Action spectrum of *psbA* gene transcription is similar to that of photoinhibition in *Synechocystis* sp. PCC 6803

Taina Tyystjärvi*, Ilona Tuominen, Mirkka Herranen, Eva-Mari Aro, Esa Tyystjärvi

Plant Physiology and Molecular Biology, University of Turku, FIN-20014 Turku, Finland

Received 9 January 2002; revised 18 February 2002; accepted 22 February 2002

First published online 11 March 2002

Edited by Ulf-Ingo Flügge

Abstract The photosystem II (PSII) reaction center protein D1 undergoes rapid light-dependent turnover, which is caused by photoinhibition. To identify the photoreceptor(s) involved in the light-dependent expression of the *psbA* gene encoding the D1 protein, we determined the action spectra of *psbA* transcription, PSII activity, photosynthesis and photoinhibition in *Synechocystis* sp. PCC 6803. In accordance with its phycobilisome antenna, PSII showed the highest activity in the spectral region from yellow to red and only low activity in the ultraviolet-A (UV-A) to green region. Photoinhibition, in turn, was fastest in UV-A to violet light and a minor peak was found in the orange region. The action spectrum of *psbA* transcription resembled closely that of photoinhibition, suggesting that photoinhibition creates a signal for up-regulation of the *psbA* gene.

Key words: Cyanobacterium; D1 protein; Photosystem II; photoinhibition; *psbA* gene

1. Introduction

Cyanobacteria, like plants, perform oxygenic photosynthesis through a series of two photosystems. Photosystem II (PSII) is a membrane-embedded protein complex that transfers electrons across the thylakoid membrane from water to the plastoquinone pool. The D1 and D2 proteins form the reaction center dimer of PSII binding the redox active components. In the light, the D1 protein has a rapid turnover which is a consequence of light intensity-dependent damage to PSII [1]. The repair of the photodamaged PSII center requires the replacement of its D1 protein with a newly synthesized copy [2], and D1 synthesis seems to be a strictly regulated process both in cyanobacteria and in plants.

In cyanobacteria, the D1 protein is encoded by a small *psbA* gene family. The *psbA* gene expression is mainly regulated at the level of transcription, but also transcript stability [3–5] and translation activity [6] affect the synthesis rate of the D1 protein in cyanobacteria. Several regulatory mechanisms have been suggested for *psbA* gene transcription. In the cyanobacterium *Synechococcus* sp. PCC 7942, the exchange between *psbAI* and *psbAII*/*III* transcripts, encoding two different forms of D1 protein, is dependent on light quantity and quality and has been suggested to be mediated either by a blue light receptor [7] or by different redox sensors [8,9]. In *Synechocystis* sp. PCC 6803 the two active *psbA* genes encode identical D1 proteins. Both redox-responsive [10,11] and redox-state-independent [5,12–14] mechanisms have been suggested for the regulation of *psbA* gene transcription in *Synechocystis* sp. PCC 6803.

Although it is well documented that the transcription of *psbA* genes is activated in the light and repressed in the dark, previous studies have not explained how cyanobacterial cells percept the light signal. The possible light receptors include photosynthetic pigments of cyanobacteria: phycobilins, chlorophyll (chl) *a* and carotenoids. Phycobilins absorb yellow to red light [15] in phycobilisomes, the main light harvesting antennae of PSII in cyanobacteria. The second principal photosynthetic pigment in cyanobacteria, chl *a*, has two absorption peaks, one in violet light ($\lambda_{max} = 438$ nm) and another in red light ($\lambda_{max} = 664$ nm). Chl *a* is the major light absorbing pigment of photosystem I (PSI). Carotenoids absorb light at 450–570 nm and function both as accessory pigments in photosynthesis and as photoprotectants [16].

Besides photosynthetic pigments, two bacteriophytochrome systems have been identified in cyanobacteria [17,18]. However, the expression of bacteriophytochrome has been measured only in the dark [18,19], and thus it is unlikely that they are involved in the regulation of *psbA* genes. In addition, two open reading frames in *Synechocystis* sp. PCC 6803 genome show high homology with photolyase and cryptochrome genes. Mutant studies have revealed that the *phrA* gene encodes a functional photolyase while the *phrB* gene product might encode a cryptochrome [20]. Cryptochromes absorb light from ultraviolet (UV) to blue and mediate many regulatory processes in plants [21].

In the present study, we measured the action spectra of *psbA* and *psbD* gene expression in order to identify the possible light receptor. It appears that the principal photosynthetic pigments or known regulatory photoreceptors are not crucial as such, but the action spectrum of *psbA* gene expression is similar to that measured for photoinhibition.

2. Materials and methods

2.1. Strains and growth conditions

The glucose tolerant strain of *Synechocystis* sp. PCC 6803, referred here to as the wild-type, was grown in BG-11 medium at 32°C under continuous illumination of 50 μmol photons m^{-2} s^{-1}. The mutant strain AR (insertionally inactivated *psbA1* and *psbA3* genes and in-

*Corresponding author. Fax: (358)-2-3338075.
E-mail address: taityy@utu.fi (T. Tyystjärvi).

Abbreviations: chl, chlorophyll; PFD, photon flux density; PSI, photosystem I; PSII, photosystem II; UV-A, ultraviolet-A

0014-5793/02/$22.00
PII: S0014-5793(02)02537-1

activated *spe* gene) was grown similarly except that the growth medium was supplemented with appropriate antibiotics [22].

2.2. Light treatments of cell cultures

The cells were harvested at the logarithmic growth phase by centrifugation at 4000×*g* for 5 min and resuspended in fresh BG-11 medium to a final chl concentration of 10 μg chl/ml, wrapped in aluminum foil and dark-incubated for 18 h in order to deplete the cell of *psb* transcripts. The cells were then illuminated at the photon flux density (PFD) of 50 μmol m^{-2} s^{-1} under different wavelengths of light for 15 and 60 min. Cells were illuminated with a slide projector through pairs of long-pass (LL) and short-pass (LS) filters (Corion): violet (400–450 nm), blue (450–500 nm), green (500–550 nm), yellow (550–600 nm), orange (600–650 nm), and red (650–700 nm). The actual spectrum for each filter pair, measured with a calibrated S2000 spectrophotometer (Ocean Optics, Dunedin, FL, USA), is shown in Fig. 1A. For treatment of the cells with UV light (a broad band peaking at 370 nm), light from an Oriel 6258 ozone-free xenon lamp was filtered through a UG-11 filter (Schott).

2.3. Extraction of RNAs and Northern blotting

Total RNA was isolated using the hot phenol method as described previously [6]. The RNAs (4 μg/well) were separated on 1.2% agarose–glyoxal gels and subsequently transferred to Hybond-N membrane (Amersham) according to standard procedures [23]. To verify equal loading of the gels and even transfer of the RNAs in blotting, the membranes were always stained with methylene blue [23]. The whole *psbA2* and *psbD2* genes were used as DNA probes. Prehybridization and hybridizations were performed as described previously [22]. Autoradiograms from three independent experiments were quantified using The Analytical Image Station (Imaging Research Inc., Canada) and action spectra were calculated by subtracting the amount of *psbA* transcript in the dark from the amount of *psbA* transcript after 1 h light treatment.

2.4. Determination of photosynthetic and PSII activity in vivo

The relative quantum yield of photosynthesis was measured with a Clark type oxygen electrode (Hansatech) using 10 mM $NaHCO_3$ as a terminal electron acceptor. The cell suspension (10 μg chl/ml, 1 ml) was illuminated with four low intensities of light defined with the Corion filters as above. The light intensities were adjusted so that the production of oxygen was linearly dependent on light intensity. From these results the apparent relative quantum yield of photosynthesis was calculated for each wavelength region. The relative quantum yield of PSII electron transfer was calculated similarly except that 0.5 mM 2,6-dichloro-*p*-benzoquinone (DCBQ) was used as a electron acceptor. Ferricyanide (0.5 mM) was added to the cell suspension to keep DCBQ in the oxidized form. The relative rates of PSII oxygen evolution and photosynthesis under UV-A illumination were measured by illuminating a 400 μl volume of *Synechocystis* cell suspension (10 μg chl/ml) in an oxygen electrode through a fused silica rod serving both as a light guide and stopper.

2.5. In vivo photoinhibition measurements

The effect of light quality in inducing photoinhibition was studied by illuminating the cells (10 μg chl/ml) at 200 μmol photons m^{-2} s^{-1} of UV-A, violet, blue, green, yellow, orange or red light. Translation inhibitor, lincomycin (500 μg/ml), was added at the beginning of illumination to block the synthesis of the D1 protein and thus to prevent the repair of PSII. Aliquots for oxygen evolution measurements were withdrawn after 1, 2, 3 and 4 h of illumination, except that for the UV-A treatments the aliquots were withdrawn after 15, 30 and 60 min of illumination. Oxygen evolution was measured with an oxygen electrode in saturating light using DCBQ as electron acceptor. The rate constant of photoinhibition (k_{pi}) was calculated from oxygen evolution data by curve fitting as by Tyystjärvi and Aro [1].

3. Results

3.1. Action spectra of expression of PSII reaction center genes in Synechocystis sp. PCC 6803

The effect of light quality on the transcription activities of *psbA* and *psbD* genes encoding the PSII reaction center proteins D1 and D2 was studied by illuminating cells for 15 and 60 min with 50 nm slices of the visible spectrum defined with high- and low-pass filters (Fig. 1A). The PFD was 50 μmol m^{-2} s^{-1}. The *psbD2* (1.3 kb) and *psbDC* (2.5 kb) transcripts, encoding identical D2 proteins, are seen as two separate bands in Northern blots (Fig. 1B). The *psbA2* and *psbA3* genes both produce approximately 1.2 kb transcripts and are therefore seen as a single band in Northern blots (Fig. 1B). The cells were pre-treated in the dark for 18 h in order to minimize the amounts of *psbA*, *psbD2* and *psbDC* mRNAs [14]. The amounts of *psbA* and *psbD2* transcripts were already slightly elevated after a 15 min illumination of the cells under any light quality, but the amount of *psbDC* transcripts remained below the detection limit (Fig. 1B). Illumination for 1 h of the cells under different wavelengths induced the expression of all studied genes. The *psbA* and *psbD2* transcripts were five to six times more abundant after 1 h of illumination under violet light than in the dark (Fig. 1B). Illumination with red or orange light induced transcription of the *psbA* and *psbD2* genes although the induction was only half as efficient as that measured under violet light. Under blue, green and yellow regions, transcription of *psbA* and *psbD2* genes was only slightly activated. The action spectrum of *psbA* transcription, calculated from the results of three independent Northern blot experiments (60 min illumination), is shown in Fig. 2D. In contrast to *psbA* and *psbD2* genes, the *psbDC* operon was most efficiently activated by orange light. Clear activation was also induced by violet, yellow and red light, while only very low activation of *psbDC* transcription was seen under blue and green light (Fig. 1B).

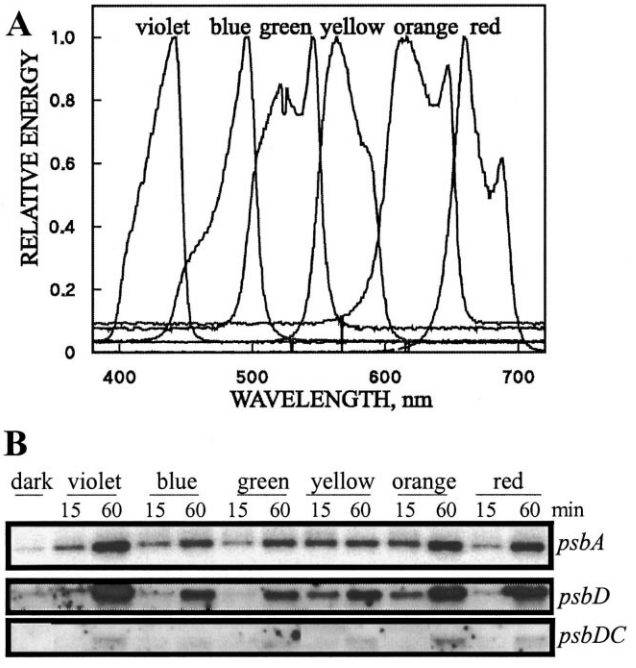

Fig. 1. Amounts of *psbA*, *psbD* and *psbDC* transcripts in *Synechocystis* sp. PCC 6803 after illumination with different wavelengths of light. A: The spectra of the wavelength regions used to illuminate *Synechocystis* cells. The spectra were normalized to the same peak intensity. B: A representative Northern blot showing the amounts of *psbA*, *psbD2* and *psbDC* transcripts after illumination with different wavelength regions for 15 and 60 min.

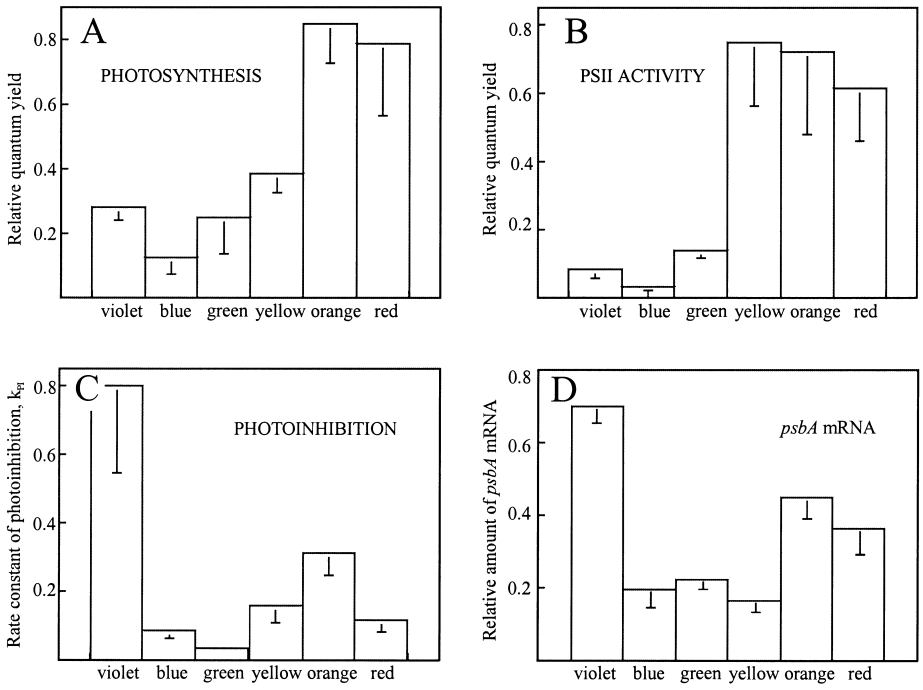

Fig. 2. Action spectra of photosynthetic activity, PSII activity, photoinhibition and *psbA* gene transcription. A: Apparent quantum yield of photosynthesis in vivo. B: Apparent quantum yield of PSII activity. C: The susceptibility of *Synechocystis* cells to photoinhibition under different wavelength regions. D: The amount of *psbA* mRNA after 60 min illumination under different light qualities.

3.2. Action spectra of photosynthetic activity, PSII activity and photoinhibition

The photosynthetic activity of *Synechocystis* cells was measured in vivo in different light qualities and the relative quantum yield of photosynthesis was calculated for each light quality (Fig. 2A). The lowest relative quantum yield of photosynthesis was observed in blue light, and the quantum yield of photosynthesis was two to three times higher under violet, green and yellow light than in blue light. Illumination with orange and red light produced a six times higher relative quantum yield of photosynthesis than was measured under violet light. The relative quantum yield of PSII activity, in turn, was low under violet, blue and green light (Fig. 2B). The highest PSII activity was measured in yellow light where the relative quantum yield of PSII activity was about 20 times higher than in blue light. The cells that were illuminated under orange and red light showed almost as high a quantum yield of PSII activity as those illuminated with yellow light (Fig. 2B). A similar action spectrum of PSII activity was recently obtained using the flash illumination technique [24].

To produce the action spectrum of photoinhibition, we measured the loss of PSII activity, during illumination at specific wavelength regions in the presence of lincomycin, and calculated the rate constant of photoinhibition for each wavelength region (Fig. 2C). Violet light damaged PSII very efficiently while blue, green, yellow and red light were much less efficient (Fig. 2C). Orange light induced fairly fast photoinhibition, but the k_{pi} value was still less than half of that measured in violet light (Fig. 2C).

3.3. UV light efficiently induces photoinhibition and activates transcription of the psbA gene

The action spectra of photoinhibition and *psbA* transcription activity show some similarity with the absorption spectrum of chl *a*. To test if chl *a* actually is the photoreceptor for photoinhibition and *psbA* gene transcription, we followed the inhibition of PSII activity and the induction of *psbA* gene transcription in vivo under UV-A light where chl *a* absorbs less efficiently than in violet and red light. Since the photosynthetic efficiency of UV-A light is very low, *psbA* transcripts may become stabilized in wild-type *Synechocystis* in the same way as in darkness [5]. We measured *psbA* transcripts also in the mutant strain AR in which *psbA* transcript stabilization does not occur at all because of inactivation of the *spe* gene [4]. The AR strain contains only one functional *psbA* gene, *psbA2* [22], that is the mainly expressed *psbA* gene in *Synechocystis* [25].

The UV-A treatment induced twice as many *psbA* transcripts as did the violet light and 10 times more than did the yellow light treatment in both wild-type and AR strains (Fig. 3A), indicating that transcription rather than transcript stability was affected and that the enhancement was on the *psbA2* gene. Furthermore, the enhancement of transcription in UV-A also rules out the involvement of chl *a* as the photoreceptor for *psbA* gene expression. UV-A treatment also induced very rapid photoinhibition: only 35% of PSII activity was left after 15 min illumination under UV-A light in the presence of lincomycin (Fig. 3B). A k_{pi} value of 4 h^{-1} was calculated from the data shown in Fig. 3B, indicating that UV light induced photoinhibition approximately five times as efficiently as violet light (Fig. 2C).

4. Discussion

Our goal was to identify the photoreceptor perceiving the light signal activating the transcription of *psbA* and *psbD* genes in *Synechocystis* sp. PCC 6803. To distinguish between possible light receptors, *Synechocystis* cells were illuminated

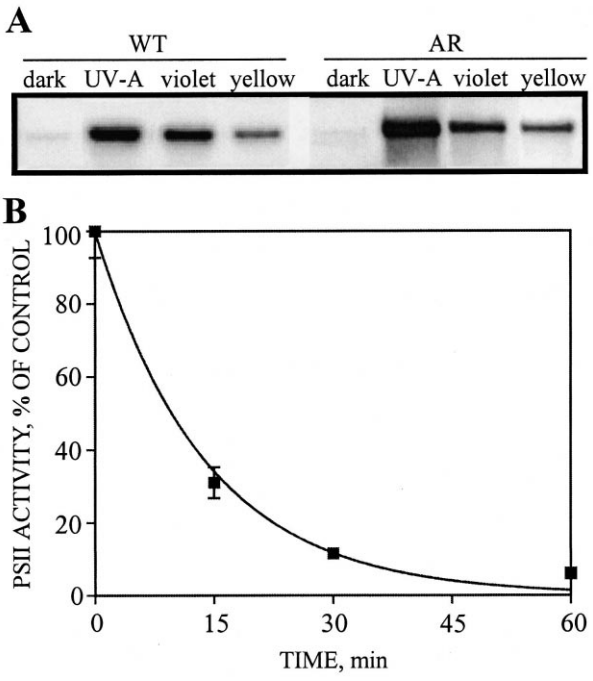

Fig. 3. UV-A treatment induces transcription of *psbA* genes and rapid photoinactivation of PSII activity. A: Wild-type (WT) and AR mutant (contains *psbA2* gene as its only functional *psbA* gene) cells were pre-treated in the dark overnight and thereafter illuminated with UV-A, violet and yellow light for 1 h. B: Wild-type *Synechocystis* cells were illuminated with UV-A light in the presence of translation inhibitor and the PSII activity was measured after 15 min, 30 min and 60 min of illumination. The fitted curve shows the k_{pi} value of 4 h^{-1}.

under different wavelengths of light. The action spectra of *psbA* and *psbD2* transcription were similar: high activity under UV-A and violet region and a minor peak in the orange–red region. These action spectra corroborate an earlier study showing very strong elevation of *psbA* transcripts under UV-B illumination [13]. It has also been shown that more *psbA* transcripts accumulate if *Synechocystis* cells are illuminated with 350–530 nm than with 580–650 nm light [26]. In another cyanobacterium *Synechococcus* sp. PCC 7942, the specific enhancement of *psbAII/III* gene expression under visible light peaks at 450 nm [7,27], which has been suggested to indicate the involvement of a blue light receptor in the regulation of transcription of *psbA* genes. A high transcription activity of *psbA* and *psbD2* genes in UV-A (emission maximum at 370 nm) and violet (400–450 nm) regions in *Synechocystis* cells (Figs. 1 and 3) could be explained by a blue light receptor. However, signal transduction via a blue light receptor cannot explain active transcription of *psbA* and *psbD2* genes in the orange–red light region, the very low transcription activity in blue light (450–500 nm) or the dependence of transcription on light intensity [22].

We next considered the regulatory mechanisms that directly couple the efficiency of PSII or photosynthetic electron transfer to the expression of PSII reaction center genes. In a previous study, Pfannschmidt and coworkers suggested that transcription of the *psbA* gene in chloroplast is controlled via the redox state of the plastoquinone pool [28]. Recently, it was suggested that the oxidation of the electron transfer chain between PSII and PSI functions as a signal for up-regulation of *psbA* genes in *Synechocystis* [26]. This hypothesis was based on measurements of the amounts of *psbA* transcripts and electron transfer activities under three light qualities [26]. Our data corroborate the finding that when blue and orange light are compared, a high amount of *psbA* transcripts and oxidation of the electron transfer chain between PSII and PSI coincide. However, comparison of more complete action spectra of PSII activity and photosynthesis to that of *psbA* transcription (Fig. 2) does not support the redox control hypothesis. For example, illumination of *Synechocystis* cells with violet or blue light favors PSI thus inducing oxidation of the electron transfer chain between PSII and PSI in both light regimes. Notably, however, the highest amount of *psbA* transcripts was measured under violet light and the lowest amount under blue light, indicating that the redox state of the electron transfer chain is a very unlikely candidate for a regulatory factor for *psbA* gene transcription. Redox-independent mechanisms are further supported by earlier studies on D1 mutant strains showing that no correlation exists between their electron transfer capacity and transcription activity of the *psbA* gene [22,29].

Because *psbA* transcription appeared not to be under redox regulation, we studied if photoinhibition could function as a regulatory factor for *psbA* and *psbD* genes. Such regulation would be very reasonable because the repair of photoinhibited PSII requires the replacement of the damaged D1 protein (and to a lesser extent D2) with a newly synthesized copy [2]. We found that the action spectra of *psbA* and *psbD2* gene transcription resemble the action spectrum of photoinhibition. In line with this result, mutation-induced changes in the turnover rate of the D1 protein are reflected in the transcription rate of the *psbA2* gene [22,29]. Similarly, illumination with increasing irradiances enhances both the turnover rate of the D1 protein and the transcription activity of the *psbA* gene [22]. Oversaturating single turnover flashes that induce photoinhibition [30] but keep the photosynthetic electron transfer chain in its oxidized state were shown to induce transcription of the *psbA* gene in *Synechocystis* cells [5], providing further support to the idea that photoinhibition mediates the enhancement of *psbA* gene expression in the light. The connection between photoinhibition and *psbA* transcription is also supported by the finding that PSII electron transfer inhibitors slowing down photoinhibition induce a slight decrease in the transcription rate of the *psbA* gene [10,12]. It is thus conceivable that photoinhibition produces a signal that induces the transcription of the *psbA* and *psbD2* genes when accelerated synthesis of D1 and D2 proteins is required to repair photodamaged PSII complexes.

Photoinhibition induced by UV or visible light damages the D1 protein, and to a lesser extent also the D2 protein, while all the other PSII proteins remain intact [2,31,32]. In line with this notion, the action spectrum of *psbDC* transcripts differs from the spectra of *psbA* and *psbD2* transcripts (Fig. 1). The activation of the *psbDC* operon occurs more slowly after the onset of illumination and its transcriptional activity is highest in the photosynthetically most efficient wavelength regions (Figs. 1 and 2A). Previous study has shown that the transcription rate of the *psbDC* operon remains constant under the PFDs of 50–1500 μmol m^{-2} s^{-1} and is not affected by those D1 mutations that cause changes in the transcription rates of the *psbA* and *psbD2* genes [14].

If photoinhibition mediates the light-induced changes for *psbA* gene expression, then what is the photoreceptor of photoinhibition? Many different photosensitizers including chl, semiquinone, tyrosine and iron–sulfur centers have been suggested to mediate photoinhibition with numerous hypotheses on the reaction mechanisms in different wavelength regions in plants [33–36]. Interestingly, the in vivo action spectrum of photoinhibition in *Synechocystis*, showing a high efficiency in violet and UV-A light and a peak in the orange–red region, is very similar to the in vitro action spectrum of photoinhibition in isolated plant thylakoids [36,37]. In accordance with our findings, UV light has been shown to be more damaging to photosynthesis than visible light in the green alga *Dunaliella salina* [38], and remarkably the in vivo action spectrum of D1 protein degradation in *Spirodela oligorrhiza* [33] closely resembles the action spectrum of photoinhibition in *Synechocystis*. This notion is important because the action spectra of PSII activity and photosynthesis distinctively differ between plants and cyanobacteria due to different antenna systems. Thus, it is obvious that the major light absorbing pigments do not sensitize PSII for photoinhibition. In a recent study [39] we suggest that light absorption by manganese ions of the oxygen evolving complex is involved in photoinhibition under both UV and visible light.

Acknowledgements: This work was supported by the Academy of Finland. Mika Keränen is thanked for help with the spectra measurements.

References

[1] Tyystjärvi, E. and Aro, E.-M. (1996) Proc. Natl. Acad. Sci. USA 93, 2213–2218.
[2] Aro, E.-M., Virgin, I. and Andersson, B. (1993) Biochim. Biophys. Acta 1143, 113–134.
[3] Mohamed, A. and Jansson, C. (1991) Plant Mol. Biol. 16, 891–897.
[4] Mulo, P., Eloranta, T., Aro, E.-M. and Mäenpää, P. (1998) Bot. Acta 111, 71–76.
[5] Tyystjärvi, T., Tyystjärvi, E., Ohad, I. and Aro, E.-M. (1998b) FEBS Lett. 436, 483–487.
[6] Tyystjärvi, T., Herranen, M. and Aro, E.-M. (2001) Mol. Microbiol. 40, 476–484.
[7] Tsinoremas, N.F., Schaefer, M.R. and Golden, S.S. (1994) J. Biol. Chem. 269, 16143–16147.
[8] Campbell, D., Zhou, G., Gustafsson, P., Öquist, G. and Clarke, A.K. (1995) EMBO J. 14, 5457–5466.
[9] Sippola, K. and Aro, E.-M. (2000) Photochem. Photobiol. 71, 706–714.
[10] Alfonso, M., Perewoska, I. and Kirilovsky, D. (2000) Plant Physiol. 22, 505–515.
[11] Li, H. and Sherman, L.A. (2000) J. Bacteriol. 182, 4268–4277.
[12] Tyystjärvi, T., Herranen, M., Mulo, P., Ohad, I. and Aro, E.-M. (1998) in: Photosynthesis: Mechanisms and Effects (Garab, G., Ed.), pp. 2909–2912, Kluwer Academic Publishers, Dordrecht.
[13] Mate, Z., Sass, L., Szekeres, M., Vass, I. and Nagy, F. (1998) J. Biol. Chem. 273, 17439–17444.
[14] Herranen, M., Aro, E.-M. and Tyystjärvi, T. (2001) Physiol. Plant 112, 531–539.
[15] Toole, C.M., Plank, T.L., Grossman, A.R. and Anderson, L.K. (1998) Mol. Microbiol. 30, 475–486.
[16] Hirschberg, J. and Chamowitz, D. (1994) in: The Molecular Biology of Cyanobacteria (Bryant, D.A., Ed.), pp. 559–579, Kluwer Academic Publishers, Dordrecht.
[17] Yeh, K.-C., Wu, S.-H., Murphy, J.T. and Lagarias, J.C. (1997) Science 277, 1505–1508.
[18] Park, C.-M., Kim, J.-I., Yang, S.-S., Kang, J.-G., Kang, J.-H., Shim, J.-Y., Chung, Y.-H., Park, Y.-M. and Song, P.-S. (2000) Biochemistry 39, 10840–10847.
[19] Garcia-Dominguez, M., Muro-Pastor, M.I., Reyes, J.C. and Florencio, F.J. (2000) J. Bacteriol. 182, 38–44.
[20] Ng, W.-O. and Pakrasi, H.B. (2001) Mol. Gen. Genet. 264, 924–930.
[21] Lin, C. (2000) Trends Plant Sci. 5, 337–341.
[22] Tyystjärvi, T., Mulo, P., Mäenpää, P. and Aro, E.-M. (1996) Photosynth. Res. 47, 111–120.
[23] Sambrook, J. and Russell, T. (2001) Molecular Cloning: A Laboratory Manual, Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY.
[24] Boichenko, V.A., Hou, J.-M. and Mauzerall, D. (2001) Biochemistry 40, 7126–7132.
[25] Mohamed, A., Eriksson, J., Osiewacz, H.D. and Jansson, C. (1993) Mol. Gen. Genet. 238, 161–168.
[26] Bissati, K.L. and Kirilovsky, D. (2001) Plant Physiol. 125, 1988–2000.
[27] Schmitz, O., Tsinoremas, N.F., Schaefer, M.R., Anandan, S. and Golden, S.S. (1999) Photosynth. Res. 62, 261–271.
[28] Pfannschmidt, T., Nilsson, A. and Allen, J.F. (1999) Nature 397, 625–628.
[29] Mulo, P., Tyystjärvi, T., Tyystjärvi, E., Govindjee, Mäenpää, P. and Aro, E.-M. (1997) Plant Mol. Biol. 33, 1059–1071.
[30] Keren, N., Berg, A., van Kan, P.J.M., Levanon, H. and Ohad, I. (1997) Proc. Natl. Acad. Sci. USA 94, 1579–1584.
[31] Trebst, A. and Depka, B. (1990) Z. Nat.forsch. 45c, 765–771.
[32] Zer, H. and Ohad, I. (1995) Eur. J. Biochem. 231, 448–453.
[33] Greenberg, B.G., Gaba, V., Canaani, O., Malkin, S., Mattoo, A.K. and Edelman, M. (1989) Proc. Natl. Acad. Sci. USA 86, 6617–6620.
[34] Jung, J. and Kim, H.-S. (1990) Photochem. Photobiol. 52, 1003–1009.
[35] Barbato, R., Frizzo, A., Friso, G., Rigoni, F. and Giacometti, G.M. (1995) Eur. J. Biochem. 227, 723–729.
[36] Turcsanyi, E. and Vass, I. (2000) Photochem. Photobiol. 72, 513–520.
[37] Jones, J. and Kok, B. (1966) Plant Physiol. 41, 1037–1043.
[38] Ghetti, F., Herrmann, H., Häder, D.-P. and Seidlitz, H.K. (1999) J. Photochem. Photobiol. B Biol. 48, 166–173.
[39] Tyystjärvi, E., Kairavuo, M., Pätsikkä, E., Keränen, M., Khriachtchev, L., Tuominen, I, Guiamet, J.J. and Tyystjärvi, T. (2001) Proceedings of the 12th International Congress of Photosynthesis.