-B, exacts a toll on biological systems, either directly, through damage inflicted upon cellular DNA and proteins, or indirectly, by inciting the production of reactive oxygen species (ROS). Cyanobacteria, as pioneers of oxygenic photosynthesis, are not immune to these effects. Elevated levels of photosynthetically active radiation (PAR) and UVR can initiate photosensitization reactions within cyanobacterial cells, impacting a multitude of physiological and biological processes, including photosynthesis, development growth, orientation, pigmentation, enzyme activity, nitrogen fixation and carbon assimilation. DNA, the repository of genetic information, stands as a prime target of UVR in cyanobacteria. UV-B radiation triggers the formation of photoproducts, such as cyclobutane pyrimidine dimers (CPDs) and pyrimidine-pyrimidone (6-4) photoproducts (6-4PP), between adjacent pyrimidine residues, compromising the integrity of the genetic code [3]. In response to this ceaseless cosmic challenge, cyanobacteria have meticulously crafted an array of defense mechanisms across sun-drenched habitats characterized by intense PAR and UVR. These strategies involve the synthesis and accumulation of a myriad of diminutive biomolecules, meticulously orchestrated to mitigate the deleterious effects of UVR. In the spotlight of "Green Alchemy: Mechanisms of Cyanobacterial Photoprotection," we embark on a journey into the intricate world of these defenses, elegantly adopted by cyanobacteria. These mechanisms serve as quintessential examples of nature's ingenious solutions to the rigors of environmental adversity, providing profound insights into the adaptability and indomitable spirit of these extraordinary microorganisms.

Surviving the Spotlight: Cyanobacterial Strategies Against High Light Intensity and UVR

Cyanobacteria employ a multitude of adaptive strategies to contend with the intense photosynthetically active radiation (PAR) and ultraviolet radiation (UVR) that affect diverse cellular targets. These strategies encompass both indirect and direct means, collectively forming a multifaceted defense system designed to combat photodamage effectively.

Primary Shielding Mechanism: Avoidance

Cyanobacteria exposed to intense photosynthetically active radiation (PAR) and ultraviolet radiation (UVR) in their

habitats employ shielding as their primary mechanism. This shielding strategy entails various adaptive behaviors, including migrating from regions with high PAR/UVR to those with lower levels or descending to greater depths. Additionally, they form complex mats consisting of diverse cyanobacterial species or filaments enclosed within amorphous silica matrices. These cyanobacteria can also modify their morphology to facilitate self-shading while simultaneously producing extracellular polysaccharides that act as protective shields [4]. In the distinctive environment of hypersaline ponds, specific cyanobacteria such as Oscillatoria cf. laetevirens and Spirulina cf. subsalsa exhibit vertical gliding motility patterns. This downward migration functions as a shield, effectively protecting the photosynthetic apparatus of these cyanobacteria from the harmful effects of UVR. It's important to note that the success of this method depends on the motility capabilities of cyanobacteria, their propensity to form mats, and environmental factors such as water column depth and turbulence. By employing shielding behaviors to avoid detrimental UVR exposure, cyanobacteria enhance their ability to thrive in challenging surroundings.

Energy Dissipation- Nonphotochemical Quenching

Cyanobacteria have evolved protective mechanisms such as Non-Photochemical Quenching (NPQ) to safeguard themselves. NPQ is a crucial defense mechanism cyanobacteria have developed to counter the harmful effects of high-intensity photosynthetically active radiation (PAR) and ultraviolet radiation (UVR). This defense mechanism involves a significant internal transition to the ground state, also known as nonradiative decay. NPQ effectively quenches the singlet excited state of chlorophyll (Chl) and releases the excess excitation energy as harmless heat through molecular vibrations. It's worth noting that NPQ is a common feature in the photosynthetic processes of various eukaryotic organisms, aiding in the protection and regulation of photosynthesis, particularly in challenging environments where the rate of light absorption exceeds the rate of photosynthesis [5]. When light is absorbed, a Chl molecule undergoes a transition from the ground state to its first singlet excited state as part of the photosynthetic process.

Energized molecules in this state can lead to three primary outcomes:

1. The energy can be transferred to another chlorophyll molecule through a process called “Förster resonance energy transfer” (FRET). This energy is subsequently progressively transferred to the photosystem reaction centers PSI and PSII, where it is utilized for photosynthesis. This particular pathway is referred to as "photochemical quenching."
2. Alternatively, the molecule in the excited state may return to its ground state, releasing the absorbed energy as heat.

These outcomes represent the ways in which energized molecules in this state are managed within the photosynthetic process.

To distinguish between the fluorescence resulting from chlorophyll quenching and photochemical quenching, which is a critical step in quantifying Non-Photochemical Quenching (NPQ), a potent light pulse is applied briefly to saturate photochemical quenching and remove its contribution to observed quenching. It's important to note that short-pulse light has minimal impact on NPQ itself [6].

In environments with high-intensity photosynthetically active radiation (PAR), the photosystem reaction centers (RCs) are susceptible to damage through photooxidation, posing a potentially lethal threat to organisms reliant on photosynthesis. Despite such high-intensity PAR conditions, energy dissipation mechanisms like NPQ play a vital role in reducing the energy reaching the photosynthetic RCs, thereby shielding them from light-induced harm. Conversely, in low-light conditions, the

photosynthetic antenna operates at peak efficiency, allowing nearly all absorbed energy to be efficiently delivered to the RCs to drive the photochemical reactions. Elevated irradiance levels significantly enhance the efficiency of light-harvesting complexes as energy dissipators, resulting in an increase in thermal energy wastage within the antenna [7]. As thermal energy dissipation intensifies and fluorescence emission associated with PSII diminishes, the surplus absorbed energy directed toward the reaction centers (RCs) diminishes as well. This intricate process, known as the photochemical quenching process, involves the membrane chlorophyll-containing light-harvesting complex (LHC) of PSII and is a characteristic feature of algae and plants. In conditions of saturating light, it is activated by the acidification of the thylakoid interior. Cyanobacteria, on the other hand, employ soluble supplementary membrane antenna complexes comprised of phycobiliproteins to fulfill a similar role. The OCP-dependent Non-Photochemical Quenching (NPQ) mechanism was elucidated through experimentation with Synechocystis sp. PCC 6803, where the process of NPQ and the role of phycobiliproteins (PBs) were demonstrated using intense blue-green light. In cyanobacteria, NPQ unfolds via the photoactivation of a soluble carotenoid protein known as the "orange carotenoid protein (OCP)," in conjunction with the soluble PBs. Subsequent research, conducted using a Synechocystis mutant devoid of PSII, unveiled that carotenoids activated by blue-green light contribute significantly to this mechanism. Moreover, findings indicated the pivotal involvement of the OCP in NPQ regulation within cyanobacteria [8].

Fluorescence Recovery Protein

To fully restore fluorescence quenching and unleash the antenna's maximum potential after OCPr destabilization in vivo, a pivotal player emerges: the Fluorescence Recovery Protein (FRP). In the absence of this specific protein, quenched Synechocystis cells struggle to regain lost fluorescence when light intensity decreases. FRP, a 13 kDa protein, exclusively exists in species featuring OCP, and notably, it lacks chromophores [9]. Both in vitro and in vivo settings benefit from FRP's presence as it accelerates the conversion of free OCPr back to OCPo. Its critical role involves aiding the dissociation of OCPr from the phycobilisomes (PBs), a process essential for the antenna's complete recovery and the restoration of its full capacity.

Cyanobacterial mutants lacking FRP exhibited an inability to recover their typical antenna capacity under low light conditions. Among the 130 accessible cyanobacterial genomes, 97 contain the gene for OCP, while 71 contain the gene for FRP. It's worth noting that Synechocystis sp. PCC 6803's FRP stands apart from those in most other strains due to an additional 25 amino acids. This extended form arises from an initial misidentification of the start site, whereas the active form in vivo is a shorter protein commencing with Met26. Notably, the gene slr1963, responsible for encoding OCP in Synechocystis, also encodes FRP, a feature found in nearly all cyanobacterial strains. As previously mentioned, the interaction between OCPr and FRP accelerates the conversion of OCPr into OCPo. Consequently, this interaction disables the OCP-dependent Non-Photochemical Quenching (NPQ) mechanism. Based on the findings outlined earlier, the key proteins involved in the OCP-dependent NPQ process are PBs, FRP, and OCP. Specifically, OCPr is the only form capable of interacting with PB, and this binding stabilizes OCPr even in the absence of light [10]. Furthermore, OCP has the ability to attach itself to APC660 trimers while simultaneously quenching their fluorescence by binding to the core of the PB. Remarkably, a single OCP molecule can completely quench the fluorescence originating from a single PB. To facilitate the disengagement of OCPr from its PBs, FRP plays a vital role in this process.

Orange Carotenoid Protein

OCP (Orange Carotenoid Protein) has been documented in the vast majority of cyanobacteria that possess phycobilisomes (PBs). To gain a deeper understanding of OCP's function, researchers initially characterized and subsequently isolated and crystallized OCP from Arthrospira maximum. OCP is a protein with a molecular weight of 35 kD, comprising the ketocarotenoid 39-hydroxyechinenone [11]. It consists of two distinct domains: a α/β C-terminal domain and an all α-helical N-terminal domain. While the C-terminal region shares homology with the nuclear transport factor 2 superfamily, the N-terminal domain of OCP exhibits a structure and sequence unique to cyanobacteria. Notably, the carotenoid 39-hydroxyechinenone spans both protein domains and is largely embedded within them. The gene responsible for encoding OCP in Synechocystis is slr1963, and it is produced constitutively, even in the absence of PBs in mutant Synechocystis strains. The significance of OCP (Orange Carotenoid Protein) in photoprotection came to light through studies involving mutants of OCP and PB in Synechocystis. Subsequently, it was established that OCP functions as a photoactive protein. When exposed to blue-green light, the protein undergoes conformational changes, as does the associated carotenoid, transitioning from its stable orange-dark state (referred to as OCPo) to a meta-stable red active form (referred to as OCPr). Importantly, the presence of a ketocarotenoid is essential for this photoactivity. Zeaxanthin, lacking the carbonyl group found in ketocarotenoids, can bind to OCP, but the resulting zea-OCP complex is found to be photo-inactive and retains its yellow color even under intense light conditions [12]. In cyanobacteria exposed to high irradiation, OCPr, the active red form of the Orange Carotenoid Protein (OCP), plays a pivotal role in reducing phycobilisome (PB) fluorescence and limiting energy delivery to the reaction centers (RCs) through enhanced thermal energy dissipation. In the absence of light, OCPr naturally transitions back to its dark and stable form, OCPo. The rate of this transformation accelerates with rising temperatures, facilitating a faster return to the OCPo state. Conversely, the conversion of OCPo to OCPr in response to light remains unaffected by temperature variations, resulting in a decline in the steady-state OCPr levels as illumination temperature increases. Notably, the kinetics of fluorescence recovery in vivo exhibit significant temperature dependence, with recovery rates slower than the dark conversion of OCPr to OCPo. This suggests that the in vivo stability of the OCPr form surpasses its in vitro counterpart under varying temperature conditions.

High Light Inducible Protein

Cyanobacteria primarily utilize photoprotective bodies (PBs) to harness light energy, while also possessing an array of high-light-induced proteins (Hlips), believed to be the precursors of the entire Light-Harvesting Complex (LHC) superfamily [13] Hlips are diminutive single-helix proteins, boasting a molecular weight of approximately 7 kDa, bearing resemblance to both the first and third helices of plant LHC antennas. They also feature a distinctive (Chl-a)-binding motif that remains conserved across the entire LHC superfamily. These genes encoding Hlips are widely distributed in cyanobacterial genomes and exhibit substantial expression under various stressful conditions. However, their precise cellular role remains elusive. Hlips contribute to photoprotective functions in several processes, including Chl-a recycling, PSII assembly, and Chl-binding protein synthesis [14].

In the species Synechocystis sp. PCC 6083, there are two Hli proteins, namely HliC and HliD, which play a crucial role in the formation of the functional core of Photosystem II (PSII). These Hlips are integral components of a larger protein complex referred to as RC*, which also includes Ycf39, a member of the short-chain

reductase/dehydrogenase superfamily, and the PSII assembly intermediate RCa. In native electrophoresis experiments, it was observed that both the Hlips-Ycf39 subcomplex and HliD by itself exhibited partial separation from the RC* complex, manifesting as distinct colored bands. Upon further investigation and purification of the Ycf39-HliD subcomplex, it was revealed that Chl-a molecules are linked to HliD alongside β-carotene [15]. This linkage enables direct energy transfer from the Chl-a Qy state to the β-carotene S1 state, resulting in the dissipation of absorbed energy. The process of carotenoid-induced quenching of Chlorophyll-a (Chl-a) through energy transfer is a feature of the Light-Harvesting Complex (LHC) superfamily. It is theorized that the mechanism of Non-Photochemical Quenching (NPQ) based on LHCs initially evolved in cyanobacteria and was subsequently adapted and modified by algae and plants as a means to regulate their light-harvesting activities.

Flavodiiron Proteins

Flavodiiron proteins (FDPs), also known as A-type flavoproteins or Flvs, play a crucial role in safeguarding the photosynthetic system of cyanobacteria against photodamage [16]. Structurally, FDPs can adopt homotetrameric or homodimeric forms, featuring a core composed of a metallo-lactamase-like domain, along with a C-terminal flavodoxin domain. Remarkably, FDPs found in cyanobacteria and several other photosynthetic eukaryotes are equipped with an additional C-terminal flavin reductase domain. This unique domain facilitates the simultaneous occurrence of O2 reduction and NAD(P)H oxidation within the same enzyme, highlighting the multifunctional nature of FDPs in the photosynthetic process.

In Synechocystis sp. PCC 6803, four important Flavodiiron proteins (FDPs) encoded by the genes sll1521 (flv1), sll0219 (flv2), sll0550 (flv3), and sll0217 (flv4) are notable players in the cyanobacterial photosynthetic system. Following Photosystem I (PSI), Flv3 and Flv1 take on the role of electron carriers, efficiently shuttling electrons and ultimately reducing molecular oxygen (O2) to water (H2O) within the living organism [17]. This electron transfer by Flv3 and Flv1 serves as a critical electron sink, safeguarding PSI against oxidative damage and adapting to varying light conditions. In a complementary role, Flv2 and Flv4, which may be specific to cyanobacteria, contribute significantly to the photoprotection of Photosystem II (PSII). Additionally, within this operon, a small protein encoded by Sll0218 collaborates with Flv4 and Flv2, collectively enhancing cyanobacterial photodefense mechanisms. Under conditions characterized by low CO2 (LC) and intense photosynthetically active radiation (PAR), the flv4-2 operon experiences robust activation. Within this context, the Flv4/Flv2 heterodimer, which binds to the thylakoid membrane in response to light, plays a pivotal role. It facilitates the opening of a unique electron transfer channel, accomplishing this by stabilizing both PSII dimers and Sll0218. For the effective transmission of energy from phycobilisomes (PBs) to PSII, the presence of Flv4/Flv2 complexes proves indispensable. This necessity arises due to the existence of uncoupled PB terminal emitters. According to the findings of this study, flv4-2 operon excision mutants exhibit an altered pattern of energy transfer, with energy distribution skewed from the PB terminating emitters toward the PSII reaction centers (RCs).

While the precise mechanism driving photoprotection induced by the flv4-2 operon remains elusive, an overexpression study has shed light on the role of Flv4/Flv2. It demonstrates that these proteins serve as crucial electron sinks at the acceptor side of Photosystem II (PSII) [18]. This action enables the maintenance of the oxidized state of the plastoquinone (PQ) pool while concurrently reducing the generation of singlet oxygen (O2) within PSII. Furthermore, it is worth noting that the effectiveness of the Flv4/Flv2-related

pathway relies on the proper functioning of regular phycobilisomes (PBs).

Secondary Shielding Mechanism: Nonenzymatic and Enzymatic Antioxidants

Upon entry into the cell, ultraviolet radiation (UVR) initiates reactions with oxygen (O2) and various organic compounds, resulting in the formation of detrimental reactive oxygen species (ROS) such as the hydroxyl radical (OH), superoxide (O2-), or hydrogen peroxide (H2O2). These ROS subsequently trigger oxidative stress. As a secondary protective measure against oxidative damage induced by UVR, cyanobacteria have developed antioxidant systems that encompass both enzymatic and nonenzymatic components.

Nonenzymatic Antioxidants

Alpha-tocopherol (vitamin E), ascorbate (vitamin C), carotenoids, as well as reduced glutathione ascorbate (vitamin C) constitute the main nonenzymatic antioxidants.

Alpha-tocopherol (vitamin E)

Alpha-tocopherol (vitamin E), a lipid-soluble chemical compound, is exclusively produced by oxygenic phototrophic organisms, among which cyanobacteria are notable examples. This compound can undergo oxidation by reactive oxygen species (ROS), resulting in the formation of tocopheryl radicals. These tocopheryl radicals, in turn, can oxidize singlet oxygen into hydroperoxide compounds. Interestingly, ascorbate, which participates in the recycling of tocopherol, has the ability to counteract both of these reactions due to its specific chemical composition [19]. Notably, the unique chemical compositions of ascorbate and tocopherol play a crucial role in these protective mechanisms. Remarkably, in experiments involving tocopherol-deficient mutants of Synechocystis PCC 6803, it was observed that these mutants can withstand both high-intensity photosynthetically active radiation (PAR) and the accumulation of singlet oxygen (1O2). This suggests that tocopherols not only shield photosystems from reactive oxygen species but also play additional roles in cellular defense mechanisms.

Carotenoids

Carotenoids play a crucial role in shielding cells from photooxidative harm by absorbing energy in the triplet state from chlorophyll and extinguishing singlet-state oxygen. On the other hand, alpha-tocopherol protects against lipid peroxidation (LPO) by scavenging reactive oxygen species (ROS). Cyanobacteria possess a diverse array of carotenoids, including myxoxanthophyll, beta-carotene, and its derivatives such as echinenone and zeaxanthin. These pigments, namely myxoxanthophyll, beta-carotene, echinenone, and zeaxanthin, effectively dissipate energy originating from photosensitized chlorophyll or singlet oxygen. Extensive research has demonstrated their remarkable antioxidative properties. Zeaxanthin, in particular, plays a crucial role in photoacclimation when cyanobacteria are subjected to UV-B stress, as exemplified in Synechococcus PCC 7942. Additionally, it was observed that a mutant strain of Synechocystis PCC 6803 with reduced zeaxanthin production exhibited heightened sensitivity to high-intensity photosynthetically active radiation (PAR) coupled with oxidative stress, in contrast to the wild type [20]. A study examined the adaptive responses of four freshwater cyanobacteria to high-intensity photosynthetically active radiation (PAR). The findings revealed that each cyanobacterial species exhibited a distinct carotenoid response under stress conditions. Specifically, it was observed that in response to UV-B irradiation, Nostoc commune displayed variations in its carotenoid pattern, with echinenone and myxoxanthophyll identified as UV-B photoprotectors that became associated with the outer membrane. In addition to its role in UV protection, glutathione, through the glutathione-ascorbate cycle, plays a critical role in several aspects of cellular defense. It

participates in the regeneration of alpha-tocopherol and ascorbate and provides protection to thiol groups within a variety of enzymes. By introducing exogenous antioxidants such as N-acetyl cysteine (NAC) and ascorbic acid, Anabaena sp. exhibited enhanced survival rates under oxidative damage induced by ultraviolet radiation (UVR) [21]. This improvement was evident in the reduction of chlorophyll bleaching, preservation of the photosynthetic machinery, prevention of lipid peroxidation (LPO), and reduction in DNA strand breaks caused by UVR-induced oxidative stress.

Ascorbate (vitamin C)

Ascorbate, in conjunction with ascorbate peroxidase (APX), plays a pivotal role in defense against hydrogen peroxide (H2O2). APX effectively reduces H2O2 to water (H2O) using two molecules of ascorbate. Furthermore, ascorbate interacts with various reactive oxygen species (ROS) such as singlet oxygen, hydroxyl radicals, and peroxyl radicals. Additionally, it fulfills multiple functions, including the regeneration of alpha-tocopherol, direct quenching of ROS, and acting as a substrate for both violaxanthin de-epoxidase and APX processes [22]. When ascorbate reacts with ROS, it leads to the generation of malondialdehyde (MDA). To restore the balance, the enzyme monodehydroascorbate reductase utilizes electrons from ferredoxin or nicotinamide adenine dinucleotide phosphate hydrogen (NADPH) to convert MDA back into ascorbate. If MDA is not promptly reduced, it tends to disproportionately yield ascorbate and dehydroascorbate (DHA).

Enzymatic Antioxidants

To counter the detrimental effects of reactive oxygen species (ROS) induced by UV-B radiation, cyanobacteria activate a repertoire of antioxidative enzymes, including superoxide dismutase (SOD), glutathione peroxidase (GPX), ascorbate-glutathione cycle enzymes (APX), monodehydroascorbate reductase (MDHAR), dehydroascorbate reductase (DHAR), catalase (CAT), and glutathione reductase (GR) [23]. These enzymatic defenders work collectively to alleviate oxidative stress, which is particularly important for cyanobacteria's survival and adaptability in environments where UV-B radiation poses a significant threat [24].

Peroxidases

Peroxidases, the detoxifying harmful H2O2, are present in nearly all life forms. An alternative peroxidase variant called “rubrerythrin-homologue (RbrA),” identified in Anabaena PCC 7120, was found to provide protection to the nitrogenase enzyme against oxidative stress [24].

Catalase

In all aerobic organisms, catalase (CAT) is a vital heme-containing tetrameric enzyme responsible for converting hydrogen peroxide (H2O2) into water and oxygen through the following reaction: 2H2O2 → 2H2O + O2. Furthermore, CAT plays a crucial role in safeguarding cell membranes against oxidative stress induced by dehydration [25], contributing to the complete recovery of cells. By analyzing twenty cyanobacterial genomes, researchers have identified three distinct types of CATs: monofunctional heme-containing CATs, bifunctional heme-containing CAT peroxidases, and nonheme-manganese CATs. Notably, unlike other hydrogen peroxide-scavenging enzymes, CAT operates independently of cellular reducing equivalents. Moreover, the activity of CAT in Prochloron sp. has been observed to correlate directly with the intensity of irradiance [22]. Additionally, Chlorella vulgaris and Anabaena doliolum demonstrated increased CAT activity in response to stressors such as UV-B radiation and copper exposure.

Ascorbate Peroxidase

Ascorbate peroxidase (APX) utilizes the reducing capacity of ascorbate to catalyze the conversion of hydrogen peroxide (H2O2)

into water. In the case of Synechocystis 6803 cells containing APX, they demonstrated the ability to effectively quench chlorophyll (Chl) fluorescence induced by H2O2, while A. nidulans cells lacking APX entirely lacked this capability. During oxidative stress, APX serves as the primary enzyme responsible for eliminating H2O2 in Synechococcus PCC 9742 (R2) cells. Three-dimensional structures of superoxide dismutase (SOD), APX, and glutathione peroxidase (GPX) were investigated. Additionally, when two Nostoc species were exposed to UV-B radiation [26], the enzymatic defense mechanisms involving antioxidants such as catalase (CAT), SOD, and APX were examined.

In response to UV-B exposure, all examined antioxidative enzymes demonstrated a significant increase in activity, with Nostoc sp. strain HKAR-2, isolated from a hot spring, exhibiting more robust antioxidative enzymatic activity compared to Nostoc sp. strain HKAR-6, isolated from a ricefield. Different cyanobacterial species exhibit varying degrees of induction in the levels of antioxidative enzymes during UV-B radiation stress, potentially influencing their survival under challenging conditions [20]. Notably, the Antarctic cyanobacterium Nostoc commune was found to possess the antioxidative enzymes catalase (CAT) and superoxide dismutase (SOD), which together contribute to its ability to withstand the environmental challenges in its natural habitat. Moreover, under high-intensity photosynthetically active radiation (PAR) treatment, increased activity of ascorbate peroxidase (APX) and SOD was observed in Nostoc spongiaeforme and Phormidium corium, respectively.

Glutathione Peroxidase

The antioxidant enzyme GPX can effectively transform a range of both inorganic and organic hydroperoxides into their respective hydroxyl analogs by employing glutathione and additional equivalents. This reaction catalyzed by GPX can be represented as follows:

[2RSH + ROOH → RSSR + H2O + ROH]

Under conditions of oxidative stress, there is a notable increase in the transcript levels of gpx1 and gpx2 in Synechocystis. Knockout mutants of these genes exhibit elevated levels of lipid peroxidation (LPO). These findings suggest that both gpx1 and gpx2 play essential roles in the survival of cyanobacteria, even under normal circumstances, as attempts to isolate a double mutant were unsuccessful, emphasizing their indispensability [27]. Notably, in Arabidopsis, the overexpression of Synechocystis GPX2 enhances tolerance to oxidative stress induced by various abiotic stressors, including high-intensity photosynthetically active radiation (PAR). Furthermore, two NADPH-dependent GPX-like proteins from Synechocystis PCC 6803 were characterized and found to be crucial for protecting cell membranes against lipid peroxidation.

Superoxide Dismutase

Superoxide dismutase (SOD) serves as the primary line of defense against nascent oxygen species, playing a crucial role in mitigating the damaging impact of superoxide ions by converting them into hydrogen peroxide (H2O2). In a notable first, Anacystis nidulans (Synechococcus) experienced the protective effects of cyanobacterial SOD against photooxidative damage. Several research studies have consistently demonstrated the protective function of cyanobacterial SOD in response to various stressors [28]. Initially identified as a copper-storing protein, SOD scavenges superoxide radicals by transforming them into H2O2, subsequently converted into water (H2O) and oxygen (O2) through a catalase-peroxide system, as previously elucidated. Additionally, in the case of the dehydrated field cyanobacterium Nostoc commune subjected to oxidative stress induced by repeated cycles of rehydration and desiccation due to exposure to ultraviolet radiation (UVR), the in situ accumulation of active FeSOD effectively reversed the detrimental effects of such stress.

Manganese superoxide dismutase (MnSOD) has been found to safeguard the heterocyst nitrogenase enzyme against inactivation caused by reactive oxygen species (ROS) production under aerobic conditions. There are four types of SODs reported based on the catalytic metals they employ: MnSOD, FeSOD, Cu/ZnSOD, and NiSOD. Comparative genomic analysis has revealed that NiSOD is the sole SOD discovered in primitive cyanobacteria, while higher-order cyanobacteria commonly possess both FeSOD and MnSOD. Conversely, Cu/ZnSOD is infrequently encountered in cyanobacteria [29]. Furthermore, it has been observed that the more advanced middle-order cyanobacterial forms exhibit a combination of Ni and Fe SODs or Fe and Mn SODs, in contrast to simpler unicellular Prochlorococcus species, which exclusively rely on NiSOD. In the most advanced heterocystous, heterotrichous, as well as filamentous cyanobacterial forms, the prevailing types of superoxide dismutase (SOD) are exclusively the Fe and Mn metalloforms.

Tertiary Shielding Mechanism:
Generation of UV-Resistant Compounds:
Cyanobacteria, when exposed to extended periods of harmful ultraviolet radiation (UVR), activate a third-tier defense mechanism by synthesizing UV-absorbing compounds. Among the noteworthy UV-screening chemicals employed by cyanobacteria for photoprotection against the harmful effects of UV-B and UV-A radiation, scytonemin and mycosporine-like amino acids (MAAs) stand out [30]. In the following sections, we will offer a brief overview of the distinctive photoprotective qualities of these UV-screening compounds.

Scytonemin
Scytonemin, primarily produced by the cyanobacterial and algal symbionts found in certain lichens, is a dimeric phenolic pigment that accumulates within the extracellular polysaccharide sheath of specific cyanobacteria. It functions as an inert UV sunscreen, known for its exceptional stability and lipid solubility, and displays a pale yellow to brown hue. In vivo, scytonemin exhibits its highest absorption at 370 nm, while in purified form, it peaks at 386 nm. Additionally, it has significant absorption at 252, 278, and 300 nm, making it a valuable photoprotective agent for organisms residing in UV-A/B-exposed environments [31]. Scytonemin absorption spectra have been identified in various cyanobacterial species, with notable peaks at 252, 300, and 386 nm. Derivatives of scytonemin include dimethoxyscytonemin, tetramethoxyscytonemin, scytonemin, and scytonemin-3a-imine. Scytonemin exists in two distinct states: reduced (Molecular weight: 546 Da) and oxidized (Molecular weight: 544 Da). Researchers have explored several cyanobacterial genomes to identify genes associated with scytonemin biosynthesis. In Nostoc punctiforme ATCC 29133, a gene cluster (NpR1276-NpR1259) comprising 18 unidirectionally transcribed ORFs was discovered. The synthesis of scytonemin relies on tyrosine and tryptophan derivatives as crucial building blocks. The author proposed a potential pathway for scytonemin production, recognizing the acycloin reaction as a pivotal step in assembling the scytonemin carbon structure. In addition to scytonemin and mycosporine-like amino acids (MAAs), various UV-absorbing biomolecules, including pteridines, prenostodione (λmax at 318 nm), and biopterin glucoside (λmax at 362 nm), have been observed in certain cyanobacteria. Microalgae harbor a range of additional compounds, such as polyamines (PAs), that play a role in photoprotection [32]. These PAs encompass common varieties like the triamine spermidine, the diamine putrescine, and the tetramine spermine, all found within the cells. Notably, in cyanobacteria, spermidine stands out as the primary polyamine present.

Mycosporine-Like Amino Acids:
MAAs are synthesized or accumulated by a diverse range of taxonomic groups across

various organisms, including both micro- and macroalgae, numerous aquatic animals, and lichen symbionts. These MAAs are characterized by their small size, hydrophilic nature, and colorless appearance, with a cyclohexenimine or cyclohexenone chromophore conjugated to the nitrogen group of an amino acid or its imino alcohol [33]. Their remarkable ability to absorb strong UV radiation (λ_{max} 310-362 nm) and high molar extinction coefficients (ε) ranging from 28,100 to 50,000 M-1 cm-1 contributes significantly to their UV-screening properties. Various MAAs have been identified and described in cyanobacteria and other eukaryotic microalgae. Furthermore, certain cyanobacteria have been found to produce novel glycosylated MAAs. Cyanobacteria employ the passive defense mechanisms of MAAs to capture photons and prevent their interaction with DNA and proteins. Thanks to MAAs, three out of every ten photons can be effectively intercepted, preventing them from affecting cytoplasmic targets within cyanobacteria [34]. Among various microalgae, especially cyanobacteria, there is a well-established appreciation for the photoprotective role of MAAs. Importantly, MAAs efficiently dissipate absorbed radiation as heat without generating reactive oxygen species (ROS).

Quaternary Shielding Mechanism: Repair and Resynthesis

In cyanobacteria, a fourth line of defense becomes crucial as they face the entry of ultraviolet radiation (UVR) into the cell, causing damage to DNA and proteins. Cyanobacteria possess multiple copies of genomic DNA, which serves as a buffer against the effects of single mutations induced by UV radiation. Furthermore, the presence of numerous DNA repair systems enhances the resilience of cyanobacteria against the potentially lethal consequences of radiation exposure. Among these repair mechanisms are photoreactivation, facilitated by the enzyme "Photolyases (PLs)," which monomerizes UV-induced dimers, as well as recombinational repair and dark or excision repair processes.

Photoreactivation

Photoreactivation, a unique process exclusive to cyanobacteria, relies on harnessing blue light to reverse UV-induced photo adducts, restoring them to their normal state through the action of well-conserved DNA repair enzymes called PLs (Photolyases). These PLs are not limited to cyanobacteria but are found throughout the three domains of life [35]. By utilizing the energy from visible and blue light, PLs perform the remarkable feat of directly monomerizing the cyclobutane ring of pyr<>pyr adducts. They exhibit a selective binding affinity for both CPDs (Cyclobutane Pyrimidine Dimers, CPD PL) and 6-4PPs (6-4 Pyrimidine-Pyrimidone Photoproducts, 6-4 PL), thereby forming a protective shield around the genome, effectively guarding it against the harmful effects of ultraviolet radiation (UVR). These PL enzymes vary in molecular weight, ranging from 50 to 61 kDa, and they exist in a monomeric protein form. Within PLs, two essential cofactors are present: methenyltetrahydrofolate (MTHF) and flavin adenine dinucleotide (FAD) [36]. In certain PLs, an alternative to MTHF takes the form of a second chromophore known as 8-hydroxy-7,8-didemethyl-5-deaza-riboflavin (8-HDF). This photoreactivation process showcases the remarkable adaptability of cyanobacteria in combating the harmful consequences of UV radiation through light-driven DNA repair mechanisms. Methenyltetrahydrofolate (MTHF) or 8-hydroxy-7,8-didemethyl-5-deaza-riboflavin (8-HDF) significantly enhances the DNA repair process, increasing its efficiency by 10-100 times, especially under low-light conditions. On the other hand, flavin adenine dinucleotide (FAD) plays a crucial role in both enzyme catalysis and damage identification. In vitro studies have revealed FAD in various chemical states, including reduced (FADH−), oxidized (FADox), and radical (FADH°) forms, with

FADH− being the active state during catalysis.

Photolyases (PLs) establish a connection with the DNA strand's core through ionic interactions. The PL dimer undergoes a conformational change, flipping into the central cavity of the protein, bringing the flavin cofactor into close proximity to the DNA. This transformation results in the formation of a stable “enzyme-substrate complex,” leading to increased selectivity and efficiency in PL's action. In the catalytic process, a blue light photon triggers the excitation of the second photo-antenna folate (either deazaflavin or MTHF) to a high-energy state, with a lifespan of 2.0 ns (for 8-HDF) or 0.5 ns (for MTHF). This energy is then efficiently transferred from the folate's fluorescence resonance to the flavin cofactor (FADH−) with a quantum yield of 0.8. As an electron moves from flavin to the pyr<>pyr adduct, the cyclobutane ring undergoes cleavage. Ultimately, the active phase of flavin (FADH−) [37], is regenerated by transferring electrons back to FADH°. Notably, several plant species, including Arabidopsis, rice, maize, and wheat, rely on the photoreactivation process mediated by PLs as their primary DNA repair pathway. This mechanism underscores the significance of light-driven DNA repair in diverse biological systems. Research involving Arabidopsis seedlings, alfalfa, and rice has highlighted the significant acceleration of dimer removal by photoreactivation. In the absence of photoreactivating (blue) light, the removal of dimers from bulk DNA appears to proceed at a slower pace, with repairs of 6-4PPs outpacing those of CPDs. However, the precise mechanism by which PLs detect defects in the DNA molecule remains unclear. Following the successful repair of CPD lesions, the PL enzyme persists in a semiquinonid state. An intriguing UV-inducible photoreactivation system has been identified in various strains of Anabaena, including Anabaena M-131, Anabaena PCC 7120, A. variabilis sp. PCC 7118 and Anabaena variabilis PCC 7937. Genes encoding PL homologs have been identified and functionally characterized in Synechocystis sp. PCC 6803, providing insights into their roles in photoreactivation. Additionally, researchers have uncovered that UV-B-induced photosynthetic suppression can undergo photoreactivation in various Anabaena species. Following exposure to UV radiation, a notable surge in the mRNA levels of recA was observed, accompanied by a corresponding increase in the abundance of a 37–38–kDa polypeptide in A. variabilis. Furthermore, investigations have revealed the engagement of the DNA repair enzyme Fpg (formamydopyrimidine-DNA glycosylase) gene, initially discovered in Synechococcus elongatus, in the photorepair of UV-B-induced DNA damage in A. variabilis PCC 7937. Recent studies have also explored the structural aspects of PL genes and their genomic relationships across marine, freshwater, and hot spring cyanobacteria, employing comprehensive analyses of amino acid and nucleotide sequences. Among the three distinct habitats, hot spring cyanobacteria were found to possess PL-expressing genes characterized by the highest guanine-cytosine (GC) content. This genomic GC content influenced both codon usage and the diversity of amino acids within PLs. Interestingly, the third position of the codon was identified as having a more pronounced effect on amino acid diversity in PLs compared to the first or second positions [38].

In addition to DNA repair, cyanobacteria engage in the synthesis of new proteins to replace those that have been damaged. A total of 493 proteins were identified as being influenced by UV-B exposure, revealing a complex and distinct response to UV-B stress. This response comprised an early shock reaction affecting 214 proteins, as well as a subsequent late acclimation response involving 279 proteins. Notably, it was revealed that the UV-B resistance in Synechocystis sp. is attributed to an accelerated turnover of the PSII RC's D1 and D2 proteins. This rapid turnover of PSII RC

proteins in cyanobacteria plays a pivotal role in enhancing UVR resistance and acclimation.

Programmed Cell Death

UV-induced ROS generation can have detrimental effects on cells, including the cleavage of nucleic acids, degradation of proteins, interference with photosynthesis, and peroxidation of lipids, which can inhibit growth and lead to cell death [32]. There is a possibility that cyanobacteria may undergo programmed cell death (PCD) in response to UV-mediated oxidative stress, as ROS has been known to regulate PCD in both plant and animal cells. Studies conducted on various cyanobacteria, including Anabaena sp. exposed to univalent-cation salts, Microcystis aeruginosa treated with H_2O_2, and Trichodesmium sp. experiencing iron deficiency and strong light irradiation, have indicated the occurrence of PCD in these organisms [33]. Findings from research on M. aeruginosa further revealed that H_2O_2 treatment induced PCD in a dose-dependent manner, and that catalase (CAT) reduced caspase activity, suggesting a potential role for PCD in responding to oxidative stress.

In M. aeruginosa cells exposed to oxidative stress, it was determined that caspase activity was responsible for inducing programmed cell death (PCD). Metacaspases (MCAs), cysteine proteinases sharing sequence homology with caspases, play a pivotal role in PCD in various organisms, spanning prokaryotes, plants, and fungi. In the cyanobacterium Trichodesmium IMS101, it was found that caspase-like protease activity triggered PCD. Comprehensive genome analyses of cyanobacteria, including A. variabilis, Synechocystis sp. PCC 6803, Synechococcus sp., and Gloeobacter violaceus, revealed the widespread presence of MCAs in these organisms. The distribution of putative MCAs genes across various cyanobacteria hints at the potential inheritance and transmission of the PCD function. The presence of genetically induced cell death in cyanobacteria implies that these organisms possess intricate regulatory mechanisms governing the balance between cell death and survival [39].

Primary Shielding Mechanisms:
Pigments (Chlorophyll-a, Carotenoids)
Absorb and dissipate excess energy
Protect cellular structures from UVR
Secondary Defense Strategies:
Non-enzymatic Antioxidants (Ascorbate, α-tocopherol, Carotenoids, Glutathione)
Defense against Reactive Oxygen Species (ROS)
Counter UVR-induced damage
Tertiary Photoprotection:
UV-Absorbing Compounds (Scytonemin, MAAs)
Additional shield against UVR
Enhance resilience under prolonged UVR exposure
Quaternary Photoprotection and DNA Repair:
DNA Repair Mechanisms
Photolyases (PLs)
Repair DNA lesions caused by UVR exposure
Programmed Cell Death (PCD):
Responses to UVR-induced stress
Caspase-like Protease Activity
Presence of Metacaspases (MCAs)
Regulatory mechanisms for balancing cell death and survival

Table 1: Guardians of Light: Cyanobacterial Photoprotection Strategies.

CONCLUSION

In the realm of photoprotection, cyanobacteria employ a multifaceted arsenal of mechanisms that have evolved over eons to safeguard their cellular components from the damaging effects of ultraviolet radiation (UVR) and other environmental stressors. The intricate web of photoprotection mechanisms, as elucidated in the spotlight on "Green Alchemy: Mechanisms of Cyanobacterial Photoprotection," showcases the remarkable adaptability and resilience of these ancient photosynthetic organisms. Primary Shielding Mechanisms: At the forefront of cyanobacterial defense against UVR are primary shielding mechanisms, where pigments like chlorophyll-a and carotenoids absorb and dissipate excess energy, thereby shielding vital cellular structures from harm. Secondary Defense Strategies: Cyanobacteria also rely on secondary defense strategies, which include non-enzymatic antioxidants such as ascorbate, α-tocopherol, carotenoids, and reduced glutathione. These molecules act as critical lines of defense against reactive oxygen species (ROS) generated by UVR exposure. Tertiary Photoprotection: The tertiary level of photoprotection introduces UV-absorbing compounds, including mycosporine-like amino acids (MAAs) and scytonemin. These chemicals provide an additional shield against harmful UV radiation, enhancing the resilience of cyanobacteria under prolonged UVR exposure. Quaternary Photoprotection and DNA Repair: As a fourth line of defense, cyanobacteria engage in DNA repair mechanisms to rectify UVR-induced damage. Photolyases (PLs), driven by blue light, play a pivotal role in repairing DNA lesions caused by UVR exposure. Programmed Cell Death (PCD): Cyanobacteria exhibit intriguing responses to UVR-induced stress, including the potential for programmed cell death (PCD). The activation of caspase-like protease activity and the presence of metacaspases (MCAs) suggest that cyanobacteria possess regulatory mechanisms for balancing cell death and survival. This comprehensive overview of cyanobacterial photoprotection mechanisms underscores the elegance of nature's solutions to environmental challenges. These mechanisms not only enable cyanobacteria to thrive in diverse habitats but also provide valuable insights for biotechnological and environmental applications, including the development of novel photoprotection strategies and the study of cellular responses to stress in various organisms. "Green Alchemy: Mechanisms of Cyanobacterial Photoprotection in the Spotlight" illuminates the intricate strategies that cyanobacteria have honed over millions of years, offering a glimpse into the resilience and adaptability of these remarkable microorganisms.

Declaration by Authors

Ethical Approval: Not Required
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Allahverdiyeva Henna; Ermakova, Maria; Bersanini, Luca; Richaud, Pierre; Ajlani, Ghada; Battchikova, Natalia; Cournac, Laurent; Aro, Eva-Mari, Y. M. (2013). Flavodiiron proteins Flv1 and Flv3 enable cyanobacterial growth and photosynthesis under fluctuating light. Proceedings of the National Academy of Sciences of the United States of America, 110(10), 4111–4116. https://doi.org/10.1073/pnas.1221194110
2. Chidgey Markéta; Komenda, Josef; Jackson, Philip J.; Dickman, Mark J.; Canniffe, Daniel P.; Konik, Peter; Pilný, Jan; Hunter, C. Neil; Sobotka, Roman, J. W. . L. (2014). A Cyanobacterial Chlorophyll Synthase-HliD Complex Associates with the Ycf39 Protein and the YidC/Alb3 Insertase. The Plant Cell, 26(3), 1267–1279. https://doi.org/10.1105/tpc.114.124495
3. Ding Nanqin; Li, Jie; Sedmak, Bojan; Song, Lirong, Y. G. (2012). Hydrogen peroxide induces apoptotic-like cell death in Microcystis aeruginosa (Chroococcales, Cyanobacteria) in a dose-dependent manner.

Phycologia, 51(5), 567–575. https://doi.org/10.2216/11-107.1

4. Ehling-Schulz Stefan; Wait, Robin; Görg, Angelika; Scherer, Siegfried, M. S. (2002). The UV-B stimulon of the terrestrial cyanobacterium Nostoc commune comprises early shock proteins and late acclimation proteins. Molecular Microbiology, 46(3), 827–843. https://doi.org/10.1046/j.1365-2958.2002.03209.x
5. Gaber Kazuya; Yamamoto, Takashi; Yabuta, Yukinori; Takeda, Toru; Miyasaka, Hitoshi; Nakano, Yoshihisa; Shigeoka, Shigeru, A. Y. (2006). Glutathione peroxidase-like protein of Synechocystis PCC 6803 confers tolerance to oxidative and environmental stresses in transgenic Arabidopsis. Physiologia Plantarum, 128(2), 251–262. https://doi.org/10.1111/j.1399-3054.2006.00730.x
6. He Donat-P., Y.-Y. H. (2002). Reactive oxygen species and UV-B: effect on cyanobacteria. Photochemical & Photobiological Sciences: Official Journal of the European Photochemistry Association and the European Society for Photobiology, 1(10), 729–736. https://doi.org/10.1039/b110365m
7. Jiang Song; Wu, Qingyu, Q. Q. (2010). Genome-wide comparative analysis of metacaspases in unicellular and filamentous cyanobacteria. BMC Genomics, 11(1), 198. https://doi.org/10.1186/1471-2164-11-198
8. Kannaujiya Rajeshwar P., V. K. . S. (2016). Impacts of diurnal variation of ultraviolet-B and photosynthetically active radiation on phycobiliproteins of the hot-spring cyanobacterium Nostoc sp. strain HKAR-2. Protoplasma, 254(1), 423–433. https://doi.org/10.1007/s00709-016-0964-0
9. Kerfeld Michael R.; Brahmandam, Vishnu; Cascio, Duilio; Ho, Kwok Ki; Trevithick-Sutton, Colleen C.; Krogmann, David W.; Yeates, Todd O., C. A. . S. (2003). The crystal structure of a cyanobacterial water-soluble carotenoid binding protein. Structure (London, England : 1993), 11(1), 55–65. https://doi.org/10.1016/s0969-2126(02)00936-x
10. Knoppová Roman; Tichý, Martin; Yu, Jianfeng; Konik, Peter; Halada, Petr; Nixon, Peter J.; Komenda, Josef, J. S. (2014). Discovery of a Chlorophyll Binding Protein Complex Involved in the Early Steps of Photosystem II Assembly in Synechocystis. The Plant Cell, 26(3), 1200–1212. https://doi.org/10.1105/tpc.114.123919
11. Latifi Marion; Zhang, Cheng-Cai, A. R. (2008). Oxidative stress in cyanobacteria. FEMS Microbiology Reviews, 33(2), 258–278. https://doi.org/10.1111/j.1574-6976.2008.00134.x
12. Maeda Yumiko; Bryant, Donald A.; DellaPenna, Dean, H. A. . S. (2005). Tocopherols protect Synechocystis sp. strain PCC 6803 from lipid peroxidation. Plant Physiology, 138(3), 1422–1435. https://doi.org/10.1104/pp.105.061135
13. Mallick Friedrich Helmuth, N. M. (2000). Reactive oxygen species: response of algal cells. Journal of Plant Physiology, 157(2), 183–193. https://doi.org/10.1016/s0176-1617(00)80189-3
14. Mühlenhoff, U. (2000). The FAPY-DNA glycosylase (Fpg) is required for survival of the cyanobacterium Synechococcus elongatus under high light irradiance. FEMS Microbiology Letters, 187(2), 127–132. https://doi.org/10.1016/s0378-1097(00)00189-0
15. Nazifi Naoki; Asano, Tomoya; Nishiuchi, Takumi; Iwamuro, Yoshiaki; Chinaka, Satoshi; Matsugo, Seiichi; Sakamoto, Toshio, E. W. (2014). Characterization of the chemical diversity of glycosylated mycosporine-like amino acids in the terrestrial cyanobacterium Nostoc commune. Journal of Photochemistry and Photobiology. B, Biology, 142(142), 154–168. https://doi.org/10.1016/j.jphotobiol.2014.12.008
16. Neilson Dion G., J. A. D. D. (2010). Structural and functional diversification of the light-harvesting complexes in photosynthetic eukaryotes. Photosynthesis Research, 106(1), 57–71. https://doi.org/10.1007/s11120-010-9576-2
17. Ning H.-L.; Wang, Lianrong; Song, Yunchun, S. G. (2002). Salt stress induces programmed cell death in prokaryotic organism Anabaena. Journal of Applied Microbiology, 93(1), 15–28. https://doi.org/10.1046/j.1365-2672.2002.01651.x
18. Passardi David; Penel, Claude; Dunand, Christophe, F. L. (2004). The class III peroxidase multigenic family in rice and its evolution in land plants. Phytochemistry, 65(13), 1879–1893.

https://doi.org/10.1016/j.phytochem.2004.06.023
19. Pathak Arun S.; Richa, R.; Rajneesh, R.; Kannaujiya, Vinod K.; Singh, Vidya; Ahmed, Haseen; Sinha, Rajeshwar P., J. S. (2017). Screening and partial purification of photoprotective pigment scytonemin from cyanobacterial crusts dwelling on the historical monuments in and around Varanasi, India. Microbiology Research, 8(1), 4–12. https://doi.org/10.4081/mr.2017.6559
20. Pathak NA; Ahmed, Haseen; Richa, NA; Sinha, Rajeshwar P., J. R. (2017). Metabolomic profiling of cyanobacterial UV-protective compounds. Current Metabolomics, 5(2), 138–163. https://doi.org/10.2174/2213235x04666160829162640
21. Priya Jagadeesan; Dhanalakshmi, Raman T; Seethalakshmi, Thangaraj; Uma, Lakshmanan; Prabaharan, Dharmar; Subramanian, G., B. P. (2007). Comparative analysis of cyanobacterial superoxide dismutases to discriminate canonical forms. BMC Genomics, 8(1), 435. https://doi.org/10.1186/1471-2164-8-435
22. Punginelli Adjélé; Routaboul, Jean-Marc; Kirilovsky, Diana, C. W. (2009). Influence of zeaxanthin and echinenone binding on the activity of the orange carotenoid protein. Biochimica et Biophysica Acta, 1787(4), 280–288. https://doi.org/10.1016/j.bbabio.2009.01.011
23. Rajneesh Shailendra P.; Pathak, Jainendra; Sinha, Rajeshwer P., N. S. (2017). Cyanobacterial factories for the production of green energy and value-added products: An integrated approach for economic viability. Renewable and Sustainable Energy Reviews, 69(NA), 578–595. https://doi.org/10.1016/j.rser.2016.11.110
24. Rakhimberdieva Y.V.; Elanskaya, Irina V.; Karapetyan, Navassard V., M. G. . B. (2007). Protein–protein interactions in carotenoid triggered quenching of phycobilisome fluorescence in Synechocystis sp. PCC 6803. FEBS Letters, 581(13), 2429–2433. https://doi.org/10.1016/j.febslet.2007.04.056
25. Rastogi Aran, R. P. I. (2013). Characterization of UV-screening compounds, mycosporine-like amino acids, and scytonemin in the cyanobacterium Lyngbya sp. CU2555. FEMS Microbiology Ecology, 87(1), 244–256. https://doi.org/10.1111/1574-6941.12220
26. Rastogi NA; Sinha, Rajeshwar P.; Singh, Shailendra P.; Häder, Donat-P., R. P. . R. (2010). Photoprotective compounds from marine organisms. Journal of Industrial Microbiology & Biotechnology, 37(6), 537–558. https://doi.org/10.1007/s10295-010-0718-5
27. Rastogi Rajeshwar P.; Moh, Sang Hyun; Lee, Taek-Kyun; Kottuparambil, Sreejith; Kim, Youn-Jung; Rhee, Jae-Sung; Choi, Eun-Mi; Brown, Murray T.; Häder, Donat-Peter; Han, Taejun, R. P. S. (2014). Ultraviolet radiation and cyanobacteria. Journal of Photochemistry and Photobiology. B, Biology, 141(NA), 154–169. https://doi.org/10.1016/j.jphotobiol.2014.09.020
28. Richa Rajeshwar P., N. S. (2015). Sensitivity of two Nostoc species harbouring diverse habitats to ultraviolet-B radiation. Microbiology, 84(3), 398–407. https://doi.org/10.1134/s0026261715030169
29. Sancar, A. (2016). Mechanisms of DNA Repair by Photolyase and Excision Nuclease (Nobel Lecture). Angewandte Chemie (International Ed. in English), 55(30), 8502–8527. https://doi.org/10.1002/anie.201601524
30. Schäfer Agustín; Sandmann, Gerhard, L. V. (2005). Functional in situ evaluation of photosynthesis-protecting carotenoids in mutants of the cyanobacterium Synechocystis PCC6803. Journal of Photochemistry and Photobiology. B, Biology, 78(3), 195–201. https://doi.org/10.1016/j.jphotobiol.2004.11.007
31. Singh Piyoosh Kumar; Sinha, Rajeshwar P.; Tyagi, Madhu B.; Kumar, Ashok, G. B. (2013). Enzymatic and non-enzymatic defense mechanisms against ultraviolet-B radiation in two Anabaena species. Process Biochemistry, 48(5), 796–802. https://doi.org/10.1016/j.procbio.2013.04.022
32. Smirnoff Glen L., N. W. (2000). Ascorbic Acid in Plants: Biosynthesis and Function. Critical Reviews in Plant Sciences, 19(4), 267–290. https://doi.org/10.1080/07352680091139231
33. Trzyna Xavier; Cordingley, John S., W. C. . L. (2006). A type-1 metacaspase from

Acanthamoeba castellanii. Microbiological Research, 163(4), 414–423. https://doi.org/10.1016/j.micres.2006.06.017
34. Tyystjärvi, E. (2008). Photoinhibition of Photosystem II and photodamage of the oxygen evolving manganese cluster. Coordination Chemistry Reviews, 252(3), 361–376. https://doi.org/10.1016/j.ccr.2007.08.021
35. Vaishampayan Rajeshwar P.; Häder, Donat-P.; Dey, T.; Gupta, A.K.; Bhan, U.; Rao, A. L., A. S. (2001). Cyanobacterial biofertilizers in rice agriculture. The Botanical Review, 67(4), 453–516. https://doi.org/10.1007/bf02857893
36. Wilson Ghada; Verbavatz, Jean-Marc; Vass, Imre; Kerfeld, Cheryl A.; Kirilovsky, Diana, A. A. (2006). A Soluble Carotenoid Protein Involved in Phycobilisome-Related Energy Dissipation in Cyanobacteria. The Plant Cell, 18(4), 992–1007. https://doi.org/10.1105/tpc.105.040121
37. Zeeshan Sheo Mohan, M. P. (2009). Differential response of growth, photosynthesis, antioxidant enzymes and lipid peroxidation to UV-B radiation in three cyanobacteria. South African Journal of Botany, 75(3), 466–474. https://doi.org/10.1016/j.sajb.2009.03.003
38. Zhang Marion; Brandt, Anna Maria; Carmel, Dalton; Silén, Henna M.; Vass, Imre; Allahverdiyeva, Yagut; Salminen, Tiina A.; Aro, Eva-Mari, P. E. (2012). Operon flv4-flv2 Provides Cyanobacterial Photosystem II with Flexibility of Electron Transfer. The Plant Cell, 24(5), 1952–1971. https://doi.org/10.1105/tpc.111.094417
39. Zhao Zi; Zhao, Jindong, W. Y. (2007). RbrA, a cyanobacterial rubrerythrin, functions as a FNR-dependent peroxidase in heterocysts in protection of nitrogenase from damage by hydrogen peroxide in Anabaena sp. PCC 7120. Molecular Microbiology, 66(5), 1219–1230. https://doi.org/10.1111/j.1365-2958.2007.05994.x

How to cite this article: Prathima R, Mutthuraj D Kshitija Aherkar, Kanthesh M Basalingappa. Green alchemy: mechanisms of cyanobacterial photoprotection in the spotlight. *Int J Health Sci Res.* 2024; 14(8):347-363. DOI: *https://doi.org/10.52403/ijhsr.20240840*

******