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Excitation energy transfer from phycobilisomes to Photosystem I in a cyanobacterial mutant lacking Photosystem II

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Photosynthetic light-harvesting has been investigated in two strains of the cyanobacterium *Synechocystis* 6803: the wild-type and a mutant lacking Photosystem II. The deletion mutant lacks the genes for the PS II proteins D2 and CP43. D1 and CP47 are also destabilised in the mutant and are not present at detectable levels in the thylakoid. Excitation energy transfer from phycobilisomes to Photosystem I was detected by low-temperature fluorescence emission spectroscopy and by measurement of action spectra for flash-induced P700 photooxidation. In the wild-type, the quantum efficiency of energy transfer from phycobilisomes to Photosystem I is determined by the physiological adaptation of the cells and is maximally about 40%. In the mutant, phycobilisomes transfer energy to Photosystem I with about 80% efficiency. It is concluded that 'spillover' from Photosystem II is not required for efficient energy transfer from phycobilisomes to Photosystem I. These results suggest the existence of a specific phycobilisome–Photosystem I complex.

Introduction

The major accessory light-harvesting complexes of cyanobacteria and red algae are the phycobilisomes, large extrinsic complexes of phycobiliproteins bound to the cytoplasmic surface of the thylakoid membrane (see Refs. 1, 2 for reviews). The structural and energy transfer properties of phycobilisomes have been the subject of extensive investigation, and they are probably the best-understood photosynthetic light-harvesting antennae [1,2]. However, relatively little is known of the association of phycobilisomes with the chlorophyll *a*-binding proteins in the thylakoid membrane. The pathways of energy transfer from phycobilisomes to photosystems I and II have recently been the subject of controversy. Phycobilisomes normally transfer the majority of their excitation energy to PS II via the terminal emitters, long wavelength pigments bound to the large 'anchor' polypeptide of the phycobilisome core [3]. It has been generally assumed that phycobilisomes are coupled exclusively to PS II core complexes. However, a significant proportion of energy absorbed by phycobilisomes does reach PS I [4,5]. This could in principle be due either to 'spillover' (i.e., excitation energy transfer from the PS II core complex to PS I [6]) [7], or due to direct energy transfer from phycobilisomes to PS I [5]. The distinction has major implications for the organisation of thylakoid membrane components: the former case implies a close association between PS II and PS I, whereas the latter case implies the existence of a specific phycobilisome–PS I complex. A number of recent results suggest that spillover is not the major pathway of energy transfer from phycobilisomes to PS I:

(i) The fluorescence decay lifetimes of PS II in intact cyanobacterial cells [8] are not shorter than in isolated PS II particles [9], suggesting that there is no major pathway of energy transfer from PS II to PS I.

(ii) The physiological adaptation of cells to light-state 2 results in the functional decoupling of about 60% of PS II reaction centres from phycobilisomes in the cyanobacterium *Synechococcus* 6301 [10]. However, the lifetime of fluorescence from the phycobilisome terminal emitters does not increase [8,10] and phycobilisome absorbed light-energy is efficiently stored in state 2 [11,12], suggesting that energy may be directly transferred to PS I instead.

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Abbreviations: *A*, absorbance; C, absorption cross-section of PS I; Chl, chlorophyll; FNR, ferredoxin-$NADP^+$ oxidoreductase; P700, primary electron donor of PS I; PBS, phycobilisome; PC, phycocyanin; PS, photosystem; WT, wild-type.

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(iii) The closure of PS II reaction centres does not lead to any increase in the efficiency of energy transfer from phycobilisomes to PS I in *Synechococcus* 6301 [5].

(iv) A PS II-depleted fraction in which functional coupling between phycobilisomes and PS I is retained has been isolated from the thermophilic cyanobacterium *Synechococcus* sp. [13].

Mutant cyanobacterial strains provide an additional method to test the various models for the organisation of the light-harvesting complexes and reaction centres. A deletion mutant of *Synechocystis* 6803 which lacks all major PS II components has been prepared in the laboratory of Dr. W. Vermaas [14]. The mutant lacks the genes for the PS II Chl *a*-proteins D2 and CP43. The remaining PS II Chl-proteins, D1 and CP47, are unstable in the mutant and are not found at detectable levels in the thylakoid membrane [14,15]. However, the synthesis and assembly of phycobilisomes and PS I appear to be normal in the mutant [14–16]. Here, I report a comparison of the efficiency of excitation energy transfer from phycobilisomes to PS I in wild-type and mutant *Synechocystis* 6803 using 77 K fluorescence emission spectroscopy and microsecond flash-induced P700 photooxidation. In the wild-type, the quantum efficiency for energy transfer from phycobilisomes to PS I reaches about 40% in cells adapted to light-state 2. In the mutant, the quantum efficiency is about 80%. I conclude that spillover is not necessary for efficient energy transfer from phycobilisomes to PS I. I present a model which explains these results in terms of competition between PS II and PS I for binding sites at the phycobilisome core.

Materials and methods

Synechocystis 6803 wild-type and psbDICDII$^-$ mutant [14] were gifts from Dr. W. Vermaas (Arizona State University, Tempe, AZ, USA). The cyanobacteria were grown at 35°C in BG11 medium [17] under white light at 50 $\mu E\,m^{-2}\,s^{-1}$ and were bubbled with 5% CO_2 in N_2. In the case of the psbDICDII$^-$ mutant, 5 mM glucose was added to the growth medium. The cells were maintained at a Chl *a* concentration of about 5 μM by daily dilution with fresh growth medium.

For 77 K fluorescence spectroscopy, the cells were harvested by centrifugation and resuspended in growth medium to a Chl *a* concentration of 100 μM. The cells were incubated at 35°C for 5 min either in the dark or under illumination with blue light (defined by a Corning 5–57 filter at 200 $\mu E\,m^{-2}\,s^{-1}$). A few drops were then adsorbed onto filter paper and rapidly frozen in liquid N_2. The sample was not mixed with glycerol, as this has been found to alter the low temperature fluorescence emission spectrum of cyanobacterial cells, particularly with phycocyanin excitation (unpublished results). 77 K spectra were recorded as described in [18] with excitation at 435 nm or 570 nm. The amplitudes of the fluorescence spectra obtained with the two excitation wavelengths were corrected according to the relative quantum flux of the excitation light at the two wavelengths. Spectra from different samples were normalised to the phycocyanin/allophycocyanin fluorescence peak (excitation at 570 nm, emission at 660 nm).

Microsecond transient absorption measurements at 700 nm were carried out as described in [5], using a 4 μs xenon flashlamp as the excitation light-source. The cell suspension (volume 500 ml, Chl concentration 5 μM) was pumped at 5 $ml\,s^{-1}$ through a 750 μl flow cuvette (pathlength 1.0 cm in the direction of the measuring beam, 0.6 cm in the direction of the flash). The sample reservoir was dark-adapted or illuminated with blue light defined by a Corning 5–57 filter at 430 $\mu E\,m^{-2}\,s^{-1}$. Action spectra for P700 photo-oxidation were calculated from the intensity-dependence of the flash-induced 700 nm absorption change at sub-saturating flash intensity as described in [5]. The different excitation wavelengths were defined by Ealing band-pass interference filters (bandwidth approx. 12 nm). The relative numbers of photons in flashes at different wavelengths were calculated from direct measurements of the flash intensity using the same photomultiplier that was used for the transient absorption measurement [5], with reference to the known spectral response function of the photomultiplier. To correct for self-screening by the sample, the average flash intensity in the sample was calculated by reference to the sample absorption spectrum.

Phycobilisome-free thylakoid membranes were prepared as described in [5]. Flash-induced P700 photooxidation was measured as described in [5], in the presence of sodium ascorbate (10 mM) and phenazine methosulphate (10 μM) as electron donors to PS I. The saturating 700 nm absorbance change in the thylakoid membranes was used to estimate the concentration of PS I, assuming a differential extinction coefficient of 64 $mM^{-1}cm^{-1}$ for P700 [19].

Absorption spectra were recorded using an Aminco DW2000 spectrophotometer with bandwidth 2 nm. The sample was placed close to the detector to minimise distortion due to scattering.

Chlorophyll *a* concentrations were estimated using the extinction coefficient of Porra et al. [20] following extraction in boiling methanol and filtration.

Results

Two methods have been used to assess the efficiency of excitation energy transfer from phycobilisomes to PS I in wild-type and mutant *Synechocystis* 6803. Fluorescence emission spectra have been recorded at 77 K and the flash-intensity dependence of

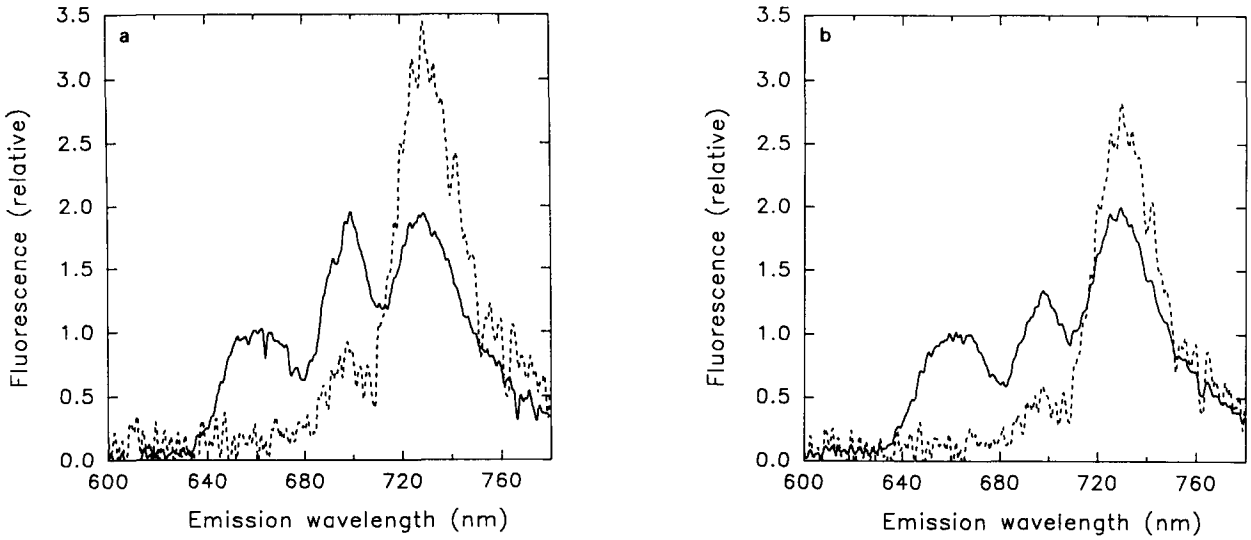

Fig. 1. 77 K Fluorescence emission spectra for cells of wild-type *Synechocystis* 6803. (A) Cells adapted to blue light (state 1). (B) Cells adapted to dark (state 2). (———) excitation at 570 nm, and (— — —) excitation at 435 nm.

P700 photooxidation has been measured at room temperature.

Fig. 1 shows 77 K fluorescence emission spectra for wild-type *Synechocystis* 6803 cells. Excitation was at 435 nm (approx. the peak of the Soret absorption band for Chl *a*) or at 570 nm (on the short-wavelength edge of the phycocyanin absorption band, which peaks at about 620 nm). Prior to freezing, the cells were adapted to blue light (Fig. 1A) or to dark (Fig. 1B): these conditions normally lead to adaptation to light-states 1 and 2, respectively [21]. The spectra show three major peaks: a broad peak at around 660 nm corresponding to a mixture of phycocyanin and allophycocyanin emission; a peak at 698 nm from the core complex of PS II and a peak at 730 nm from PS I [22]. There is also a shoulder at around 690 nm which includes components from the long-wavelength 'terminal emitters' of the phycobilisomes and the PS II core complex [7,22]. Cells adapted to blue light (Fig. 1A) show the characteristics of state 1 adaptation [7,21]: with 570 nm excitation the ratio of the PS II to the PS I fluorescence peaks is about 50% higher than in dark-adapted cells (Fig. 1B) indicating increased relative energy transfer to PS II. The ratio of the heights of the PS I (730 nm) fluorescence peak with 570 nm and 435 nm excitation provides a rough indicator of the relative efficiency of energy transfer from phycobilisomes to PS I. This ratio is 0.56 in state 1-adapted cells (Fig. 1A) and 0.71 in state 2-adapted cells (Fig. 1B). This indicates that the state 2 transition leads to decreased energy transfer from phycobilisomes to PS II and increased energy transfer from phycobilisomes to PS I [23].

Fig. 2 shows 77 K fluorescence emission spectra for dark-adapted cells of the psbDICDII$^-$ mutant. In contrast to the wild-type, the 77 K fluorescence spectra of the mutant cells were unaffected by dark or blue-light adaptation (not shown). As expected these spectra lack the 698 nm peak from PS II both with 570 nm excitation and with 435 nm excitation. The spectra show increased fluorescence at 690 nm from the terminal emitters of the phycobilisome core, suggesting a proportion of functionally decoupled phycobilisomes. However, the ratio of the terminal emitter fluorescence to the fluorescence from phycocyanin and allophycocyanin is about 2.9, much lower than the ratios of about 6–10 generally observed in isolated, intact cyanobacterial phycobilisomes [24]. This suggests that either a quenching process is present which reduces the fluorescence yield from phycobilisomes which are

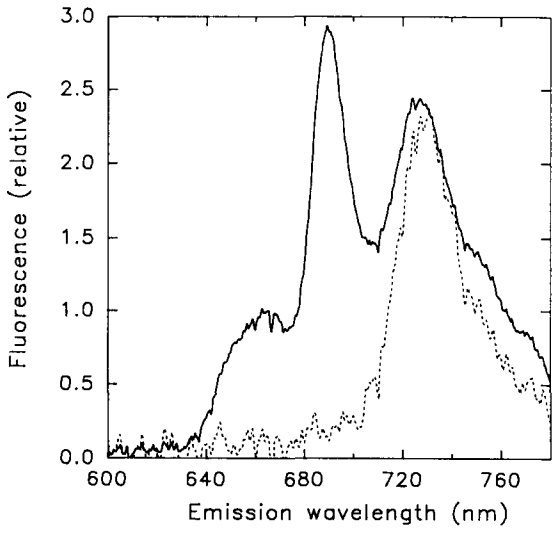

Fig. 2. 77 K Fluorescence emission spectra for cells of the psbDICDII$^-$ mutant. (———) excitation at 570 nm, and. (— — —) excitation at 435 nm.

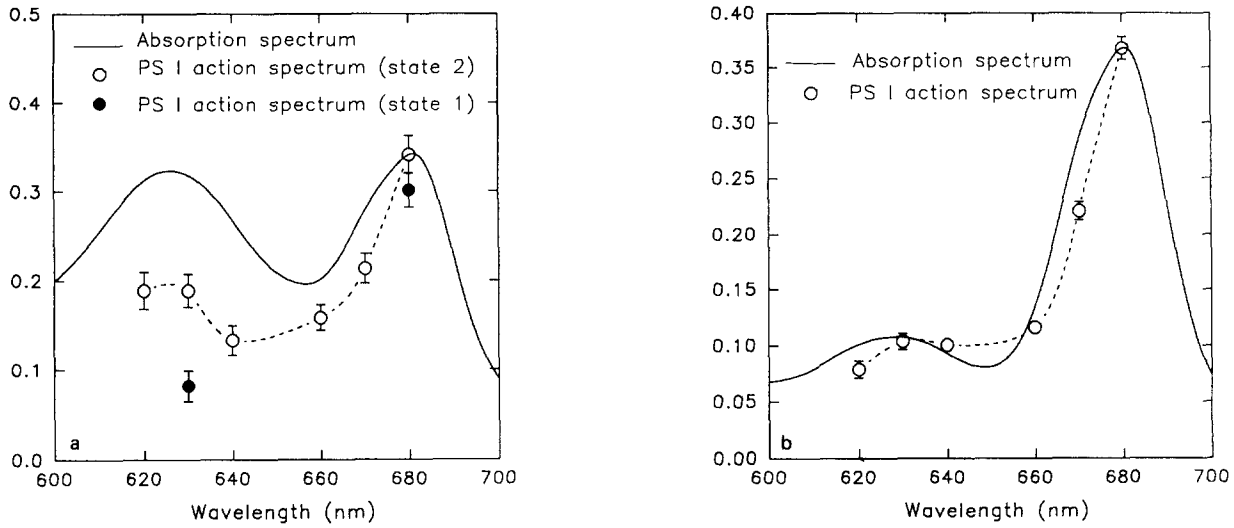

Fig. 3. Absorption spectra and PS I action spectra for wild-type *Synechocystis* 6803. PS I action spectra were obtained from the intensity-dependence of flash-induced P700 photooxidation. (A) Whole cells. (B) Phycobilisome-free thylakoid membranes.

functionally decoupled [16], or energy transfer is possible from the phycobilisome terminal emitters to PS I. The latter possibility is supported by the strong PS I fluorescence emission peak with 570 nm excitation (Fig. 2). In addition, the ratio of the heights of the PS I peak with 570 nm and 435 nm excitation is 1.04 in the mutant (Fig. 2), as compared to 0.71 in the wild-type in state 2 (Fig. 1B). This suggests more efficient energy transfer from phycobilisomes to PS I in the PS-II-free mutant.

This conclusion was confirmed by action spectra for flash-induced P700 photooxidation, using the technique described in [5]. Fig. 3 shows absorption and P700 action spectra for intact wild-type cells (Fig. 3A) and phycobilisome-free thylakoid membranes (Fig. 3B). The absorption and action spectra are arbitarily normalised to the 680 nm Chl *a* peak. In the phycobilisome-free thylakoid membranes, the P700 action spectrum fits closely to the absorption spectrum, except around 670 nm (Fig. 3B). It is possible that this region of the absorption spectrum includes a strong contribution from Chl *a* associated with PS II. The cell absorption spectrum also shows a peak at about 625 nm corresponding to the phycocyanin absorption maximum (Fig. 3A). A peak can also be seen in this region in the P700 action spectrum for cells in state 2 (Fig. 3A) but it is less prominent in comparison to the 680 nm peak than in the absorption spectrum. The ratio of the 625 nm peak to the 680 nm peak is 0.93 in the absorption spectrum, but 0.56 in the P700 action spectrum (Fig. 3A). However, in the phycobilisome-free thylakoid membranes, this ratio is even lower; it is about 0.3 both

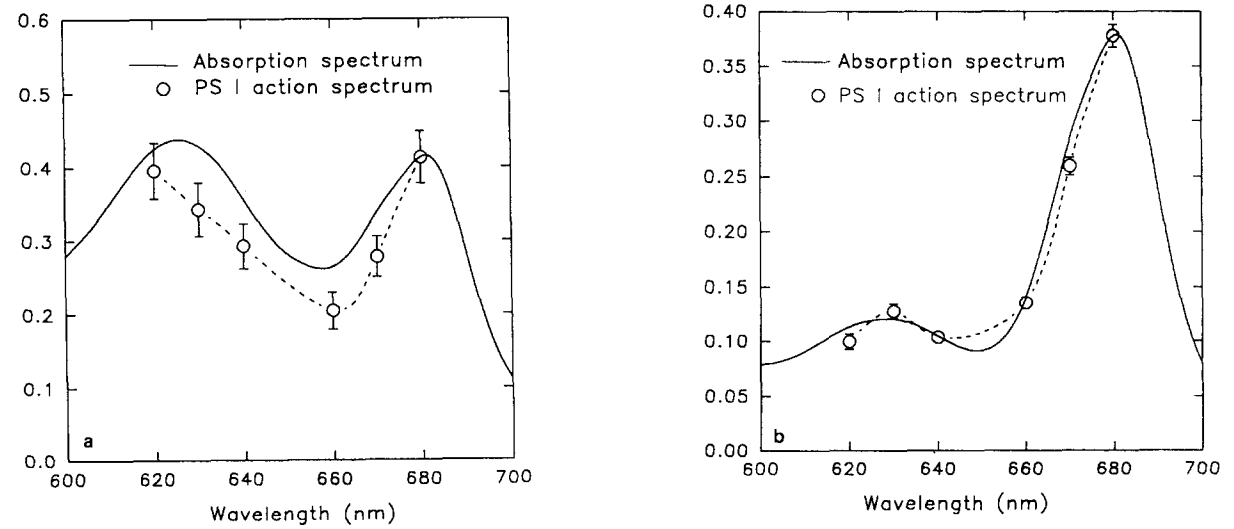

Fig. 4. Absorption spectra and PS I action spectra for the psbDICDII$^-$ mutant. PS I action spectra were obtained from the intensity-dependence of flash-induced P700 photooxidation.(A) Whole cells. (B) Phycobilisome-free thylakoid membranes.

in the absorption spectrum and in the P700 action spectrum (Fig. 3B). This indicates that there is significant energy transfer from phycobilisomes to PS I in cells in state 2. In cells in state 1, however, the ratio drops to about 0.27 (Fig. 3B), similar to the ratio in phycobilisome-free thylakoid membranes.

Absorption and P700 action spectra for cells and thylakoid membranes from the psbDICDII$^-$ mutant are shown in Fig. 4. In the phycobilisome-free thylakoid membranes, the absorption and P700 action spectra have almost exactly the same shape (Fig. 4B). The P700 action spectrum for the cells shows a phycocyanin peak which is only slightly less prominent than in the absorption spectrum (Fig. 4A), indicating efficient energy transfer from phycobilisomes to PS I.

In order to quantify the efficiency of energy transfer from phycobilisomes to PS I it is necessary to deconvolute the Chl and phycocyanin peaks. Table I shows the results of such a deconvolution according to the formulae of Myers et al. [25]. Following deconvolution, the quantum efficiency of energy transfer from phycobilisomes to PS I can be estimated relative to the quantum efficiency of energy transfer from Chl *a* to PS I. The quantum efficiency of energy transfer from Chl *a* to PS I can be assumed to be close to unity in the psbDICDII$^-$ mutant. In the wild-type, about 8% of Chl is associated with PS II (assuming 1 PS II per 600 Chl [26] and 50 Chl in the PS II antenna [25]), so the quantum efficiency of energy transfer from Chl to PS I will be about 0.92. According to these assumptions, the quantum efficiency for energy transfer from phycobilisomes to PS I is about 0.4 in wild-type cells in state 2, but about 0.8 in cells of the psbDICDII$^-$ mutant (Table I).

The concentration of PS I as determined from the 700 nm absorbance change at saturating flash intensity was about 1/160 Chl in the mutant and 1/180 Chl in the wild-type. Assuming about 1 PS II per 600 Chl in the wild-type [26] the PS II/PS I ratio was about 0.3. The phycocyanobilin/Chl ratio, as determined from absorption spectra according to the formulae of Myers et al. [25] was 0.44 in the wild-type and 0.51 in the mutant. Assuming a typical phycocyanin content of about 18 $(\alpha\beta)_6$ hexamers per phycobilisome and 18 phycocyanobilin chromophores per hexamer [1], the ratio of PS I reaction centres to phycobilisomes was about 4 both in the wild-type and the mutant.

Discussion

The results presented here clearly show that the PS II is not required to mediate efficient energy transfer from phycobilisomes to PS I, since the quantum efficiency for phycobilisome–PS I energy transfer is considerably higher in a mutant cyanobacterial strain lacking all PS II Chl-proteins than it is in the wild-type (Table I). These results therefore support the idea that the major route of excitation energy transfer from phycobilisomes to PS I is direct rather than 'spillover' from PS II, and suggest the existence of a specific phycobilisome–PS I complex [5,27]. It is particularly interesting to compare these results with the time-resolved fluorescence data from the same mutant obtained by Bittersmann and Vermaas [16]. These authors attributed two fluorescence decay lifetimes to the long-wavelength terminal emitters of the phycobilisomes in the mutant: a 1.5 ns lifetime with about 25% of total amplitude and a 545 ps lifetime with about 75% of total amplitude. The authors suggested that the 1.5 ns lifetime came from a minority of functionally disconnected phycobilisomes [16]. The presence of some functionally disconnected phycobilisomes is indicated in the present work by the enhanced 690 nm fluorescence peak from the phycobilisome terminal emitters at 77 K in the mutant (Fig. 2). The 545 ps lifetime was attributed to phycobilisomes coupled to the thylakoid membrane and perhaps therefore structurally altered to allow more efficient non-radiative quenching to occur [16]. The present results suggest that the 545 ps lifetime in fact came from phycobilisomes functionally coupled to PS I: the 75% amplitude of the faster decay component [16] is in good agreement with the 80% quantum yield for energy transfer from phycobilisomes to PS I (Table I). The rapid decay

TABLE I

Efficiency of phycobilisome – PS I energy transfer in Synechocystis 6803 wild-type and psbDICDII$^-$ mutant

The ratios of absorption and PS I absorption cross-section at 625 nm and 680 nm are taken from the data in Figs. 3A and 4A and deconvoluted into phycocyanin and chlorophyll contributions according to the formulae of Myers et al. [25]. The PS I absorption cross-section at 625 nm is estimated from the average of the 620 nm and 630 nm points. The quantum efficiency of phycobilisome–PS I energy transfer relative to Chl-PS I energy transfer is estimated by dividing the deconvoluted absorption cross-section ratio ($C^{PC}_{625}/C^{Chl}_{680}$) by the deconvoluted absorption ratio ($A^{PC}_{625}/A^{Chl}_{680}$). The quantum efficiency of phycobilisome–PS I energy transfer is then estimated on the assumption that the quantum efficiency of energy transfer from Chl *a* to PS I is 1.0 in the mutant and 0.92 in the wild-type (see text).

	A_{625}/A_{680}	$A^{PC}_{625}/A^{Chl}_{680}$	C_{625}/C_{680}	$C^{PC}_{625}/C^{Chl}_{680}$	PBS-PS I efficiency
WT (State 2)	0.93	0.72	0.55 ± 0.05	0.31 ± 0.05	0.40 ± 0.08
WT (State 1)	0.93	0.72	0.27 ± 0.06	0.01 ± 0.06	0.02 ± 0.08
psbDICDII$^-$	1.04	0.83	0.89 ± 0.10	0.67 ± 0.10	0.81 ± 0.12

lifetime in the PS I Chl antenna at room temperature (20 ps in *Synechocystis* 6803 [16]) would make energy transfer from phycobilisomes to PS I indistinguishable from a non-radiative quenching process in a time-resolved fluorescence measurement [10].

Bittersmann and Vermaas discounted the possibility that the 545 ps lifetime came from phycobilisomes coupled to PS I on the basis of a measurement of the light-saturation kinetics of PS I-dependent electron transport at 610 nm and 680 nm (results reported, but not shown, in [16]). This measurement appeared to show that 610 nm light could not be efficiently used by PS I, in apparent contradiction to the present results. However, an assay for PS I-dependent electron transport will be affected by competition between the added and native PS I electron acceptors. The result reported by Bittersmann and Vermaas may have been due to very rapid uptake of electrons by the native PS I acceptors in PS I coupled to phycobilisomes, in which case only the electron transport activity of PS I reaction centres decoupled from the phycobilisomes would have been detected by the assay. The report by Schluchter and Bryant [28] that ferredoxin-$NADP^+$ oxidoreductase is coupled to phycobilisomes would provide a structural basis for this effect. The existence of a specific phycobilisome–PS I complex would provide a clear physiological role for phycobilisome-FNR linkage.

The 545 ps lifetime for the phycobilisome terminal emitters in the mutant is comparable to the 370–550 ps lifetime of the phycobilisome terminal emitters in wild-type cells of *Synechocystis* 6803 [16], suggesting that phycobilisome–PS I energy transfer may not be much slower than phycobilisome–PS II energy transfer. It was recently postulated by Su et al. [22] that energy transfer from phycobilisomes to PS I may occur directly from the phycocyanin-containing phycobilisome rods. However, the short lifetime for the phycobilisome terminal emitters in the PS-II-free mutant [16] and in *Synechococcus* 6301 cells in state 2 [8,10] indicates that energy transfer from phycobilisomes to PS I occurs predominantly via the terminal emitters of the phycobilisome core, like energy transfer from phycobilisomes to PS II [3]. This implies that phycobilisomes can bind in a rather similar way to both PS II and PS I. The existence of a Chl-protein which can link both PS II and PS I to the phycobilisome, as postulated in [5], remains a possibility. However, if such a protein exists it cannot be CP43 (as suggested by Nilsson et al. [29]) or any other known PS II Chl-protein, since all these proteins are absent in the PS-II-free mutant [14,15].

Wild-type cells of *Synechocystis* 6803 perform state 1-state 2 transitions which change the proportions of energy transferred from phycobilisomes to PS II and PS I (Figs. 1 and 3) as they do in other cyanobacteria (see [30,31] for reviews). It has been suggested that the

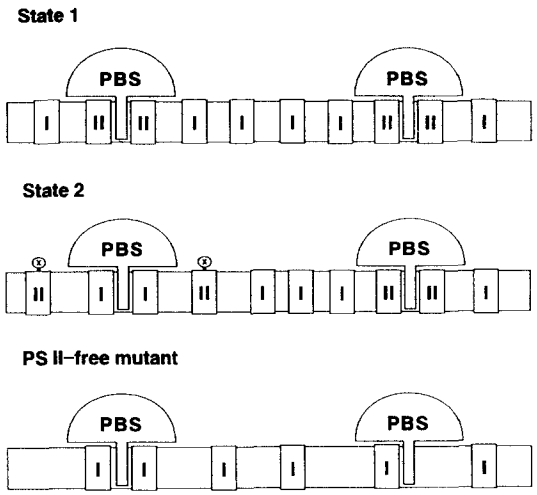

Fig. 5. Model for competition of PS II and PS I for binding sites at the phycobilisome core.

transition to state 2 involves the decoupling of a proportion of PS II core complexes from the phycobilisomes, and the binding of PS I core complexes in their place [5,27]. The trigger for state transitions appears to be the redox state of plastoquinone or a closely associated electron transport component [21]. I was unable to induce any change in the 77 K fluorescence emission spectrum of the PS-II-free mutant by a range of treatments intended to change the redox state of plastoquinone, including illumination and incubation in glucose-free medium. In particular, no decoupling of PS I from the phycobilisomes was apparent in the PS-II-free mutant under the conditions that induced a transition to state 1 in the wild-type (data not shown). This suggests that the presence of PS II is required for state transitions to occur. Fig. 5 illustrates a possible explanation in terms of the competition of PS II and PS I for binding sites at the phycobilisome core. It is postulated that in state 1, phycobilisomes have a higher affinity for PS II than for PS I, and therefore bind almost exclusively PS II core complexes. The transition to state 2 involves the covalent modification (probably phosphorylation [27,31]) of a proportion of PS II core complexes, lowering the affinity of the phycobilisomes to these PS II and allowing PS I core complexes to bind to the phycobilisomes in their place (Fig. 5). In the PS-II-free mutant, the absence of PS II core complexes allows PS I core complexes to bind to most of the available binding sites at the phycobilisome core (Fig. 5). This would explain why phycobilisome–PS I energy transfer is more efficient in the PS-II-free mutant than in wild-type cells (Table I). The presence of some functionally decoupled phycobilisomes in the PS-II-free mutant, despite the low phycobilisome/reaction centre ratio, suggests that the phycobilisome–PS I linkage may be more labile than the phycobilisome–PS II linkage.

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