CP29 is essential for photosynthetic state transitions

CP29, A MONOMERIC LIGHT-HARVESTING COMPLEX II PROTEIN, IS ESSENTIAL FOR STATE TRANSITIONS IN CHLAMYDOMONAS REINHARDTII
Ryutaro Tokutsu, Masakazu Iwai, and Jun Minagawa
From Institute of Low Temperature Science, Hokkaido University, Sapporo 060-0819, Japan.
Running head: CP29 is essential for photosynthetic state transitions
Address correspondence to: Jun Minagawa, Institute of Low Temperature Science, Hokkaido University, North 19 West 8, Sapporo 060-0819, Japan. Phone: (81)-11-706-5471; FAX: (81)-11-706-5471; E-mail: minagawa@lowtem.hokudai.ac.jp

In oxygen-evolving photosynthesis, the two photosystems, photosystem I (PSI) and photosystem II (PSII), function in parallel and their excitation levels must be balanced to maintain an optimal photosynthetic rate under various light conditions. State transitions balance excitation energy between the two photosystems by redistributing light-harvesting complex II (LHCII) proteins. Here, we describe two RNA interference (RNAi) mutants of the green alga Chlamydomonas reinhardtii with one of the minor monomeric LHCII proteins, CP29 or CP26, knocked down. These two proteins have been identified in PSI-LHCI supercomplexes that harbor mobile LHCII proteins from PSII under a state where PSII is preferentially excited (State 2). We show that both the CP29 and CP26 RNAi mutants undergo reductions in the PSII antenna size during a transition from State 1 (a state where PSI is preferentially excited) to State 2, as reflected by non-photochemical quenching of fluorescence, low temperature fluorescence spectra, and functional absorption cross section. However, the undocked LHCIIIs from PSII do not re-associate with PSI in the CP29-RNAi (b4i) mutant because the antenna size of PSI was not complementary increased. The mobile LHCIIIs in the CP26-RNAi (b5i) mutant, however, re-associate with PSI, whose PSI-LHCII/II supercomplex is visualized on a sucrose density gradient. This study clarifies that CP29, not CP26, is an essential component in state transitions and demonstrates that CP29 is crucial when mobile LHCIIIs re-associate with PSI under State 2 conditions.

In photosynthetic organisms, the conversion of light energy into electrochemical energy is driven by two photosystems, photosystems I (PSI) and II (PSII). These photosystems possess peripheral antenna systems that are termed light-harvesting complexes I (LHCI) and II (LHCII), respectively. To optimize photosynthetic electron transport efficiency and avoid damage during light stress conditions, plants and green algae control the distribution of excitation energy between the two photosystems (state transition) (1,2). When the plastoquinone pool is reduced by preferential excitation of PSII (3), a protein kinase that phosphorylates LHCII bound to PSII is activated through the cytochrome bf complex (4,5). The phosphorylation of LHCII leads to the lateral migration of LHCII to the PSI in the unappressed region of thylakoids, where it acts as the peripheral antenna for PSI (State 2). Oxidation of the plastoquinone pool induces the opposite effect, recovering State 1 (6).

The peripheral antenna system for PSII consists of major and minor LHCII proteins as outer trimeric and inner monomeric antennas, respectively (7). Single-particle image analysis of electron micrographs revealed that these peripheral antenna proteins are bound to both sides of the PSII core and each consists of one LHCII trimer and two LHCII monomers in higher plants (8) and green algae (9). CP29 and CP26 are the two conserved minor monomeric LHCII proteins among higher plants and green algae, while another minor monomeric LHCII, CP24, is only present in higher plants (10). Recent reports have suggested pivotal roles for minor monomeric LHCII(s) for state transitions in the unicellular green alga Chlamydomonas reinhardtii. A transfer of the cells from State 1 to State 2 increases the number of phosphorylated residues in CP29 from 2 to 4, and these residues were mapped at the interface of the PSII core and peripheral antenna proteins (11). CP29 and CP26 are dissociated from the PSII core upon phosphorylation (12). Furthermore, the two minor LHCIIIs, in addition to one major LHCII Type II (also known as LhcbM5), are associated with PSI under State 2 conditions.
CP29 is essential for photosynthetic state transitions

Because these minor LHCIIIs border major LHCII trimers as well as the PSII core (14), any LHCII proteins associated with PSII are forced to dissociate when the minor LHCIIIs are undocked. Thus, it has been hypothesized that both major trimeric and minor monomeric LHCIIIs act as linker proteins between the former and the PSII core (13,15). A recent report of an Arabidopsis thaliana mutant lacking CP26 shows plants undergo normal state transitions without CP26 (16). It is not clear whether CP26 is essential for driving state transitions in green algae, because it might be a passenger in the mobile LHCIIIs and passively associated with the PSI-LHCl/II supercomplex in State 2. Currently, it is not known whether CP29 is dispensable for state transitions. In this report, we generated individual RNAi mutants in C. reinhardtii that knock down either CP29 or CP26, and characterized their traits. Both mutants were studied to examine whether they undergo state transitions by several potential methods including phosphorylation of LHCII proteins, fluorescence quenching analysis, antennae measurements, fluorescence emission and excitation spectra, and formation of a PSI-LHCl/II supercomplex. The results clearly demonstrate that the peripheral LHCII proteins in the CP29-RNAi (b4i) mutant dissociate from PSII but do not re-associate with PSI under State 2-promoting conditions. This observation indicates that CP29 is an essential component for state transitions in C. reinhardtii. The CP26-RNAi (b5i) mutant behaves like the wild type (WT), differentiating the role of CP26 in state transitions. We also discuss the low fluorescence nature of the free LHCIIIs that appeared in the b4i mutant in State 2.

Experimental procedures

Strains and media- The C. reinhardtii WT strain TW3 (thi10 cw15 mt+) and a state transition-less mutant DLSΔ (17) were acquired from Drs. K. Shimogawara and F.-A. Wollman, respectively. These control strains and the two RNAi mutants generated in this study (b4i and b5i) were grown photoautotrophically in a high-salt (HS) minimal medium (18) supplemented with 10 ng/mL thiamine and 1.5 mM L-tryptophan, with bubbling 5% (v/v) CO2-enriched air at 23°C under moderate light conditions (120 μE m⁻² s⁻¹).

Plasmid construction- To generate the inverted repeat Lhcb4 and Lhcb5 constructs, short fragments corresponding to the N-terminal part of CP29 and CP26 were PCR amplified using the WT cDNA as a template and the primers b4-75F (ggagtcgaatctcctcgcagccagaa; the BamHI and EcoRI sites are underlined), b4-408R (tcgtctgagctcggactctcgcagct; the PstI site is underlined), b5-76F (tggtgtgtagctccgccgcaagacgcgacgacg; the BamHI and EcoRI restriction sites are underlined), and b5-248R (gtctgtcgagctcggagctcgggctgctg, the PstI site is underlined). Longer fragments were amplified using the primer pairs b4-75F and b4-318R (gtctgtgagctcggagctcgggctgctg; the PstI site is underlined) and b5-76F and b5-355R (gtctgtgagctcggagctcgggctgctg; the PstI site is underlined). Longer regions contained the adjacent coding sequence that functions as a loop structure in the inverted repeats. Both fragments were cloned in the antisense direction into the multiple cloning sites of the vector pBluescript II SK(+) (Stratagene). The resulting inverted repeat cassettes were excised with EcoRI and inserted into the vector NE537 (19), kindly provided by Dr. H. Cerutti, to make NE537/IRLhcb4 and NE537/IRLhcb5.

Nuclear transformation and selection- Nuclear transformations of Chlamydomonas were carried out by electroporation as described previously (20). TW3 cells in the exponential phase (2 × 10⁶ cells/mL) were transformed with 4 μg of either Dral-linearized NE537/IRLhcb4 or NE537/IRLhcb5. The transformed cells were incubated under dim light at 23°C for 2 weeks on TAP (Tris-acetate-phosphate) (21) plates containing 10 ng/mL thiamine, 1.5 mM L-tryptophan, 5 μg/mL paromomycin, and 2.5-20 μM 5-fluorouridine.

Isolation of photosynthetic protein complexes-Thylakoid membranes were isolated as described previously (13). A slightly modified buffer containing 25 mM 2-morpholinoethanesulfonic acid (MES), 0.33 M sucrose, 5 mM EDTA, and 1.5 mM NaCl (pH 6.5) was used to suspend the membranes. Cells were disrupted by BioNeb® (Glas-col, Terre Haut, IN) at 2.5 kg/cm² twice. Sucrose density gradient ultracentrifugation was performed as described previously (13).
CP29 is essential for photosynthetic state transitions

SDS-PAGE, 2-DE, and immunoblotting- SDS-PAGE, 2-DE, and immunoblotting were conducted as described previously (12). The phosphorylated polypeptides were detected with an antibody against phosphothreonine (Zymed Lab., San Francisco, CA).

MS/MS analysis- MS/MS analysis was performed with a Finningan LTQ linear ion trap mass spectrometer (Thermo Fisher Scientific) and SEQUEST software (revision 3.3; Thermo Fisher Scientific) as described previously (12).

Fluorescence quenching analysis- Traces of fluorescence yield were recorded under light-dark cycles as reported previously (22).

Fluorescence Induction and Relaxation fluorometry- The functional absorption cross-section (σPSII) that reflects the functional antennae size of PSII was measured using a FIRe fluorometer system (Model FIRe; Satlantic, Nova Scotia, Canada) as described previously (12).

RESULTS

Efficient and specific inhibition of CP29 and CP26 expression. To determine the specific roles of CP29 and CP26, we generated individual mutants devoid of each of these minor monomeric LHCII proteins. RNAi has been recognized as an effective method to knock down genes in organisms whose genomes are not readily amenable to gene targeting (23,24) and has also recently become a powerful tool in studies on C. reinhardtii (24-26). To knock down CP29 and CP26, we constructed plasmids carrying hairpin RNA sequences corresponding to a sequence from the Lhcb4 and Lhcb5 genes, respectively. The hairpin RNA sequences (consisting of ≈200 base pairs from the 5’ translated region stem structure and ≈100 base pairs of loop structure) were inserted downstream of the RBCS2 promoter into the plasmid NE537; this plasmid also harbors a hairpin RNA sequence for the tryptophan synthase β-subunit, which helps in screening for targeted gene interference (19).

C. reinhardtii transformants with the resultant plasmids NE537/b4i and NE537/b5i were selected using 5-fluoroindole resistance. The amount of CP29 and CP26 proteins in these transformants was quantified by Western blot analysis using an antibody raised against each protein (Fig. 1A). Mutants showing a near complete inhibition of the CP29 and CP26 proteins were named b4i and b5i, respectively, and were used throughout this study. The other LHCII proteins appear not to be affected (Fig. S1 and Table S1). These RNAi mutants show more extensive suppression of the target proteins than our previous RNAi lines (27), whose state transition ability was briefly mentioned in Kargul et al. (15).

Phosphorylation of LHCIIIs during a transition to State 2. LHCII phosphorylation is crucial for the migration of LHCIIIs from PSII to PSI during state transitions (28). We first tested phosphorylation of the LHCII proteins in vivo under State 2-promoting conditions to examine the effects of the RNAi (Fig. 1B). A state transition-less mutant DLSD in which the cytochrome bf complex subunits PetD and PetL are fused (17) and WT were used as a negative and positive control, respectively. Normal LHCII phosphorylation was not the case for the negative control DLSD, where all LHCII proteins were only minimally phosphorylated. Both RNAi mutants thus meet a prerequisite for LHCII migration.

Phosphorylation of LHCIIIs during a transition to State 2. LHCII phosphorylation is crucial for the migration of LHCIIIs from PSII to PSI during state transitions (28). We first tested phosphorylation of the LHCII proteins in vivo under State 2-promoting conditions to examine the effects of the RNAi (Fig. 1B). A state transition-less mutant DLSD in which the cytochrome bf complex subunits PetD and PetL are fused (17) and WT were used as a negative and positive control, respectively. Normal LHCII phosphorylation, except for each of the targeted minor LHCIIIs, was observed in the two RNAi mutants as well as in WT under State 2 conditions. This normal phosphorylation was not the case for the negative control DLSD, where all LHCII proteins were only minimally phosphorylated. Both RNAi mutants thus meet a prerequisite for LHCII migration.

Dissociation of the mobile LHCIIIs from PSII. We performed three sets of fluorescence experiments, fluorescence quenching, functional absorption cross-section of PSII (σPSII), and low-temperature fluorescence spectra, to investigate whether the mobile LHCII proteins dissociate from PSII during state transitions. In
CP29 is essential for photosynthetic state transitions

WT, the maximal yield of fluorescence ($F_m, F_m'$) decreases upon shifting the cells from light to dark (Fig. 2). The observed decrease in fluorescence corresponds to an induction of $q_T$ quenching (22,29,30). Both RNAi mutants exhibited $q_T$ quenching as WT (Fig. 2). On the other hand, the DL$\Delta$ mutant showed no decrease in maximal fluorescence yield (Fig. 2). Likewise, decreases in $\sigma_{ PSI}$ were observed in both the b4i and the b5i mutants, as in WT (Table 1). Intriguingly, the extent of $q_T$ quenching and decrease in $\sigma_{ PSI}$ in the b4i mutant was large compared with WT and the b5i mutant (Table 1); this observation suggests that a larger number of LHCIIIs are undocked from PSII during state transitions in the absence of CP29.

Re-association of the mobile LHCIIIs to PSI.

During state transitions, undocked LHCIIIs from PSII re-associate with PSI to form a PSI-LHCII/II supercomplex (31,32). Functional coupling of the mobile LHCIIIs to PSI is reflected in an increase in the fluorescence emission peak at 718 nm at 77 K (33). The b5i mutant showed a substantial increase in the fluorescence emission at 718 nm under State 2-promoting conditions (Fig. 3A), indicating that the mobile LHCIIIs could be functionally coupled to PSI in the absence of CP29. However, the b4i mutant showed no increase in the PSI fluorescence, although a decrease in PSII fluorescence was observed (Fig. 3A). The DL$\Delta$ mutant showed no change in either PSII or PSI fluorescence during state transitions.

Another line of evidence for the non-coupled mobile LHCIIIs in the b4i mutant was provided by the fluorescence excitation spectra at 77 K. Samples were scanned by excitation from 620 to 690 nm and the fluorescence was monitored at the PSI-specific emission at 718 nm to record spectral components transferring excitations to PSI. To visualize additional spectral components that transfer excitations to PSI only in State 2, the excitation spectra in State 1 were subtracted from that of State 2 (Fig. 3B). The difference spectra for WT shows two peaks, one at 650 nm (chlorophyll b) and the other at 678 nm (chlorophyll a), which is superimposable on the absorption spectra of purified LHCII proteins (Fig. 3B). Thus, in State 2, certain LHCIIIs feed energy to component(s) fluoresces at 718 nm in WT. Whereas the b5i mutant showed the same difference spectra as WT, the b4i mutant displayed no spectral differences. Like the DL$\Delta$ mutant, this CP29-less mutant has no LHCII that could feed energy to PSI in State 2 (Fig. 3B). These data strongly indicate that the undocked LHCIIIs are not functionally re-coupled with PSI in the absence of CP29, whereas a normal state transition occurs even in the absence of CP26.

Formation of PSI-LHCII/II supercomplex in State 2.

To further examine physical associations of the mobile LHCIIIs to PSI, we performed sucrose density gradient ultracentrifugation of the thylakoid membranes from the control and mutant strains under State 2-promoting conditions (Fig. 4A). In WT, the chlorophyll protein complexes were separated into four green bands, as previously reported (Fig. 4A); A1, A2, A3, and A3' bands represent fractions containing free LHCII, the PSII core complex, the PSI-LHCII supercomplex, and the PSI-LHCI/II supercomplex, respectively (13). Whereas the A3' band was present in the thylakoids from both WT and the b5i mutant, it was absent in the b4i mutant under State 2 conditions (Fig. 4B). The PSI-LHCII/II supercomplex in the b5i mutant contains all the LHCIIIs associated with the supercomplex in WT except for the knocked-out CP26 (Fig. 4B). A pool of phosphorylated CP26, CP29, and LHCII type I, which are dissociated from PSII during a transition to State 2 (12), was detected in the PSI-LHCI/II supercomplex fraction in WT and the b5i mutant; however, all of the phosphorylated LHCII proteins were found in the free LHCII fraction in the b4i mutant (Fig. 5). These results supported that the mobile LHCIIIs do not re-associate with PSI in the absence of CP29.

DISCUSSION

In the present study, we demonstrated that one of the minor monomeric LHCII proteins CP29 is crucial for the formation of the PSI-LHCII/II supercomplex during state transitions. In our previous report using a WT strain, CP29 and CP26 were attached to PSI in State 2 (13); also see Fig. 4). Their tight association with the PSI core suggested that they have roles as linkers between the PSI core and the major LHCII trimers within the PSI-LHCII/II supercomplex. The present result is important because it shows CP29 is essential for state transitions while another minor monomeric LHCII CP26 is not. The results obtained with the
CP29 is essential for photosynthetic state transitions

b5i mutant agree with the recent report on the koCP26 mutant in A. thaliana (16). Furthermore, the specific role of CP29 during state transitions was clarified in this study. It is required for re-association of the mobile LHCIIIs with PSI (the latter step of transition from State 1 to 2), whereas not for dissociation of the mobile LHCIIIs from PSII (the former step of transition from State 1 to 2). Previously, an electron density near a PSI subunit PsaH was observed on a single-particle image of the PSI-LHCI/II supercomplex; this density was ascribed to CP29 because it was the only LHCII protein that was present in the sample (15). CP29 is a hyperphosphorylated protein under State 2 conditions (11,13,15) and phosphorylated CP29 is indeed undocked from PSII upon phosphorylation (12). The current results corroborate previous reports and further provide evidence for the specific role of CP29 in state transitions.

Interestingly, the mobile LHCIIs were dissociated from the PSII core even in the absence of CP29 during a transition from State 1 to State 2; this dissociation was shown via an induction of qT quenching (Fig. 2), a decreased fluorescence emission at 680 nm (Fig. 3A) and a reduced functional absorption cross-section of PSII (Table 1). It is thus inferred that the other phosphorylated protein(s), including another minor LHCII CP26 and the PSII core subunits CP43 and D2, are sufficient to undock the mobile antennas. In fact, the mobile LHCIIs are even more extensively dissociated from PSII in the absence of CP29 (Figs. 2 and 3A). Because CP29 locates at the border between PSII core and LHCII trimers (34), its absence could make the binding of LHCII trimers rather weak. The unstable PSII-LHCII supercomplex was also reported in an antisense line of CP29 in A. thaliana, where it was impossible to isolate the PSII-LHCII supercomplex (14). The extended dissociation of mobile LHCIIs from PSII observed in the b4i mutant is compatible with these previous findings.

In the b4i mutant, “free LHCIIs” likely exist under State 2-promoting conditions. Such un-bound LHCII proteins have been thought of as highly fluorescent because they have nothing to transfer the energy to (38). Surprisingly, they exhibited low fluorescence under State 2-promoting conditions (qT quenching; Fig. 2), suggesting that the free LHCIIs are in a quenching state. qT quenching observed here could be accounted for by a simple undocking of LHCIIs from PSII and their conversion to a quenching state, although qT quenching has been conventionally interpreted as fluorescence lowering resulting from migration of peripheral antenna from PSII (a high-fluorescence center) to PSI (a low-fluorescence center).

Acknowledgments—We gratefully acknowledge Kosuke Shimogawara and Francis-André Wollman for providing C. reinhardtii strains, Heriberto D. Cerutti for a plasmid NE537, Alexander V. Ruban for valuable discussion on fluorescence measurements, and Yuichiro Takahashi and Shin-ichiro Ozawa for their help in the MS/MS analysis.
CP29 is essential for photosynthetic state transitions

REFERENCES
CP29 is essential for photosynthetic state transitions


FOOTNOTES

*Our work was supported by the JSPS Research Fellowship for Young Scientists (No. 18004526 to M.I.) and Grants-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science and Technology (No. 18GS0318 to J.M.).

The abbreviations used are: RNAi, RNA interference; 2-DE, two dimensional electrophoresis; DM, n-dodecyl-β-D-maltoside; LHCI and LHCII, light-harvesting complex I and II; PSI and PSII, photosystem I and II; WT, wild type.

FIGURE LEGENDS

Fig. 1. Immunoblot analysis of CP26 and CP29 in the C. reinhardtii mutants b4i and b5i. (A) Expression levels of CP26 and CP29 in the mutants b4i and b5i were compared with those of WT.
CP29 is essential for photosynthetic state transitions

Thylakoid samples (1.0 μg chlorophyll) were separated by SDS-PAGE and immunoblotted using the specific antibody against CP26 or CP29. Averaged expression levels are shown by values as compared to WT (n=4). (B) Phosphorylated polypeptides in WT, b4i, b5i, and DLSΔ cells in State 1 (S1) and State 2 (S2) were detected with anti-phosphothreonine antibody.

Fig. 2. Fluorescence quenching analysis during light-dark cycles. The white and black bars at the top indicate actinic illumination with white light at 120 μE m⁻² s⁻¹ and darkness, respectively. The maximum fluorescence yields of WT (blue; closed squares), b5i (yellow green; triangles), b4i (red; circles), and DLSΔ (black; open squares) cells were recorded at every minute when a saturation pulse was given at 3000 μE m⁻² s⁻¹, and normalized to the steady state yields in the light (0 min). Open and closed triangles indicate the points where fluorescence spectra were measured in Fig.3. Each data point represents a mean of 4-5 measurements. Error bars indicate standard error.

Fig. 3. Low-temperature fluorescence spectra during state transitions. The fluorescence spectra of WT (blue), b5i (yellow green), b4i (red), and DLSΔ (black) cells during the light-dark cycles were measured at 77 K. (A) Fluorescence emission spectra under State 1-promoting conditions (thin line, measured at a point indicated by the open triangle in Fig. 2) and State 2-promoting conditions (thick line, measured at a point indicated by the closed triangle in Fig. 2). Samples were excited at 440 nm. Spectra were normalized to the maximum fluorescence yields simultaneously measured at room temperature. (B) State 2-specific excitation spectra for the PSI fluorescence. Excitation spectra for the PSI fluorescence at 718 nm were normalized to 697 nm, the position of the PSI absorption band, and differences between the excitation spectra under State 2- and State 1-promoting conditions were calculated. For comparison, absorption spectra of purified LHCII are shown (green).

Fig. 4. Chlorophyll protein complexes separated on a sucrose density gradient. (A) Thylakoids from WT, b5i, and b4i cells prepared under State 2-promoting conditions were solubilized and subjected to a sucrose density gradient ultracentrifugation. The green bands were designated A1, A2, A3, and A3′ as previously described (13). (B) Immunoblot analysis of the A3′ fractions were performed with specific antibody. Proteins with 2.5 μg of chlorophyll were loaded in each lane.

Fig. 5. Phosphorylated polypeptides in the chlorophyll-protein complexes in WT, b5i, and b4i strains under State 2-promoting conditions. Polypeptides of the A1, A2, A3, and A3′ bands from the State 2 thylakoids were subjected to immunoblotting with an anti-phosphothreonine antibody. The sample representing the A3′ band in the b4i mutant, which was not observed, corresponds to the fraction equivalent to the A3′ band in WT and b5i mutant.

TABLES

Table 1. Relative antenna sizes during state transitions. The relative maximal fluorescence yield ($F'_{m}$) and an absorption cross-section of PSII ($\sigma_{PSII}$) of the WT, b4i, b5i, and DLSΔ cells in State 1 (S1) and State 2 (S2) shown in Fig. 2 are given. Values are means ± standard errors (n=4).

<table>
<thead>
<tr>
<th></th>
<th>WT</th>
<th>b4i</th>
<th>b5i</th>
<th>DLSΔ</th>
</tr>
</thead>
<tbody>
<tr>
<td>$F'<em>{m}$ (S2) / $F'</em>{m}$ (S1)</td>
<td>0.80 ± 0.06</td>
<td>0.62 ± 0.01</td>
<td>0.78 ± 0.01</td>
<td>0.99 ± 0.01</td>
</tr>
<tr>
<td>$\sigma_{PSII}$ (S2) / $\sigma_{PSII}$ (S1)</td>
<td>0.83 ± 0.05</td>
<td>0.74 ± 0.04</td>
<td>0.79 ± 0.07</td>
<td>1.02 ± 0.02</td>
</tr>
</tbody>
</table>
CP29 is essential for photosynthetic state transitions

Figure 1

(A) WT b4i b5i
CP26
100 100 <10 % of WT
CP29
<5 100 % of WT

(B) WT b4i b5i DLSΔ
CP43 S1 S2 S1 S1 S2 S2
CP26 S1 S2 S1 S2
CP29 + D1 S1 S2 S1 S2
LHCl type I S1 S2 S1 S2
CP29 is essential for photosynthetic state transitions

Figure 2

[Graph showing fluorescence yield over time with different classes]
CP29 is essential for photosynthetic state transitions

Figure 3
CP29 is essential for photosynthetic state transitions

Figure 4
CP29 is essential for photosynthetic state transitions

Figure 5
CP29, a monomeric light-harvesting complex II protein, is essential for state transitions in Chlamydomonas reinhardtii
Ryutaro Tokutsu, Masakazu Iwai and Jun Minagawa

J. Biol. Chem. published online January 13, 2009

Access the most updated version of this article at doi: 10.1074/jbc.M809360200

Alerts:
• When this article is cited
• When a correction for this article is posted

Click here to choose from all of JBC’s e-mail alerts

Supplemental material:
http://www.jbc.org/content/suppl/2009/01/14/M809360200.DC1

This article cites 0 references, 0 of which can be accessed free at
http://www.jbc.org/content/early/2009/01/13/jbc.M809360200.citation.full.html#ref-list-1