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REVIEW

Turnover of D1 protein encoded by *psbA* gene in higher plants and cyanobacteria sustains photosynthetic efficiency to maintain plant productivity under photoinhibitory irradiance

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Abstract

The photosynthesis and related plant productivity aspects of plants and cyanobacteria depend upon the functioning of photosystem 2 (PS2), associated with D1 and D2 heterodimer reaction centre core proteins. The D1 protein is encoded by *psbA* gene, genetically localized on the plastid genome (cpDNA), contains funtional cofactors of PS2 in association with D2 protein, and also functions for radiant energy transformation through oxidation of water and reduction of plastoquinone. Surprisingly, D1 protein accounts for even less than 1 % of the total thylakoid membrane protein content. In spite of that, its rate of turnover is very much comparable to ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBPCO) large subunit, most abundantly present in green tissue. The normal functioning of PS2 possesses damage-repair cycles of D1 protein. Generally, rate of photodamage does not exceed the rate of repair under optimal growth conditions, therefore, no adverse effect on photosynthetic efficiency is manifest. However, under strong irradiance coupled with elevated temperature, level of photodamage exceeds the rate of repair, resulting in photoinhibition, photodegradation of D1 protein, and lowering photosynthetic efficiency linked with plant productivity eventually. The features of D1 turnover process are reviewed, particularly with respect to molecular mechanisms.

Additional key words: irradiation stress; lincomycin; photosynthesis; protein synthesis and degradation; recovery; *Synechocystis*.

Introduction

Cyanobacteria, green algae, and vascular plants respond to changes in the light environment in which they grow. Various irradiances affect chloroplast development along with its composition, structure, and functioning of the photosynthetic apparatus (Anderson 1986, Melis 1991, Anderson *et al.* 1997, 1998, Barber *et al.* 1997). Primarily, Kok (1956) has reported that excess irradiance, higher than that needed to saturate photosynthesis during plant growth, causes photoinhibition, accompanied by lowering of electron transport activity and oxygen evolution capacity of chloroplasts (Powles 1984, Aro *et al.* 1993, Niyogi 1999). This is followed by impaired CO_2 assimilation that reduces plant growth and productivity under adverse environmental conditions: strong irradiance coupled with elevated temperature (Long *et al.* 1994, Singh and Chaturvedi 1997a,b, Singh *et al.* 1999). Target of such kind of irradiation-dependent damage lies in the D1 reaction centre core protein of PS2 (Kyle *et al.* 1984, Ohad *et al.* 1984, Demeter *et al.* 1987). The D1 protein is a component in the chloroplast with an extraordinary rapid rate of turnover (Eagelsham and Ellis 1974). Pfister *et al.* (1981) confirmed its herbicide (urea and atrazine) binding affinity. The D1 protein is a core part of PS2 reaction centre, eventually isolated and purified from higher plant chloroplasts (Barber *et al.* 1987, Nanba and Satoh 1987). Trebst (1987) proposed a topological model of D1 protein consisting of five trans-membrane helices organized in the similar manner as L-subunit of the bacterial reaction centre. D1 and D2 polypeptides make hetero-dimer, identical to the L and M subunits of the bacterial reaction centre. The D1 protein links plastoquinone Q_B, commonly known as two electron accepting species of the quinone of the PS2.

D1 protein is highly conserved throughout the plant kingdom with an about 80 % amino acid residues identity between cyanobacteria and angiosperms (Jansson and

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Maenpaa 1997). In higher plants and algae, *psbA* gene encoding D1 polypeptide typically exists as a single copy gene, whereas it belongs to a small gene family, representing multiple forms of the *psbA* (*psbAI, psbAII, psbAIII,* and *psbAIV*) in cyanobacteria (Vrba and Curtis 1989, Mohamed *et al.* 1993, Kulkarni and Golden 1994). In the cyanobacterium *Synechocystis* strain PCC6803, the *psbA* gene family contains three members, viz, *psbAI, psbAII,* and *psbAIII* (Jansson *et al.* 1987). However, *psbAI* gets expressed very rarely in *Synechocystis* 6803, while other two forms, *i.e.*, *psbAII* and *psbAIII* share almost 99 % nucleotide identity and they do encode an identical D1 protein product (Ravnikar *et al.* 1989, Metz *et al.* 1990) in a light-regulated manner (Mohamed and Jansson 1989, 1990, 1991). *psbAII* accounts for more than 90 % of the produced *psbA* transcripts (Mohamed *et al.* 1993). The presence of *psbAI* gene in *Synechocystis* is enigmatic and so far no indications are in record about its expression. By using site-directed mutagenesis, which has substituted *psbAI* by *psbAI-K* in *Synechocystis* 6803 (wild type), generated *Synechocystis* 6803 mutant strain *psbAI-K* successfully enabled to activate expression of *psbAI* along with *psbAII* and *psbAIII.* Though, a heterogeneous appearance was often observed for *psbAI* mRNA in *psbAI-K* mutant, may be due to faster degradation or lesser stability than the *psbAII/psbAIII* transcripts. The *psbAI-K* mutant of *Synechocystis* having *psbAI* gene under forced activation has expressed two forms of the D1. A novel D1 protein (D1$^{\text{I}}$) along with naturally occurring D1 are encoded by *psbAII/psbAIII* (Salih and Jansson 1997). The *psbAI* has only 40-45 % similarity with *psbAII* and *psbAIII* (Osiewacz and McIntosh 1987, Ravnikar *et al.* 1989, Mohamed *et al.* 1993). It is yet to be clarified whether the *psbAI* in *Synechocystis* strain PCC6803 plays any specific role under special circumstances or it is merely an evolutionary remnant. Even after analyzing the original strains of *Synechocystis* PCC6803 from Pasteur Cyanobacteria Collection (Paris, France), the *psbAI* gene was found to be inactive under all typical laboratory conditions. This may be due to high divergence in the amino acid sequences of the *psbAI*, compared to *psbAII/ psbA III*, suggesting *psbAI* as a pseudogene.

All forms of the *psbA* gene (*psbAI, psbAII, and psbAIII*) are transcriptionally active in *Synechoccus* PCC 7942 (*Anacystis nidulans* R2) for producing two forms of the D1 protein products, individually capable for supporting photoautotrophic growth under natural conditions in *A. nidulans* (Golden *et al.* 1986, Krupa *et al.* 1990, Campbell *et al.* 1996). The cyanobacterium *Anabaena* sp. PCC 7120 has four sister copies of *psbA* gene: *psbAII, psbAIII, psbAIV* encode identical polypeptides that differ from the *psbAI* product by 21 amino acid residues (Vrba and Curtis 1989). However, regulation of *psbA* multigene family is best understood in *Synechococcus* sp. PCC 7942 (*A. nidulans* R2), having three copies of *psbA* gene encoding two forms of the D1 protein. The form I (product of *psbAI*) of the D1 protein differs by 25 amino acid residues of 360 amino acids from form II (product of both *psbAII/psbAIII*). The D1:1 (form I) predominates under low irradiance but is transiently replaced by D1:2 (form II) upon shifting under high irradiance. The D1:1 has lower photochemical energy capturing efficiency with lesser resistance to photoinhibition, compared to D1:2. Therefore, the higher activity of D1:2 may allow rapid photochemical dissipation of excess energy into the photosynthetic electron transport chain and also confer enhanced D1 turnover with the photoprotection ability during photoinhibition, while D1:1 extends benefits under low irradiance (Golden *et al.* 1986, Campbell *et al.* 1996).

The D1 protein has five helices designated as A, B, C, D, and E (see Fig. 1). The helix A starts from N-terminal and ends by E-helix at the C-terminal of the protein (Trebst 1987). Various evidences suggest that the primary cleavage occurs in the D-E helix during irradiation stress (Greenberg *et al.* 1987, Mattoo *et al.* 1989, Trebst and Depka 1990, Salter *et al.* 1992, Van der Bolt and Vermaas 1992, De Las Rivas *et al.* 1992). Thus, the D-E helix has utmost significance for the PS2 functioning under irradiation stress. This helix also provides binding niche for the two electron-accepting species of the Q_B quinone (Trebst 1991). Occupancy of the Q_B site is linked with photoinhibition sensitivity (Kyle *et al.* 1984, Gong and Ohad 1991). The N-terminal end of the D1 polypeptide contains PEST-like sequences showing an abundance of proline, glutamine, serine, and threonine associated with the many proteins having rapid turnover efficiency (Rogers *et al.* 1986). The PEST-like sequence (225-238 amino acids) having 14 amino acids of the D1 polypeptide does not contain proline (Mattoo *et al.* 1989, Mulo *et al.* 1997). The QEEET motif (241-245 amino acids) is located just adjacent to the PEST-like region consisting of Gln-Glu-Glu-Glu-Thr amino acids, while N-terminal sequencing of an 8-kDa fragment has indicated that the cleavage may occur closer to Arg-238, possibly at Phe-239 (Shipton and Barber 1991). The close proximity of the proposed cleavage site near to the PEST-like region in D1 protein indicates that it may be involved in regulating turnover of the D1 under irradiation stress. In chloroplast genome PEST-like region has been reported only in D1 protein (Greenberg *et al.* 1987), and it is not conserved with the analogous protein from other photosynthetic organisms (Mattoo *et al.* 1989). The deletion of the PEST-like region of D1 polypeptide successfully modified the Q_B-binding pocket in the ΔPEST mutants (226-233 amino acids) of *Synechocystis* PCC 6803, with photoautotrophic growth and irradiance-saturated rates of oxygen evolution similar to wild type. This indicates that PEST sequence is not absolutely needed for D1 turnover *in vivo* although it affects the redox equilibrium between Q_A and Q_B (Nixon *et al.* 1995).

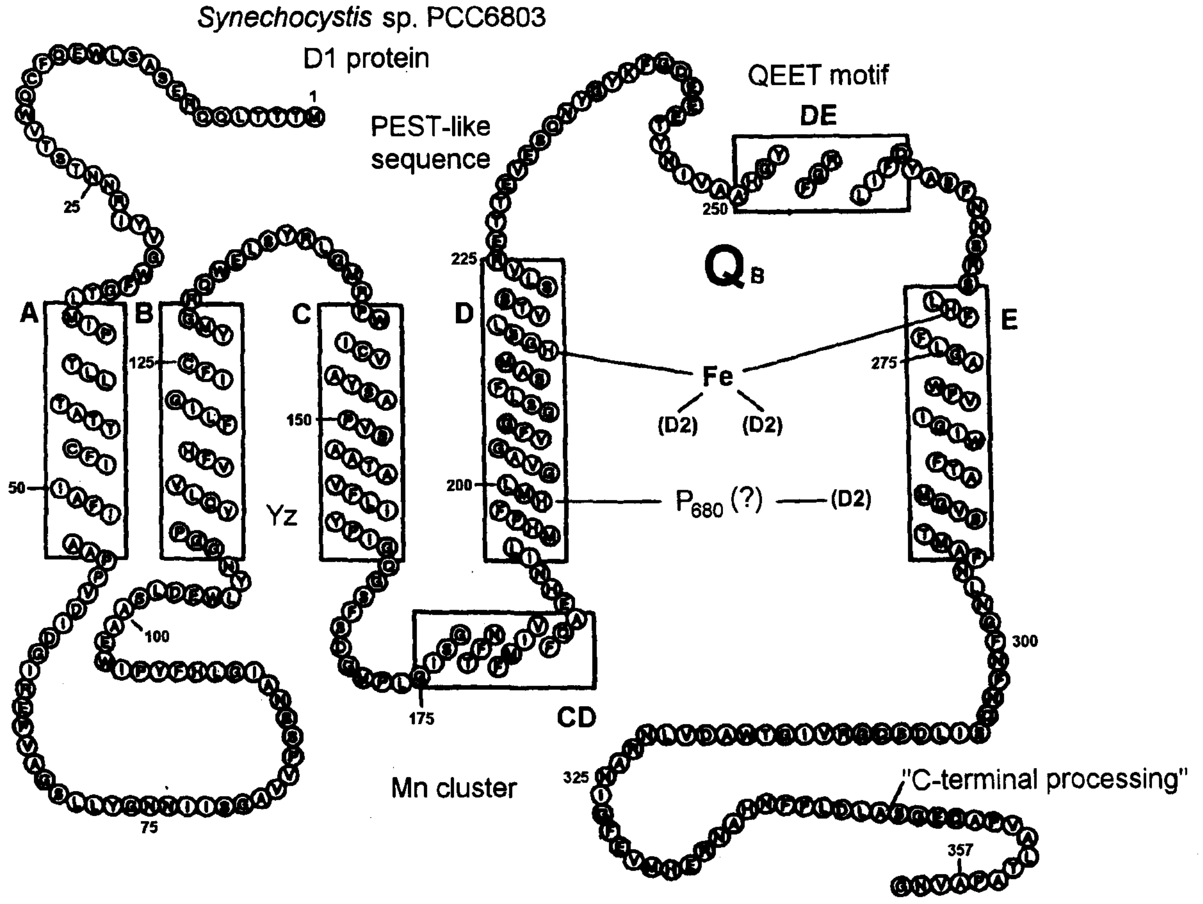

Fig. 1. Schematic diagram of D1 protein encoded by *psbAII* gene in *Synechocystis* PCC 6803, showing architechtural organization of amino acid residues localized in A, B, C, D, and E helices, PEST-like region (225-238 amino acids) and QEEET motif (241-245 amino acids) along with orientation of amino (N) and carboxy (C)-terminals in the thylakoid membrane.

D1 protein synthesis

It occurs on 70S ribosomes bound to thylakoid membranes in all oxygenic photosynthetic organisms (Mattoo and Edelman 1987). Basically, D1 synthesis starts with its precursor form pD1 having 33.5 kDa molecular size, which gets integrated into the non-appressed stroma thylakoids, and further processing takes place for transforming pD1 to mature D1, before being transported into the appressed grana structures of the thylakoids (Mattoo and Edelman 1987). It is translated as a precursor in all organisms having PS2 except *Euglena* (Morden and Golden 1989). The D1 precursor is post-translationally processed by carboxy-terminal processing protease (CtpA) at C-terminus (near 344 amino acid) for generating mD1 form (Anubudurai *et al.* 1994, Yamamoto and Satoh 1998). The processing rate of D1 precursor depends upon chlorophyll (Chl) availability, because half-life of D1 precursor decreases as Chl content of the cell increases. However, D1 processing enzyme (CtpA) is present at the similar level regardless of irradiation and Chl contents, which indicates that directly or indirectly Chl availability promotes D1 translation and efficient processing of the D1 precursor as well (He and Vermaas 1998). The CtpA protein has a significant homology as identified in *E. coli* (Hara *et al.* 1991, Silber *et al.* 1992). The dark-grown plants particularly lack D1 protein (Klein and Mullet 1987), even though polysome-associated *psbA* mRNA is present (Mullet *et al.* 1990). Therefore, in higher plants and cyanobacteria probably no D1 synthesis/re-synthesis takes place if the organism is grown in complete darkness (Aro *et al.* 1993, Singh and Satoh 1999). However, the factor(s) participating in the activation of D1 synthesis are yet to be clarified. The D1 protein accounts even less than 1 % of the total thylakoid membrane protein content. In spite of this, its rate of synthesis/re-synthesis (turnover) is very much comparable with the rbcL, large subunit (LSU) of the RuBPCO in chloroplast (Bottomley *et al.* 1974, Eagelsham and Ellis 1974, Edelman and Reisfeld 1978, Mattoo *et al.* 1984). Being a heart of PS2 core complex it possesses a very high potential for both its synthesis and photo-degradation, in case required for the maintenance under strong irradiance (Prášil *et al.* 1992). This kind of behaviour of D1 protein extends an ability to minimise photodamage of PS2 with faster repair through rapid D1 turnover under photoinhibitory surroundings.

Involvement of D1 in photoinhibition and its recovery

In general, photoinhibition extends photodegradation of the D1, 32 kDa reaction centre core protein of PS2 (Prášil *et al.* 1992, Aro *et al.* 1993, Barber *et al.* 1997, Niyogi 1999) with some exceptions (Cleland *et al.* 1990, Schnettger *et al.* 1994). Anderson *et al.* (1997) have reported that D1 protein turnover in green plants is linearly related to the maximal photosynthesis up to saturating irradiance, but above it, non-functional PS2 centres with intact photodamaged D1 protein get accumulated and D1 protein turnover becomes no longer linearly correlated to the irradiance. However, neither molecular mechanism for the D1 protein turnover *in vivo* nor its physiological significance is yet available satisfactorily (Keren *et al.* 1997, Mulo *et al.* 1998). This has been seriously realised during the last years. Because the D1 turnover is essential for the survival of oxygenic photosynthetic organisms under adverse environmental conditions, viz, excess photosynthetic photon flux density (PPFD) linked with elevated temperature. The D1 protein turnover is associated with repair process of photoinhibited photosynthetic efficiency of PS2 as observed in *Synechocystis* PCC 6803 (Singh and Satoh 1999, see Figs. 2 and 3), because of an inherent susceptibility of the PS2 reaction centre damage caused by excess irradiance. Consequently, prolonged high irradiance exposure enhances the level of photoinhibition that results in reducing *in vivo* photosynthetic efficiency of both higher plants and cyanobacteria (Long *et al.* 1994, Singh and Chaturvedi 1997b, Singh and Satoh 1999).

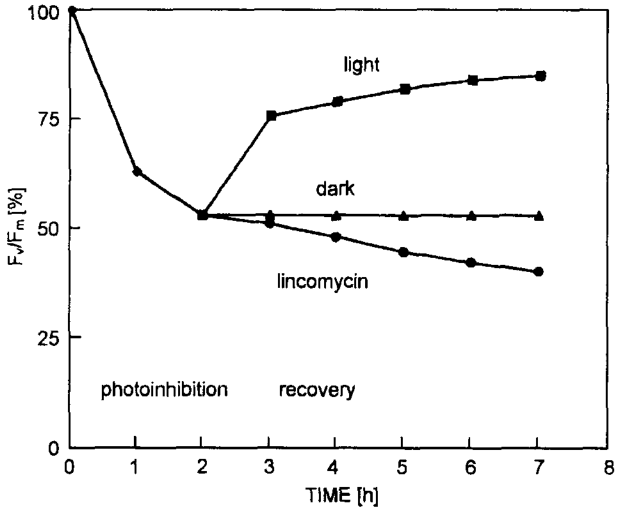

Fig. 2. Photoinhibition of photosystem 2 photosynthetic efficiency (F_v/F_m) in *Synechocystis* PCC 6803 cells. The cells were grown up to early logarithmic growth phase, monitored spectrophotometrically by cell turbidity ($A_{730} \approx 0.6$). The cells were placed under photoinhibitory treatment (500 µmol m^{-2} s^{-1}, 30 °C, 2 h). Subsequently, F_v/F_m was recorded by using photosynthesis efficiency analyser (*PEA, Hansatech*, UK) with 50 % red radiation. Recovery was also monitored either after keeping under growth irradiance (with or without translational inhibitor lincomycin) or darkness. Cells equivalent to 5 g(Chl) m^{-3} were used for all measurements.

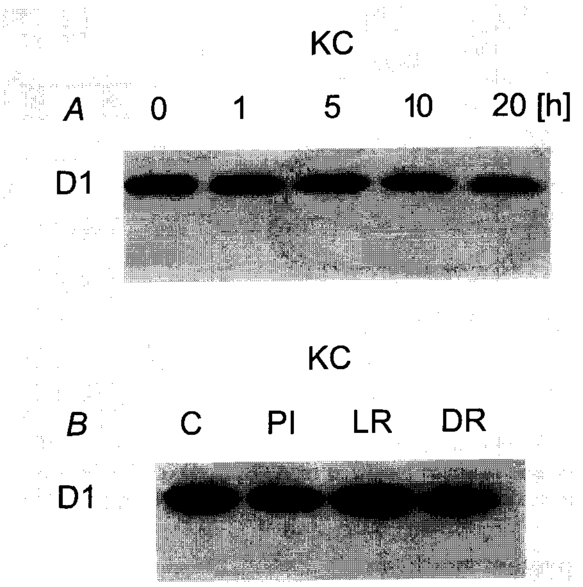

Fig. 3. An immunoblot showing photodegradation and recovery kinetics of *psbAII* encoded thylakoid membrane D1 protein from cyanobacterium *Synechocystis* PCC 6803 wild type strain (KC). *A*: Cells were grown under low irradiance (30 µmol m^{-2} s^{-1}, 30 °C) to the active log phase ($A_{730} \approx 0.6$), and then shifted under photoinhibitory irradiance (500 µmol m^{-2} s^{-1}, 30 °C) for 0, 1, 5, 10, and 20 h. *B*: Lane PI, cells were shifted from growth irradiance (C) to high irradiance (500 µmol m^{-2} s^{-1}, 30 °C) to create *ca.* 50 % photoinhibition. Lanes LR and DR: cells were allowed for recovery either in growth irradiance or in darkness (30 °C) for 5 h. Cells were harvested at each specific step and subsequently used for thylakoid isolation. Thylakoid membranes were fractionated on SDS-PAGE (16 %) containing 6 M urea on chlorophyll basis (2 µg per lane). Resolved polypeptides were transferred onto nitrocellulose membrane and immunologically detected with D1-specific primary antibody followed by anti-rabbit secondary antibody using ECL kit (*Amersham*). The D1 turnover can be compared under different growth conditions.

Surprisingly, photoinhibition even occurs under non-photoinhibitory conditions. Thus D1 degradation in plants occurs not only under high PPFD, but also continues during normal physiological growth conditions (Kyle *et al.* 1984, Ohad *et al.* 1984, Greer *et al.* 1986, Sundby *et al.* 1993, Keren *et al.* 1997). However, supersaturating irradiance (2200 μmol m^{-2} s^{-1}) induces structural and functional modifications in the thylakoid membrane, and also promotes alteration in the composition of PS2 and PS1 as reported in *Dunaliella salina* (green alga). In this alga, enhanced rate of damage entails an accumulation of photodamaged reaction centres (approx. 80 % of the total PS2) and the formation of smaller quantity of PS1 (about 10 % of the cells grown under low irradiance). These modifications permit cells to resume photosynthesis and cell growth under irradiance stress. Furthermore, these changes also contribute in the PS2/PS1 ratio from 1.4 : 1 under low irradiance to 15 : 1 under irradiation stress (Vasilikiotis and Melis 1994). The altered thylakoid membrane organisation ensures that a small fraction of the PS2 remains functional even under continuous irradiation stress which sustains photosynthetic electron flow from H_2O to ferredoxin with the rate sufficient for chloroplast/thylakoid photosynthesis to maintain cell growth (Vasilikiotis and Melis 1994, Singh and Satoh 1999). The D1 degradation may also be promoted under UV (Greenberg *et al.* 1987, Trebst and Depka 1990), visible and far-red radiations of the solar spectrum (Gaba *et al.* 1987). However, under optimal growth surroundings (irradiance and temperature) the rate of photodamage never exceeds the rate of repair (Singh and Chaturvedi 1997a). Upon exposure under strong irradiance coupled with various abiotic and biotic stresses, viz, elevated temperature, drought, salinity, waterlogging, microbial infection, *etc.* may make D1 unable to keep pace parallel with the rate of repair process, resulting in photoinhibition of photosynthesis. The molecular mechanisms controlling photoinhibition and PS2 repair have been studied for more than two decades (Prášil *et al.* 1992, Aro *et al.* 1993, Anderson *et al.* 1997, Barber *et al.* 1997, Niyogi 1999). The photoinhibition and its recovery process are basically dependent upon the level of photodegradation of D1 reaction centre core protein, continuously degraded and re-synthesized in the irradiance-dependent manner (Tyystjärvi and Aro 1996). The continuous excess PPFD exposure also makes D2 reaction centre protein vulnerable to photoinhibition as observed in *Synechocystis* PCC6803 (Singh and Satoh, unpublished).

Recently, Baena-Gonzalez *et al.* (1999) reported that the extent of phosphorylation of PS2 core proteins in differentially light-treated pumpkin leaves is equivalent in dimers and monomers, and directly depends on irradiance. Upon migration of phosphorylated PS2 monomers in stroma-exposed thylakoids, probably following damage of the D1 protein with the dissociation of the light-harvesting complex of PS2, gradual dephosphorylation of monomers in the stroma lamellae allows the reparation of the photodamaged PS2 core complex. The recovery process requires the replacement of damaged D1 protein by newly synthesized D1 (Ohad *et al.* 1984, Mattoo *et al.* 1989). It is regulated by non-photoinhibitory irradiance: even a very weak irradiance (30-60 μmol m^{-2} s^{-1}) may promote/regulate the process of recovery in higher plants and cyanobacteria. Darkness is not proper for re-synthesis of any nascent D1 and therefore limits the recovery process (Singh and Chaturvedi 1997a, Singh and Satoh 1999). The recovery process also does not take place in the presence of translational inhibitors specific for plastid/chloroplast protein synthesis (Singh and Satoh, unpublished). Thus, both catabolism and anabolism of the 32 kDa D1 protein are photoregulated. However, mutants of *Synechocystis* PCC 6803 lacking the *psbO* gene (encodes 33 kDa manganese stabilizing protein associated with water-splitting complex) become more vulnerable to photoinhibition than the wild type. The increased sensitivity to photodamage is accompanied by accelerated turnover of the D1 protein along with rapid rate of recovery by transfer to the non-photoinhibitory conditions (Komenda and Barber 1995). Therefore, the D1 protein turnover may determine photosusceptibility of photosynthesis and restoration of photoinhibited photosynthetic efficiency as well. This may be a kind of natural strategy employed by all photosynthetic organisms to compensate photoinhibitory losses, simply by operating an efficient D1 turnover (removal and replacement) to generate nascent D1 which enables the operating system functional for photosynthetic electron transport linked with CO_2 assimilation. The stepwise photoinhibition as diagnosed by loss in photosynthetic efficiency (F_v/F_m), PS2 oxygen evolving capacity followed by D1 photodegradation (Mulo *et al.* 1998, Singh and Satoh 1999) probably turns in reducing crop yield under field conditions, up to different extent depending upon the growth surroundings and plant genotypes.

Influence of irradiation on accumulation of *psbA* transcript

The PS2 biogenesis displays an intricate pattern of light regulation. Stimulation of *psbA* transcript by irradiation was first demonstrated in maize (Bedbrook *et al.* 1978). In higher plants, *psbA* transcript is genetically chloroplastic and may increase its abundance during chloroplast biogenesis in response to irradiation (Gruissem 1989). The irradiation of etiolated, dark grown (4.5 d-old) barley seedlings rapidly induced the synthesis of the *psbA* gene product, but the transcript level did not increase appreciably, parallel to the irradiance, indicating that the gene is regulated at the level of translation rather than transcription (Klein and Mullet 1987). However, transcripts of this gene are also confirmed under darkness in *Synechocystis* PCC 6803 (Mohamed and Jansson 1989) with the indication that it is not solely regulated by the irradiation at transcription level (Lönneborg *et al.* 1988, Mohamed and Jansson 1989, Smart and McIntosh 1991, Tyystjärvi *et al.* 1996). To clarify this discrepancy, He and Vermaas (1998) did not observe a significant amount of the *psbA* transcript in the wild type cultures of *Synechocystis* 6803, kept in the darkness for 48 h without glucose. The presence of glucose (5 mM) has extended ability for expressing *psbA* transcript even in the darkness, but in lesser quantity as compared to the presence or absence of glucose under irradiation; hence glucose does not favour an enhanced level of *psbA* expression.

Ancestral relationship of *psbA* gene encoding D1 protein

It is generally accepted that chloroplast arose from one or more endosymbiotic events between an ancestral cyanobacterium and eukaryotic organism (Giovannoni *et al.* 1988). The D1 polypeptide of cyanobacteria contains 360 amino acid residues compared to 353 in *Prochlorothrix hollandica* (prochlorophyte) and higher plants, except *Chlamydomonas*, which represents only 352 amino acid residues (Morden and Golden 1989). The prochlorophyte *P. hollandica* and plastid D1 polypeptides sequences share a seven amino acid gap in comparison to cyanobacterial sequences at position six from the C-terminus end of the D1 polypeptide. Thus, phylogenetically these amino acid gaps place *P. hollandica* closer to green plants/chloroplasts than cyanobacteria. In view of this, *P. hollandica* is considered an intermediate of cyanobacteria and chloroplasts of higher plants. *P. hollandica* as prochlorophyte, oxygen-evolving photosynthetic prokaryote contains chlorophylls *a* and *b*, similar to higher plants (Burger-Wiersma *et al.* 1986) but does not contain phycobiliproteins, which is an essentiality for cyanobacteria (Lewin and Withers 1975). The two sister copies of *psbA* gene found in *P. hollandica* encode identical amino acid sequences. The seven-amino acid gap near C-terminus, maintained by *P. hollandica psbA* gene, further favours close relation towards higher plants which contain only a single copy of the *psbA* gene. Therefore, the *psbA* gene might originate with multiple sister copies in cyanobacteria during evolution, kept on minimising its copy number up to the level of two in *P. hollandica*, and eventually resulted in retaining solitary *psbA* gene copy in the higher plants. Bruick and Mayfield (1999) also suggested that chloroplast translational regulation is a hybrid between prokaryotic and eukaryotic systems.

Conclusions

Photoinhibition impairs photosynthetic efficiency (F_v/F_m), oxygen evolving capacity of PS2, carboxylation efficiency, and photobleaching of photosynthetic pigments along with photodegradation of D1 reaction centre core protein. These processes play an important role in reducing plant/crop yield under strong irradiance. The photoinhibition results in the loss of photosynthetic efficiency *in vivo*. Both the D1 catabolism and anabolism are regulated by radiant energy that causes photoinhibition and its recovery depending upon the photon flux density. The D1 turnover is a crucial part of the restoration system because of an inherent vulnerability of PS2 reaction centre damage caused by irradiance stress. The photoinhibition imposes photodegradation of D1 protein, not only in isolation, also incorporates D2 protein with the possibility to reduce size of PS2 reaction centre core complex to prevent over-excitation of the photosynthetic apparatus (Horton *et al.* 1996). Thus, D1 photo-degradation and its re-synthesis (turnover) confer a crucial role in sustaining photosynthetic efficiency linked with plant productivity under adverse environmental variables. Therefore, regulation of D1 turnover, along with an integrated approach to improve radiant energy use efficiency may be helpful to overcome photoinhibitory losses based on photobiotechnology.

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