Journal of Experimental Botany, Vol. 56, No. 411,
Light Stress in Plants: Mechanisms and Interactions Special Issue, pp. 383–388, January 2005
doi:10.1093/jxb/erh230 Advance Access publication 27 August, 2004

Journal of Experimental Botany

The central role of the green alga *Chlamydomonas reinhardtii* in revealing the mechanism of state transitions

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Received 10 May 2004; Accepted 17 June 2004

Abstract

This review focuses on the essential role played by the green alga *Chlamydomonas reinhardtii* in revealing both the mechanism and the physiological consequences of state transitions. Two aspects are considered. The first is the role of the cytochrome b_6f complex in regulating state transitions, in light of the recently obtained 3D structure. The second is the switch between linear and cyclic electron flow that follows state transitions in *Chlamydomonas*. Structural and dynamic elements that might be involved in such a switch, as well as its consequences on the energetic metabolism, are discussed.

Key words: *Chlamydomonas reinhardtii*, energetic metabolism, linear and cyclic electron flow, state transitions.

Mechanism of state transitions

State transitions were first observed in unicellular photosynthetic organisms (Bonaventura and Myers, 1969; Murata, 1969), and have been described as a mechanism for matching the absorption capacity of the two photosystems with changes in the spectral composition of light.

In plants and green algae, state transitions describe the reversible association of the major antenna complex (LHCII) with either photosystem (PS)II (in state 1) or PSI (in state 2) (reviews in Allen, 1992; Gal *et al.,* 1997; Wollman, 2001; Rochaix, 2002). This process relies on the phosphorylation of LHCII by a membrane-bound protein kinase. Phosphorylation leads to the migration of a fraction of the LHCII away from the PSII-rich grana stacks of the chloroplast, by lateral diffusion in the lipid phase. This results in the accumulation of this protein in the unstacked membranes of the stroma (reviewed in Allen, 1992; Gal *et al.,* 1997; Wollman, 2001), where PSI is mostly located (reviewed in Albertsson, 2001).

Two hypotheses have been proposed to explain such a phenomenon. LHCII migration might be triggered by conformational changes occurring within the protein upon phosphorylation. These conformational changes have been observed in LHCII (Nilsson *et al.*, 1997), and have been proposed to play a role in the docking of P_i-LHCII to PSI. As shown in *Arabidopsis thaliana* mutants, this process is mediated by the small subunit PsaH (Lunde *et al.*, 2000; see also Haldrup *et al.*, 2001, for a review). As an alternative hypothesis, electrostatic repulsion generated by the increase of negative charges in the thylakoids has been proposed to trigger P_i-LHCII detachment from PSII (reviewed in Allen, 1992). In the frame of this hypothesis it has also been proposed that state transitions might result in a partial unstacking of thylakoid grana because of the increased negative charge density, thus enhancing spillover from PSII to PSI (Georgakopulos and Argyroudi-Akoyunoglu, 1994).

The LHCII kinase is activated by the reduction of the plastoquinone (PQ) pool (Allen *et al.*, 1981; Horton and Black, 1981). The interplay between the PQ redox state and the occurrence of state transitions can be summarized in a very simple scheme (Fig. 1; Allen, 1992). Reduction of the plastoquinone pool (either by the activity of PSII, or by other cellular metabolic processes) activates the kinase, which is, in turn, inactivated by PQH_2 oxidation by PSI activity. Dephosphorylation of P_i-LHCII is achieved by a phosphatase, which is supposed to be constitutively active (Allen, 1992), although a possible regulation by the recently discovered immunophilin-like 40 kDa lumenal TLP protein has been recently proposed (Fulgosi *et al.*, 1998)

The cytochrome b_6f complex plays a key role in transducing the redox signal from the plastoquinol pool to the kinase. This has been shown firstly by the absence of state transitions in mutants of the green alga *Chlamydomonas*

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reinhardtii unable to assemble the b_6f complex. These findings have been lately confirmed in vascular plants (reviewed in Gal *et al.*, 1997; Wollman, 2001). The first step of this signal transduction pathway is the binding of PQH_2 to the quinol binding site (Qo site) of the cytochrome complex. This possibility has been proposed on the basis of transient acidification experiments in isolated chloroplasts (Vener *et al.*, 1995, 1997), and then confirmed by the generation of a mutant strain of *Chlamydomonas*, where the cytochrome b_6f complex was assembled, but unable to fix plastoquinol (Zito *et al.*, 1999).

It is known that conformational changes occur in the lumenal portion of the Rieske subunit of the b_6f complex upon binding of PQH_2 to the Qo site (reviewed in Breyton, 2000; see also Kurisu *et al.*, 2003, for a discussion). These changes have been claimed to play an essential role in LHCII-kinase activation (reviewed in Vener *et al.*, 1998; Wollman, 2001). Their role in the activation of the kinase has clearly been shown in *Chlamydomonas* (Finazzi *et al.*, 2001).

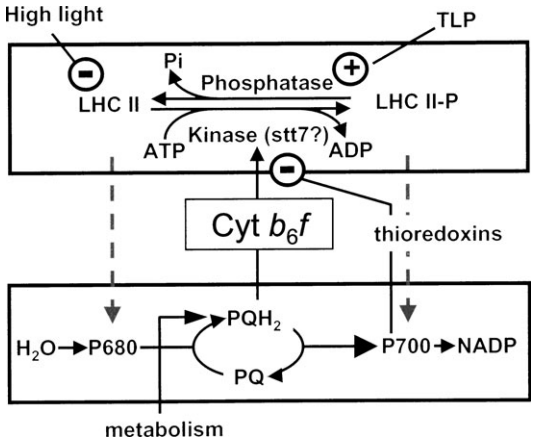

Fig. 1. Schematic representation of the different processes involved in the regulation of state transitions. Non-phosphorylated LHCII transfers absorbed energy to PSII. Reduction of the plastoquinone pool by either imbalance in excitation energy distribution or cellular metabolism leads to the activation of the LHCII-kinase. P_i-LHCII then transfer energy to PSI, increasing its activity. This provides a feedback that inactivates the kinase, though oxidation of the PQ pool, resulting in LHCII dephosphorylation by the prevailing of phosphatase activity. Other factors might modulate this processes: direct down-regulation of the kinase activity by PSI-driven reduction of thioredoxins might take place, as well as high light-induced conformational changes that reduce the accessibility of the phosphorylation site. In addition, regulation of phosphatase activity by an immunophilin-like 40 kDa lumenal TLP protein might take place. (After Allen, 1992.)

The activating signal generated in the lumenal side of the thylakoids (where the Qo site is placed) needs to be transduced through the membrane bilayer, since the active domain of the kinase is situated on the stromal side of the membrane. The nature of the elements involved in this signal transduction pathway are still unknown. The recent isolation in *Chlamydomonas* of the kinase responsible for state transitions (stt7, Depege *et al.*, 2003) has provided one possible explanation for this process, as this protein presents a putative transmembrane helix. This helix might, therefore, be directly involved in sensing plastoquinol binding to the Qo site, as already suggested (Vener *et al.*, 1998). However, the existence of an intrinsic signalling pathway within the cytochrome b_6f complex cannot be excluded a priori. The 3D structure of the cytochrome b_6f complex from *Chlamydomonas*, recently solved by X-ray crystallography (Stroebel *et al.*, 2003), has provided some unexpected features that might be relevant to understanding the mechanism of LHCII-kinase activation.

It has been shown that the tetrapyrrole ring of the chlorophyll molecule, which is present in the cytochrome complex, is exposed to the lipid phase (Fig. 2A). By contrast, its phytol chain is located much deeper within the complex where it could interact with the quinone in the Qo site (Fig. 2B; Stroebel *et al.*, 2003). The chlorophyll molecule might therefore provide a direct pathway for signalling the binding of the quinol from the Qo site to a more peripheral region of the complex, where kinase docking is expected to take place. Indeed, the region where the chlorophyll ring is exposed to the lipid phase is located in close proximity to the zone where kinase docking to the cyochrome b_6f was proposed to occur, on the basis of the functional analysis of a mutant of *Chlamydomonas* (Zito *et al.*, 2002). In this mutant, the small PetL subunit and subunit IV of the b_6f complex were fused

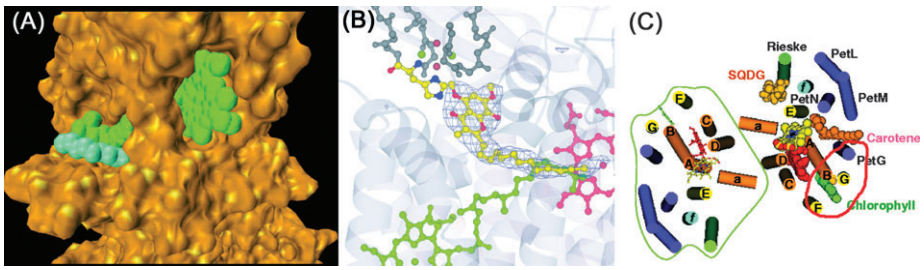

Fig. 2. Structural features of the cytochrome b_6f complex from *Chlamydomonas reinhardtii*. (A) Lipid exposure of the cytochrome b_6f chlorophyll tetrapyrrole ring (green). Position of the quinone is also indicated in blue. (B) Possible interaction between the phytol chain of the cyt b_6f chlorophyll (green) and the isoprenoid chain of Qo site bound quinol, here represented by the plastoquinone analogue tridecylstigmatellin (yellow). Red: b_l haem. (C) Possible localization of the LHCII-kinase docking site on cytochrome b_6f complex (red circle), as suggested by functional characterization of the chimeric mutant *DLS* of *Chlamydomonas* (see text). (After Stroebel *et al.*, 2003.)

(Fig. 2C). While this mutation did not prevent binding of PQH_2, LHCII kinase activation was completely abolished, probably because of a decreased interaction with the cytochrome complex.

Other factors than the redox state of the plastoquinone pool and the cytochrome b_6f complex have recently been proposed to play an active role in the modulation of state transitions. Aro and coworkers (Rintamaki *et al.*, 2000) have suggested that thioredoxins might exert a direct negative control on the kinase itself, down-regulating its activity in high light conditions (Fig. 1). Inhibition of LHCII phosphorylation by high light has also be interpreted in terms of the occurrence of some conformational changes in the LHCII molecule itself, which might prevent its phosphorylation (Zer *et al.*, 2003). Both these mechanisms might represent an efficient mechanism to prevent phosphorylation in saturating light, where state transitions are not required, because the absorption of the two photosystems is light saturated, but the plastoquinone pool is probably largely reduced, owing to the kinetic limitation of its oxidation at the Qo site (reviewed in Bendall, 1982).

Physiological consequences of state transitions

In plants, the role of state transitions is to balance the absorption properties of the two photosystems in low light. Their occurrence does not seem to be essential for plant survival. This is suggested by the very limited phenotype of the *A. thaliana* mutant that lacks PSI-H, and therefore that does not perform state transitions (Lunde *et al.*, 2000). This mutant does not show any visual phenotype when grown under different light quantities and qualities, and displays only very small consequences on the photosynthetic capacity (Lunde *et al.*, 2003).

State transitions in *Chlamydomonas* are larger than in plants: 85% of the LHCII is implicated in this process in this alga (Delosme *et al.*, 1996), while only 20–25% of the LHCII complexes is normally involved in plants (Allen, 1992). Because of this very large redistribution of the LHCII complexes, state transitions in *Chlamydomonas* do not seem to fulfil the role of balancing light absorption between the two photosystems, as in plants. Instead, they tend to increase PSI performance at the expense of that of PSII. For this reason, it has been suggested that they might represent a mechanism that allows a switch between linear and cyclic electron flow around PSI (Vallon *et al.*, 1991).

This hypothesis has been tested experimentally by the application of pump and probe spectroscopy to intact cells of *Chlamydomonas*. This technique has already proven to be extremely useful in the study of electron flow in PSII, PSI, and cytochrome b_6f complexes (reviewed in Joliot *et al.*, 1998). Its application to the study of state transitions (Finazzi *et al.*, 1999) has provided detailed information on the relative contribution of linear and cyclic electron flow to photosynthesis. When light-induced electron injection into the cytochrome b_6f complex was probed in state 1 and state 2 adapted cells, a differential sensitivity to the addition of the PSII inhibitor DCMU was observed. This inhibitor blocked electron flow in state 1 only, suggesting that PSII activity was not required to reduce the PQ pool in state 2 (Fig. 3; Finazzi *et al.*, 1999). On the other hand, an identical sensitivity to the addition of the b_6f inhibitor DBMIB was observed in both state 1 and state 2 conditions (Finazzi *et al.*, 1999). This result is consistent with the occurrence of

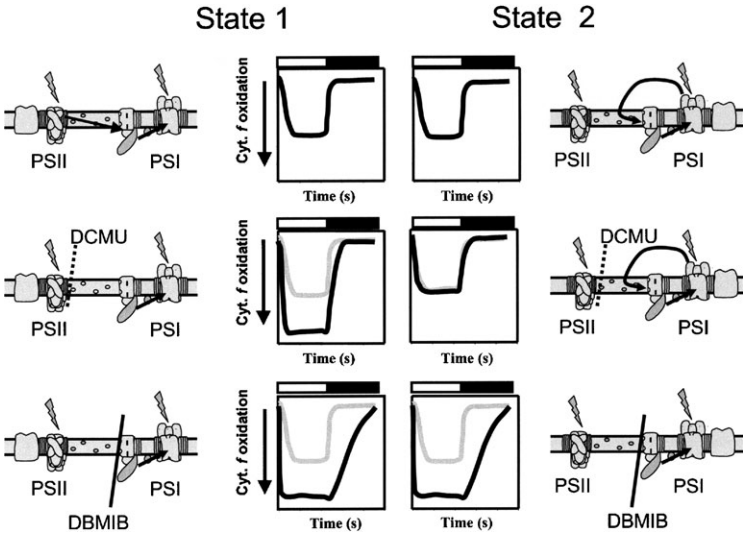

Fig. 3. Switch between linear and cyclic electron flow in *Chlamydomonas reinhardtii*. Cytochrome *f* redox changes are presented, measured in continuous light by absorbance absorption. Consequences of the addition of both the PSII inhibitor DCMU (B) and the b_6f inhibitor DBMIB (C) on the redox state of cytochrome *f* are shown for both state 1 and state 2 adapted cells (dark line, inhibitor; grey line, control). An explanation for each result is provided besides the experimental trace. (After Finazzi *et al.*, 1999.)

a switch between linear and cyclic flow upon state 2 transition, as proposed by Vallon *et al*. (1991).

The strict relationship that exists between state transitions and DCMU sensitivity of electron flow through the cytochrome b_6f complex was confirmed by the analysis of the stt7 mutant of *Chlamydomonas*, which is locked in state 1 because of the knocking out of the LHCII kinase (Fleischmann *et al.*, 1999). In this mutant, electron flow remained sensitive to DCMU inhibition under both state 1 and state 2 promoting conditions (Finazzi *et al.*, 2002). Further evidence for the existence of a correlation between state transition and cyclic flow onset were provided by the measurement of thylakoid swelling upon illumination of *Chlamydomonas* mutants devoid of the ATP-synthase CF_0–F_1 (Majeran *et al.*, 2001). This process, which is due to the building of a transthylakoid ΔpH, was inhibited by DCMU addition in state 1, but not in state 2 conditions. This is consistent with the building of a ΔpH by linear flow in state 1, and by cyclic flow around PSI in state 2. More recently, it has been shown that a reduced metabolic interaction between mitochondria and chloroplasts, which is observed in respiratory mutants of *Chlamydomonas*, promotes a systematic transition to state 2. This results in a reduced oxygen evolution capacity and in an enhanced cyclic flow activity around PSI, as indicated by photoacustic measurements (Cardol *et al.*, 2003).

The study of the relationship between state transitions and the occurrence of cyclic electron flow has been extended to conditions approaching the physiological ones (i.e. phototrophic growth under moderate light intensity). Under these conditions, cells appear to be in an intermediate state between state 1 and state 2, and both linear and cyclic flow seem to take place at the same time (Forti *et al.*, 2003).

Mechanism of the shift between linear and cyclic flow in *Chlamydomonas*

The tight relationship between state transitions and switch between linear and cyclic electrons seems to be specific to *Chlamydomonas* cells, as no relationship between state transitions and cyclic electron flow has been reported in *A. thaliana* (Lunde *et al.*, 2003).

The peculiar relationship between the occurrence of state transitions and the appearance and disappearance of cyclic flow observed in *Chlamydomonas* raises the question of the molecular mechanism(s) that regulates the switch between the two processes. As already proposed by Vallon *et al.* (1991), the concomitant accumulation of both the LHCII and the cytochrome b_6f complex in PSI-enriched stroma membranes upon state 2 transition is probably the determinant for this switch. It might modulate the relative efficiency of linear and cyclic flow for both kinetic and structural reasons. Under state 2 conditions, the ability of PSI to reduce the PQ pool, which is normally lower than that of PSII, might be increased. On the one hand, the decrease of PSII absorption and the concomitant increase of PSI light utilization is expected to decrease PSII-driven electron flow, while enhancing the probability of PQ reduction by PSI. This would lead to a situation where cyclic flow might be prevailing for kinetic reasons. On the other hand, the accumulation of the cytochrome b_6f complex in the stroma lamellae during state 2 transition is expected to increase the physical separation between PSII, which is located in the grana, and the other complexes of the electron transfer chain. This might also promote a switch to cyclic electron flow, for structural reasons.

In spite of these considerations, the intimate mechanism leading to the switch between linear and cyclic flow in *Chlamydomonas* upon state transitions is still unclear. The recent elucidation of the structure of the cytochrome b_6f complex has brought new informations that might be relevant to understanding this process. It has been shown that an additional, unexpected c' type haem exists in the cytochrome b_6f complex of both *Chlamydomonas* (Stroebel *et al.*, 2003) and *Mastigocladus laminosum (*Kurisu *et al.*, 2003). This haem is located in the plastoquinone reducing site (Qi), but is more exposed to the stromal space than the other transmembrane haems. Therefore the c' haem might be directly accessible to water-soluble partners. Among them, it is tempting to propose ferredoxin-NADP reductase (FNR). This enzyme is able to associate with either the cytochrome b_6f complex (Zhang *et al.*, 2001), or PSI, via the PsaE subunit (reviewed in Scheller *et al.*, 2001). Its alternative binding to these complexes might provide a mechanism to couple cyclic electron flow to state transitions. Indeed, because FNR cannot bind to the stacked membranes of the grana (Jennings *et al.*, 1979), its interaction with the cytochrome b_6f complex might occur only when the latter is present in the stroma lamellae. The accumulation of the b_6f in the stroma membranes upon state 2 adaptation might, therefore, promote a preferential binding of FNR with this complex. This would enhance plastoquinone reduction, rather than NADP reduction by PSI, leading to an increased kinetic efficiency of cyclic electron flow.

Conclusion

From an energetic point of view, state transitions in *Chlamydomonas* seem to represent a shift from an oxygenic type of photosynthesis (that generates both reducing power and ATP, state 1) to an anaerobic bacterial one, where only ATP is synthesized (state 2). This switch may represent an advantage in terms of the capacity of adaptation to environmental changes. By maintaining a high quantum yield of ATP synthesis in state 2, cells might be able to maintain vital processes and, therefore, successfully to face stress conditions, where photosynthetic CO_2 assimilation and respiration are inhibited. Consistently, it has been

observed that a systematic transition to state 2 is induced in *Chlamydomonas* under nutrient deprivation conditions (reviewed in Davies and Grossman, 1998), which often lead to a systematic decrease of PSII activity, and, therefore, of the linear electron flow. Under more physiological conditions, i.e. intermediate conditions between state 1 and state 2, cyclic flow might provide the extra ATP required by the Calvin–Benson cycle (in excess of that produced by the linear electron transport). It seems therefore that *Chlamydomonas* is able to match the extent of cyclic flow to the energetic cellular needs by modulating the amplitude of state transitions.

Recent papers have pointed out that, in plants, the concomitant inhibition of plastoquinone reduction via the Fd (Munekage *et al.*, 2002) and by the NDH (NAD(P)H dehydrogenase) pathways (Munekage *et al.,* 2004), leading to the suppression of both the possible pathways for cyclic electron flow, results in a down-regulation of photosynthesis, a reduced photoprotection (at least in the first case), and consequently a diminished growth efficiency. These results probably suggest that, in plants as well, a relationship might exist between changes in the redox state of the PQ pool, the occurrence of cyclic flow, and the ability to cope with changing physiological conditions. The extent to which the two phenomena might be related remains to be assessed (see also Peltier and Cournac, 2002, and Wollman, 2001, for a further discussion on this topic)

On the other hand, it is interesting to note that the same signalling pathway that is involved in triggering the switch between linear and cyclic flow in *Chlamydomonas* (changes in the redox state of the PQ pool), and probably in the modulation of the cyclic flow in plants, seems to be implicated in a more general process as well, i.e.'chloroplast redox signalling' (reviewed in Pfannschmidt, 2003). This phenomenon refers to a series of regulatory processes, which apparently mediate gene expression to a modification of the cellular redox state. The existence of common signalling elements between the two phenomena might provide an easy way to regulate the otherwise rather complicated interplay between ATP changes, redox poise in the chloroplast, and signal transduction for genetic regulation between chloroplast and nucleus, and within the chloroplast itself (reviewed in Goldschmidt-Clermont, 1998; Wollman, 2001).

Acknowledgements

Discussions with Pierre Joliot are acknowledged. Daniel Picot is specially thanked for discussions on the structure–function relationship in the cytochrome b_6f complex, and for providing Fig. 2.

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