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REVIEW ARTICLE

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Light-induced short-term adaptation mechanisms under redox control in the PS II-LHCII supercomplex: LHC II state transitions and PS II repair cycle

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Abstract Oxygenic photosynthesis takes place in the thylakoid membranes of cyanobacteria, algae and higher plants. While cyanobacteria have adapted to relatively constant environments, higher plants had to evolve mechanisms to adapt to continuous environmental changes. These include changes in light intensity, temperature and availability of water. One of the great challenges in plant cell biology is therefore to determine the regulatory mechanisms employed by higher plants and some algae to adapt to these constant environmental changes. The particular emphasis of this review is the description and characterisation of light-induced redox-controlled processes regulating the photosynthetic reactions, which involves maintaining maximal electron transport flow through the PS II–Cytb_6f-PS I-F_oF_1ATP$_{ase}$ electron transport chain and minimising light-induced oxidative damage to PS II which drives the highly oxidising water-splitting reaction. Two of the mechanisms involved in such short-term regulation processes are known as light harvesting complex II (LHC II) state transitions and photosystem II (PS II) repair cycle. They are followed by, and indeed may be a precondition in order to establish, the onset of the subsequent long-term mechanisms of regulation. In particular, the redox control of LHC II state transitions by reversible phosphorylation has been in the focus of many investigations, leading to many new results demonstrating the complexity of thylakoid-associated redox control mechanisms.

Introduction

The transfer of electrons and protons in reduction–oxidation (redox) reactions is known to have fundamental effects on the energy balance in biological systems. Two organelles in photoautotrophic plants and green algae, chloroplasts and mitochondria, are sites for coordinated electron transport chains. Both electron chains are based on redox-active components, able to work as electron/proton-donors and -acceptors. Any change in the rate of the electron flow immediately influences the redox state of the electron transport components and, in a second step, redox-sensitive reactions in the entire organelle. Light-induced electron flow via the thylakoid membrane directly influences the redox state of its components, e.g. the plastoquinone-containing PQ pool, cytochrome b_{559} and the Q_o site of the cytochrome b_6/f complex. Changes in their redox states are known to be the initial step for redox-mediated reactions within the chloroplast as a response to variations in environmental conditions (see Scheibe 1991; Allen and Williams 1998; Link 1999). A fast and efficient control system to react to changes in the environment is essential for the survival of photosynthetically active organisms. Homeostasis, the maintenance of a constant internal environment in the face of external changes, is an active adaptation process that may be either physiological or developmental. In order to compensate for changes in light intensity and quality, photosynthetically active organisms react either with activation/deactivation mechanisms or with *de novo* synthesis/degradation processes of photosynthetic and metabolic enzymes. This kind of short-term and long-term photoacclimation requires a coordinated regulation of the enzymatic reactions and the nuclear and plastidial gene expression (Allen 1993, 1995). Most of these regulation systems are based on redox control mechanisms via redox-active components inside the cell (Allen and Nilsson 1997; Pfannschmidt et al. 1999). A common feature is the fact that changes in the redox state of single electron transport components lead to the activation of specific reactions in order to protect the organism. Several of those reactions may be connected to each other via signal transduction pathways. The consequence is a stepwise adaptation to environmental changes. During evolution, cells developed a variety of answers to external and internal stress (Foyer 1996). Many of these adaptations exhibit direct consequences in terms of structure

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and function of the thylakoid-associated protein complexes. In this respect, the photosynthetic apparatus in the thylakoid membrane acts like a sensor system which, via of intermediate electron carriers, initiates signal transduction pathways for downstream metabolic and gene regulatory responses (Link 1999). Apart from the redox-active intermediates, such as plastoquinone and the cytochromes, the PS II complex and its connected antenna system LHC II (see Paulsen 1995) seem to play an especially important regulatory role in environmental adaptation processes. The PS II-LHC II supercomplex is central to several stress responses induced by light and temperature changes or nutrition deficiency.

The PS II-LHC II supercomplex is the main target site for redox-regulated adaptation mechanisms upon environmental changes

Photosystem II (PS II) is the centre of the photosynthetic water oxidation reaction, and so environmental changes such as in light intensity and light quality immediately influence the energy state of its electron and proton transport components, namely cytochrome b_{559} and the quinones Q_a and Q_b. The redox state of PS II plays an important role in regulation mechanisms during light-induced adaptation processes. Generally, the PS II complex, together with its light antenna system LHC II (Fig. 1), can be described as the 'natural' site for stress-induced redox control. The turnover of PS II is a crucial regulatory step in the entire electron transport process and is decisive for the redox state of the plastoquinone pool. Oxidation of PS II and reduction of Q_b and Q_a is limited by the diffusion rate. In order to avoid light stress-related damages a sensoric regulation system in PS II for the induction of specific protection mechanisms does make sense. Under excess light conditions, several initial processes contribute to the protection of plants to quench reactive chlorophyll and oxygen species by promoting the dissipation of excess absorbed light energy as heat (Horton et al. 1994). This energy dissipation is measured as non-photochemical quenching of chlorophyll fluorescence (NPQ). NPQ prevents the overreduction of the PS II reaction centres and, by competing with photochemistry, might even influence further light regulation mechanisms such as LHC state transitions (Krause and Weiss 1991). However, it is well established that the major component of NPQ is a pH-dependent type of quenching (Horton et al. 1994). Recent results have demonstrated that NPQ is closely correlated with the amounts of zeaxanthin and anteraxanthin formed by the operation of the xanthophyll cycle (Niyogi et al. 1997) and that the PS II subunit PsbS might be the site for NPQ (Li et al. 2000).

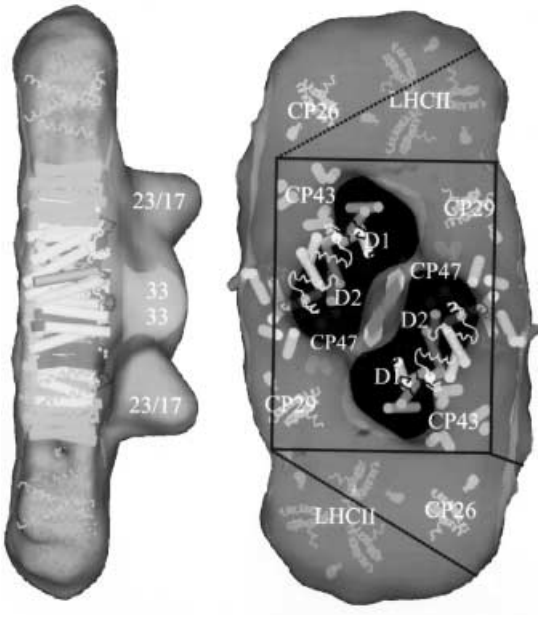

Fig. 1 Structural model of a LHC II-PS II supercomplex by Nield et al. (2000): side (*left*) and lumenal top view (*right*). *23/17* Extrinsic PS II peptides; *CP26*, *CP29*, *CP43*, *CP47*, *D1*, *D2* PS II peptides; *LHC II* Light harvesting complex II

The overreduction of the PQ pool under continuous excess light conditions and the subsequent induction of redox controlled reaction is the direct consequence of the fact that the internal PS II protection systems, trying to avoid any overexcitation of chlorophylls and the production of radical oxygen species (ROS), are not working efficiently enough to protect the photosynthetic apparatus from severe damage.

The reduced state of the PQ pool is now the initial signal for further protection mechanisms, mainly induced by reversible phosphorylation/dephosphorylation cascades as is known for many other biological regulation systems (see Krebs 1994).

The consequence of an increase in the electron transfer from PS II to PSI is a change in the redox state of the intermediate components. In a two-step mechanism, electrons and protons are transferred to plastoquinone (PQ) and thereby reduced to plastohydroquinone (PQH_2). The rate of reduced PQH_2 was suggested at an early stage of investigations to be important in redox control processes in the chloroplast (see Bennett 1991). At the centre of interest concerning PQ-meditated redox-regulation processes was the activation control of thylakoid-associated kinases. Several studies pointed to a key role of PQ as a redox regulator for thylakoid kinase(s). The evidence came from various different investigations: the kinase(s) were activated progressively by a long series of single turnover flashes of light (Allen et al. 1981), leading to the suggestion that multiple molecules of the redox regulator are involved. Furthermore, it was noted that PQ is up to 25 times more abundant in thylakoids than other redox components (Anderson 1986). Redox titration experiments measuring the activation of the kinase(s) in the dark gave midpoint redox potentials close to that of PQ (Horton et al. 1981).

These days, the redox state of the PQ pool is widely accepted to be involved in several redox-regulated reactions not only in the chloroplast but also in the cytosol

and the nucleus (Danon and Mayfield 1994; Allen 1995; Vener et al. 1995; Reinbothe and Reinbothe 1996; Link 1999; Pfannschmidt et al. 1999). Many of these redox reactions are demonstrated to be part of the adaptation mechanism in response to environmental changes.

Short-term adaptation processes: LHC II state transitions and PS II repair cycle

The LHC II/PS II supercomplex (Fig. 1) is the target site for the two major short-term responses to changed light conditions presented in the following sections.

The fully reduced PQ pool induces the activation of several redox-controlled mechanisms including the activation of a serine/threonine kinase which specifically phosphorylates peptides of the LHC II complex of PS II (Bennett 1979). The phosphorylation of LHC II proteins induces LHC II state transitions, so-far the best described short-term mechanism, which enables the plant to regulate and control the light energy distribution between the two photosystems (Gal et al. 1997). In particular, plants are exposed to transient short-term changes in light intensity and quality, leading to an imbalance between the excitation rates of PS II and PS I. The regulation of optimal utilisation of absorbed light energy and avoidance of oxidative damage constitutes a major problem for all oxygen-evolving cells. However, adaptations to transient changes are achieved by regulation of photochemistry: in order to balance the absorbed energy distribution between PS II and PS I, reversible phosphorylation of the LHC II antenna results in reversible coupling with the photosystem cores (Fork and Satoh 1986).

The current understanding is that the phosphorylation of distinct Lhcb peptides on a threonine residue at their *N*-termini induces conformational changes leading to a dissociation of parts of the trimeric complex (see Allen 1992; Andersson and Barber 1994). The monomerisation of LHC II is the precondition for translocation of parts of the LHC II complex from PS II to PS I (see Allen et al. 1981; Telfer et al. 1983; Allen and Holmes 1986). State transitions were first discovered by Bonaventura and Myers (1969) and by Murata (1969). They described a control system of excitation energy transfer in photosynthesis visible by light-induced changes of chlorophyll *a* fluorescence in *Porphyridum cruentum* and *Chlorella pyrenoidosa*. It is now generally established that cells in which the LHC II-antenna is associated with PS II are referred to as cells in state 1, characterised by a high chlorophyll *a* fluorescence emission level. Cells in which parts of the LHC II complex are dissociated from PS II, referred to as state 2, are characterised by comparable low fluorescence emission levels. Consequently, the main feature of state 2 to state 1 transitions in higher plants and green algae is the enhancement in fluorescence emission from PS II. This situation can be induced in vitro by illumination with strong (PS I) red light around 710 nm. On the other hand, transitions from state 1 to state 2 can be induced by state 2 light (PS II light around 680 nm), which is the redistribution of excitation energy in favour of PS I, which is initially rate limiting but now becomes more effective in capturing light energy. Under state 2 conditions the PQ pool is fully reduced and the redox-regulated LHC kinase is activated. The consequence is a LHC II complex which is phosphorylated to a high degree. The phosphorylated part of LHC II migrates from the PS II-enriched grana area to the PSI enriched stroma area of the thylakoid membrane, where it preferentially provides PS I with light energy. Recently, the potential PS I docking site for LHC II was identified as the PsaH subunit of PS I (Lunde et al. 2000). Under these conditions the light-induced electron flow via PS II is reduced. This mechanism not only provides for a better and more efficient distribution of light energy between the two photosystems but also prevents overexcitation of PS II-chlorophyll *a*. The process controlling the migration of phosphorylated LHC II is not yet fully understood. Phosphorylated LHC II has to migrate at a rate of about 250 nm per min (Telfer et al. 1983). This is much slower than expected for simple diffusion forces. One explanation could be that interactions of P-LHC II with charged lipids in the bilayer membrane slows down the speed of migration. Another reason might be the trimeric structure of LHC II and the obvious time delay caused by the dissociation into monomers before migration of parts of the LHC II complex can occur. The light activation of the membrane-bound LHC II kinase was first indicated by John Bennett, who demonstrated its sensitivity to the blocking reagent DCMU (Bennett 1979). Later, Allen et al. (1981) showed that the kinase activity in isolated pea chloroplasts was dependent on the redox state of the plastoquinone pool. Since that time, several research groups have tried unsuccessfully to identify and isolate the LHC II kinase. Only one biochemical approach has been partly successful where PS II core complexes were isolated containing the active LHC kinase (Race and Hind 1996; Hartwig et al. 1998). Incubation of these samples with recombinant LHC II as a substrate led to an in vitro phosphorylation of recombinant LHC II. Furthermore, the investigations with recombinant pea Lhcb1 proteins missing an *N*-terminal segment including the possible phosphorylation site revealed that LHC II proteins are possibly phosphorylated at more sites.

A more promising molecular genetic approach was carried out by Snyders and Kohorn (1999) to identify thylakoid kinases. They succeeded in identifying cDNAs which encode for a protein that uses LHCP as its target substrate (Smith and Kohorn 1991) and subsequently found a family of thylakoid-associated kinases (TAKs) in *Arabidopsis thaliana* (Snyders and Kohorn 1999). An interesting feature was that TAKs are partial homologous to the human $TGF_{\beta}1$-receptor protein. $TGF_{\beta}1$-receptor type 1 is known to carry a signal sequence which is activating the phosphorylation of the $TGF_{\beta}1$-receptor type 2. If TAKs are demonstrated to be of specific importance for LHC II phosphorylation and shown to be generally common in all higher plants and green algae, their homology to $TGF_{\beta}1$ receptors would be the first indication that signal cascades are involved in the activation and regulation of LHC II state transitions.

Alternatively, molecular genetic approaches, targeting nuclear encoded *genes* that are involved in LHC II state transitions, have been employed. These approaches included the construction and screening of tag-induced "knock out" mutants in *Chlamydomonas reinhardtii* that are defective in the mechanism of state transitions (Fleischmann et al. 1999; Kruse et al. 1999).

State transitions are known to occur in a similar way in cyanobacteria. Here, light energy distribution is regulated by lateral movement of their soluble light harvesting system, the phycobilisomes, between the photosystems (Mullineaux et al. 1997). A gene has been identified that seems to play an important role in the regulation of this cyanobacterial state transitions (Emlyn-Jones et al. 1999). However, the regulation of state transitions in cyanobacteria is even less clear, with no reversible phosphorylation of light antenna proteins identified so far.

Evidence for more then one redox-regulated thylakoid protein kinase came from Schuster et al. (1986) who showed that phosphorylation of some PS II core proteins continued under photoinhibitory light conditions, whereas LHC II phosphorylation was abolished. In addition Sundby et al. (1989) demonstrated that bicarbonate stimulates phosphorylation of the LHC II peptides and inhibits PsbH phosphorylation. The existence of two different kinases would make sense regarding the fact that the phosphorylation of distinct PS II proteins allows specific factors to be independently controlled within the chloroplast. Similarly to the mechanism of LHC II phosphorylation, the PS II kinase is regulated by the redox state of the PQ pool. It has been demonstrated that under conditions where the PQ pool remains fully reduced a number of PS II core peptides in higher plants become reversible phosphorylated at their *N*-termini including PsbH, PsbB (CP43), PsbA (D1) and PsbD (D2) (Ikeuchi et al. 1987; Marder et al. 1988; Michel et al. 1988). The role of PS II subunit phosphorylation remained uncertain. Investigations concentrated on a functional involvement in the regulation of the *turnover* of the PS II reaction centre protein D1 (Barbato et al. 1992; Barber and Andersson 1992; Rintamäki et al. 1996; Kruse et al. 1997). The turnover of the D1 protein, as part of the repair cycle of PS II, increases under conditions of exposure to excess light. The degradation of the RC protein, and to a lesser extent that of other PS II core proteins, is the result of alteration of the PS II core due to oxidative damage induced under excessive light. The main role of phosphorylation of PS II core subunits is thought to be a reduction of the degradation rate of D1 and D2 proteins (Aro et al. 1993; Ebbert and Godde 1994; Koivuniemi et al. 1995; Rintamäki et al. 1996), thus ensuring that the rate of degradation is compatible with the rate of new D1 synthesis. According to Barbato et al. (1992) the D1 protein degradation processes involved in a conversion process of dimeric PS II to the monomeric form, the subsequent release of the proximal antenna peptide CP43 and lateral movement of monomeric CP47-RC subcomplexes from the granal region into the stromal region, are similar to LHC II migration (Fig. 2). The turnover of D1 itself can be seen as part of the protection mechanism of the PS II

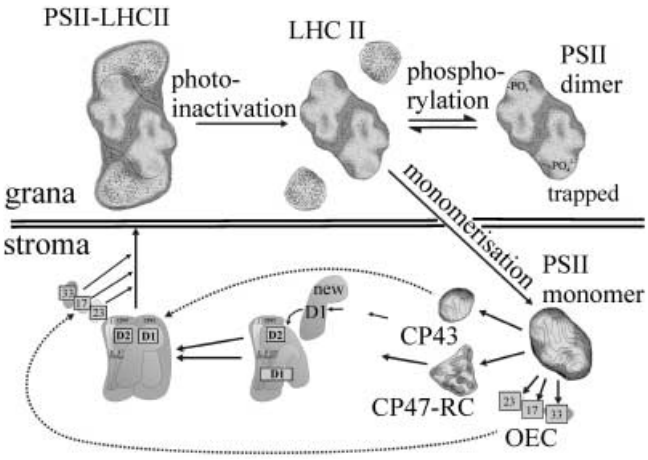

Fig. 2 Model explaining the role of *N*-terminal phosphorylation of PS II proteins and dimer/monomer interconversion for synchronising the replacement of the D1 protein after photoinhibitory damage and CP 43 release based on two models by Barbato et al. (1992) and Kruse et al. (1997) (adapted from Jon Nield and James Barber, Imperial College London). *PO_4* Phosphate group; *CP47-RC* PS II reaction centre + CP47; *OEC*: oxygen evolving complex

reaction centre against high light-induced damage and is based on an enzymatic degradation of damaged D1 and re-incorporation of newly synthesised D1 (see Barber and Andersson 1992). Phosphorylation experiments have demonstrated the important role of reversible PS II protein phosphorylation for the PS II dimer to monomer conversion in terms of short-term adaptation mechanisms in reaction to increasing illumination and overexcitation of the PS II reaction centre (Kruse et al. 1997). From these results it was feasible to suggest that the key step of PS II repair, the D1 turnover, is mainly regulated by the control of PS II dimer to monomer conversion via phosphorylation/dephosphorylation processes in PS II (Fig. 2).

Complex answers to environmental changes rely on complex networks: the fine tuning of LHC state transitions and PS II repair cycle

In both the kinase-related redox solutions described in the section above the phosphorylation of LHC II proteins and PS II proteins is reversible. It is well known that under conditions where the PQ pool is re-oxidised, phosphatases become active and catalyse the dephosphorylation of the kinase-target peptides in LHC II and PS II (see Allen 1992). It is not yet established whether the phosphatases are controlled by the redox state of the PQ pool comparable to the kinase control mechanism (Carlberg and Andersson 1996) or whether their activity is constitutive but suppressed in a reduced environment, while the corresponding kinases are active (Silverstein et al. 1993).

The existence of at least two distinct kinase/phosphatase mechanisms, one for the LHC complex and the other for the PS II complex, is a feasible system because both redox-controlled reactions have different tasks at

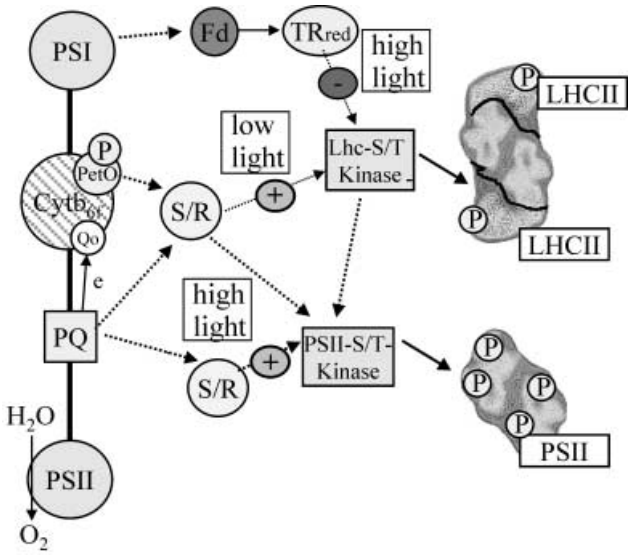

Fig. 3 Model of a cooperative regulation of the LHC II kinase and the PS II kinase under different light conditions. The LHC II kinase is deactivated under high light (–) by reduced thioredoxin and activated under low light conditions (+) by a reduced PQ pool and phosphorylated PetO of the $Cytb_6/f$ complex. The PS II kinase is activated under high light via a reduced PQ pool. *S/R*: possible sensor/regulator systems controlling and/or combining the activation of both kinases

different times (see Bennett 1991). State transition induced by LHC phosphorylation is a very fast reaction process and seems to play an important role, in particular under low light conditions (Pursiheimo et al. 1998). Furthermore, the LHC II phosphorylation reaction is known as a strategic answer to light changes with a half-life of about 4 min (Telfer et al. 1983). In contrast, the PS II kinase is active when illumination is increasing and still active under photoinhibitory conditions, when the LHC II kinase is already inhibited (Bennett 1991). Since both kinases are activated via the redox state of components of the electron transport chain, it is feasible to suggest that a coordinated network signal transduction controls both reactions (Fig. 3).

The complexity and variety of redox-controlled processes in the chloroplast requires a signal control- and pathway fine tuning system for the coordination of redox signals induced by the electron transport via PS II and the reduction of the PQ pool. Fine tuning in the chloroplast is thought to rely on redox sensors and regulators, possibly in a similar way to bacterial two-component regulation systems (see Allen 1993).

Regulation of LHC II state transitions under different light conditions: the $Cytb_6/f$ complex and the ferredoxin/thioredoxin system

Although the major metabolic function of Cyt b_6/f is the transduction of energy in membrane systems, this complex seems to have an additional role in signal transduction in chloroplasts (see Keren and Ohad 1999). The current understanding is that PQ-binding on the Q_o site of the $Cytb_6/f$ complex induces the activation of a kinase that either phosphorylates LHC peptides directly or activates another LHC-kinase (de Vitry et al. 1999; Vener et al. 1997, 1999; Zito et al. 1999). Investigations with antisense plants in *Arabidopsis thaliana* support this idea. Anderson et al. (1997) showed that antisense plants with a reduced expression rate of $Cytb_6/f$ and an overreduced PQ pool do not submit any redox signals to the LHC kinase.

With the identification of a phosphoprotein with a molecular mass of 15.5 kDa in the $Cytb_6/f$ complex, called suV, new evidence raised for a functional involvement in the regulation of the LHC II state transitions. The suV peptide, encoded by the petO gene, is conserved in other photosynthetic eukaryots, co-purifies with $Cytb_6/f$ and is absent in $Cytb_6/f$ deletion mutants. Of particular interest is that this peptide is phosphorylated upon state transitions and is thought to be a part of the signal transduction pathway between the PQ pool, the Q_o-site and the LHC kinase leading to the induction of state transition (Hamel et al. 2000). However, whether suV is directly involved in LHC kinase signal transduction is not yet demonstrated. It should be noted that the petO gene product seems to be absent in cyanobacteria.

A second regulatory mechanism for the regulation of LHC II phosphorylation was recently identified by Rintamäki et al. (2000). They demonstrated that the regulation of LHC II phosphorylation is mediated by the chloroplast ferredoxin–thioredoxin system. In this context a model was presented highlighting the major role of reduced thioredoxin for the inhibition of the LHC II kinase activity under high-light illumination. In contrast, low-light conditions seemed to activate the kinase by the reduction of the $Cytb_6/f$ complex via the PQ pool.

The model in Fig. 3 summarises the current knowledge concerning the complex coordination of the LHC II kinase and the PS II kinase. So far no participating redox sensor/regulator system has been identified. However, the observation that some *Chlamydomonas* nuclear mutants with defects in state transitions (Kruse et al. 1999) are defective in functional PS II repair cycle as well (O. Kruse 2001, unpublished) makes it feasible to suggest that the regulation of both short-term adaptation mechanisms is linked in a certain way. Whether this linkage is based on a direct kinase to kinase communication or on the common use of the same sensor/regulator system is part of future investigations.

The oligomeric states of LHC II and PS II are important for their function in the redox controlled adaptation processes of LHC II state transitions and PS II repair cycle

Structural aspects concerning the oligomeric state of PS II are important for their role in plastidial redox control processes, since it is established for many other oligom-

eric protein complexes that their function is directly dependent on the oligomeric structure of their complexes (see Reilly et al. 1995; Schaller et al. 1995; Yu et al. 1996). In this circumstance the functionality of both the described redox-regulated reactions to varying light conditions, LHC II state transitions and PS II repair cycle, are depended on the oligomeric state of their target complexes, LHC II and PS II. Biochemical and electron microscopy work on PS II-LHCII supercomplexes revealed a dimeric two symmetry-related structure of PS II with at least one trimeric LHCII complex (Boekema et al. 1995, 1999). Further detailed analyses of the PS II-LHCII supercomplex on the role of extrinsic proteins (Boekema et al. 2000a) and on the arrangement in crystalline macrodomains within the thylakoid membrane (Boekema et al. 2000b) highlighted the importance of a certain functional structure not only for the stacking of the grana membrane but also for efficient transfer of excitation energy.

The three-dimensional reconstitution of LHC II, obtained by Kühlbrandt and Wang (1991) at 6 Å resolution and 3.7 Å cryo-electron microscopy data by Kühlbrandt and Downing (1989) revealed that LHC II shows a trimeric arrangement. The negative charge induced by reversible phosphorylation certainly affects the structure of this oligomeric complex (Nilsson et al. 1997). Different models have been proposed for the control of LHC state transitions by reversible phosphorylation (Barber 1982, 1986; Staehelin and Arntzen 1983; Allen and Holmes 1986). However, there is common consensus that phosphorylation alters the net electrical charge of the complex at the membrane surface, causing a change leading to electrostatic repulsion that overcomes forces holding neighbouring LHC II complexes at a particular angle to each other.

Garnier et al. (1990) demonstrated that, apart from reversible phosphorylation, a second feature is essential for functional LHC II state transitions: the trimeric structure has to be maintained by the anionic lipid phosphatidylglycerol (PG) containing a special trans-hexadecaonic fatty acid (PG-16:1_{trans}) at the sn2-position of the glycerol. Mutants from *Chlamydomonas reinhardtii*, where this lipid was absent, did not form trimeric LHC II complexes and were not able to carry out state transitions. The inability to state transition reactions was significantly restored when PG-16:1_{trans} was re-introduced in vivo into the thylakoid membrane. Under conditions where PG-16:1_{trans} is absent LHC II could not be stabilised in its trimeric state and no granal stacks were formed. The consequence was that LHC II complexes lost their site for phosphorylation and their ability to carry out state transitions (Dubertret et al. 1994).

These observations confirmed results from Remy et al. (1984) who demonstrated the reconstitution of LHC II complexes in vitro by simply mixing LHC II with liposomes containing PG–16:$1_{\Delta 3trans}$. Krupa et al. (1992) demonstrated that the oligomeric LHC II treated with the phospholipase PLA_2 leads to monomerisation. Later on Nussberger et al. (1993) and Hobe et al. (1994) confirmed all these results. They succeeded in reconstituting the trimeric LHC II complex from monomeric overexpressed subcomplexes. LHC-monomers were mixed with lipids and pigments and subsequently the reconstituted trimers were shown to form two-dimensional crystals identical to crystals of the native trimeric complex. PG turned out to be the essential lipid for a successful reconstitution. With these experiments, Nussberger et al. (1993) and Hobe et al. (1994) finally showed that PG is indeed a structural and functional component of the trimeric LHC II complex.

The oligomeric state of the PS II complex was controversially discussed for many years until it finally was established that PS II exists as a dimer in vivo (Boekema et al. 1995; Rögner et al. 1995; Hankamer et al. 1997). Despite the discussion about the functional oligomeric state of PS II, Barbato et al. (1992) postulated in a model that the crucial precondition for a functional D1 protein turnover mechanism in PS II is a dynamic change of the oligomeric structure of PS II. They concluded from their experimental data and from results obtained by Melis (1991) that the degradation and turnover of the D1 protein involves both PS II dimer to monomer conversion and the lateral movement of the monomer from granal to stromal regions of the thylakoid membrane. Furthermore, their model implies the release of the proximal antenna protein CP43 prior to D1 exchange. In this circumstance it is remarkable that both PS II peptides, D1 and CP43 are reversible phosphorylated, perhaps by the same redox-regulated kinase.

As found for LHC II, the main target protein for light-induced adaptation processes in PS II, the D1 peptide is reported not just to undergo reversible phosphorylation. Similarly to the LHC II peptides, the D1 protein seems to interact functionally and structurally with the anionic lipid PG (Kruse et al. 1994; Kruse and Schmid 1995). These observations are in agreement with results from Fragata et al. (1991) who stimulated photosynthetic activities by incorporating PS II complexes into PG vesicles. Later on Fragata et al. (1997) highlighted the specific role of PG for PS II activity in experiments where they demonstrated that the addition of the bivalent cation Mg^{2+} further stimulates PS II complexes after being reconstituted into PG-liposomes. Recently, reconstitution experiments with PS II complexes obtained from spinach demonstrated that PG is directly involved in PS II dimerisation (Kruse et al. 2000). Furthermore, direct evidence for the requirement of PG in PS II for photosynthesis came from Hagio et al. (2000) who obtained a *Synechocystis PCC6803* mutant that requires PG for photosynthetic growth. All these data highlight that two major factors are responsible for the oligomerisation of both, PS II and LHC II and hereby essential for the two major redox-controlled adaptation mechanisms PS II repair cycle and LHC state transitions: reversible phosphorylation and PG-protein interaction. It remains an open question whether both modifications interact.

It is obvious that, similarly to reversible phosphorylation, PG-binding to PS II and LHCII results in conformational changes by adding negative charges to the complexes. Stys and co-workers (Stys et al. 1998) suggested for

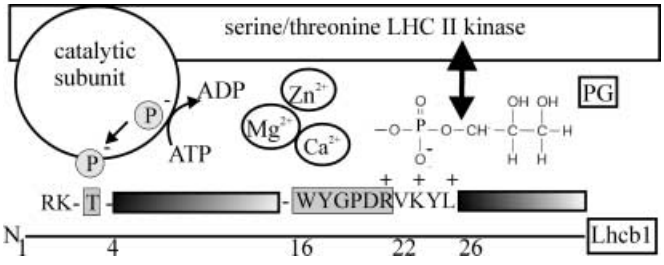

Fig. 4 Model of the *N*-terminal part of the mature Lhcb1 protein of *Spinacea oleracea* illustrating the vicinity of the phosphorylation site (threonine 3) to the putative PG-binding site close to the trimerization motif (positions 16 to 21) and a hypothetical mechanism of kinase–lipid interaction to activate the phosphorylation reaction. *PG* Phosphatidylglycerol; *P* phosphate group; + positively charged amino acids

LHC II that the PG binding site is close to the potential phosphorylation site and in functional contact to the LHC II kinase. This assumption is in good agreement with results obtained by Hobe et al. (1994). In vitro experiments elucidated the typical "trimerization motif" WYXXXR at the *N*-termini of trimeric LhcII peptides within the highly conserved sequence WYGPDR (Fig. 4). A possible role of this motif in the formation of a binding site for the anionic phospholipid phosphatidylglycerol is discussed. The group of positively charged amino acids adjacent to the trimerisation motif represents an ideal interaction site for the negatively charged PG (Fig. 4). One reason for a functional interaction between PG binding and reversible phosphorylation could be explained by the possibility that the potential kinases require phospholipids in their vicinity as it is known for certain phospholipid kinases (Rando 1998; Brill et al. 2000). Investigations on the regulation of protein kinase C demonstrated that this kinase is activated by phosphatidylserine (PS). PS can easily be substituted by other phospholipids. In addition, an interference of the negatively charged phospholipid with Ca^{2+} ions is suggested for the complete activation process of the protein kinase C. Both functional aspects of the protein kinase C activation, negatively charged phospholipids and positively charged ions, are now known to play a functional role for the LHC kinase-mediated process of state transitions. For the LHC II kinase it was demonstrated that Zn^{2+} and Mg^{2+} ions stimulate the LHC II kinase activity at low ATP concentrations (Markwell et al. 1984). Consequently, the possibility that the activation process of the LHC kinase is similar to that of phospholipid kinases cannot be excluded. A putative interaction process between lipid binding and reversible phosphorylation at the *N*-terminus of the Lhcb1 protein of spinach is summarised in a working model in Fig. 4.

Conclusions

In particular thylakoid-associated redox control plays a major role in plastidial adaptation mechanisms as answers to environmental stress. However, the origins of redox controlled signal transductions from the photosynthetic electron transport chain is still an open question. It is feasible to suggest from the new data that the LHC II kinase is regulated by different light-dependent redox processes including the $Cytb_6/f$ complex and the ferredoxin/thioredoxin system. The fact that the PS II kinase seems to be independent from this regulation pathways indicates that the overall regulation of the thylakoid-associated kinases is more complex. Although a more important role of $Cytb_6/f$ is highlighted, the importance of the redox state of the PQ pool is still in the centre of discussions. Both, PS II kinase regulation as well as gene transcription control of some photosynthetic proteins seems to be linked to the redox state of PQ. It is clear that alternative electron pathways are involved in redox control mechanisms, in particular in the dark when no electrons are provided by the light-driven reduction of $Cytb_6/f$ from the PS II complex via the PQ pool. These processes in close correlation to mitochondrial electron transport and possible chloroplast-mitochondria interactions have to be investigated in more detail.

New results concerning the role of PG-binding to both complexes, LHC II and PS II, highlight the feasibility that reversible phosphorylation processes and lipid-binding are both essential for functional state transitions and repair cycle. One major challenge in the future is to elucidate whether these two modifications are functionally linked.

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References

Allen JF (1992) Protein phosphorylation in regulation of photosynthesis. Biochim Biophys Acta 1098:275–335

Allen JF (1993) Redox control of transcription: sensors, response regulators, activators and repressors. FEBS Lett 332:203–207

Allen JF (1995) Thylakoid protein phosphorylation, state1- state2 transitions, and photosystem stoichiometry adjustment: redox control at multiple levels of gene expression. Physiol Plant 93:196–205

Allen JF, Holmes NG (1986) A general model for regulation of photosynthetic unit function by protein phosphorylation. FEBS Lett 202:175–181

Allen JF, Nilsson A (1997) Redox signalling and the structural basis of regulation of photosynthesis by protein phosphorylation. Physiol Plant 100:863–868

Allen JF, Bennett J, Steinback KE, Arntzen CJ (1981) Chloroplast protein phosphorylation couples plastoquinone redox state to distribution of excitation energy between photosystems. Nature 291:21–25

Allen JP, Williams JC (1998) Photosynthetic reaction centres. FEBS Lett 438:5–9

Anderson JM (1986) Photoregulation of the composition, function and structure of thylakoid membranes. Annu Rev Plant Physiol 37:93–136

Anderson JM, Price GD, Chow WS, Hope AB, Badger MR (1997) Reduced levels of cytochrome bf complex in transgenic tobacco leads to marked photochemical reduction of the plastoquinone pool, without significant change in acclimation to irradiance. Photosynth Res 53:215–227

Andersson B, Barber J (1994) Composition, organization and dynamics of thylakoid membranes. In: Bittar EE (ed) Advances in molecular and cell biology, vol 10. JAI Press, Greenwich, UK, pp 1–53

Aro EM, Virgin I, Andersson B (1993) Photoinhibition of photosystem II. Inactivation, protein damage and turnover. Biochim Biophys Acta 1143:113–134

Barbato R, Friso G, Rigoni F, Dalla Vecchia F, Giacometti GM (1992) Structural changes and lateral redistribution of photosystem II during donor side photoinhibition of thylakoids. J Cell Biol 119:325–335

Barber J (1982) Influence of surface charges on thylakoid structure and function. Annu Rev Plant Physiol 33:261–295

Barber J (1986) Organization and dynamics of protein complexes within the chloroplast thylakoid membrane. Biochem Soc Trans 14:1–4

Barber J, Andersson B (1992) Too much of a good thing: light can be bad for photosynthesis. Trends Biochem Sci 17:61–66

Bennett J (1979) The protein kinase of the thylakoid membrane is light-dependent. FEBS Lett 103:342–344

Bennett J (1991) Protein phosphorylation in green plant chloroplasts. Annu Rev Plant Physiol Plant Mol Biol 42:281–311

Boekema EJ, Hankamer B, Bald D, Kruip J, Nield J, Barber J, Rögner M (1995) Supramolecular structure of the photosystem II complex from green plants and cyanobacteria. Proc Natl Acad Sci USA 92:175–179

Boekema EJ, Roon H van, Breemen JFL van, Dekker JP (1999) Supramolecular organization of photosystem II and its light-harvesting antenna in partially solubilized photosystem II membranes. Eur J Biochem 266:444–452

Boekema EJ, Breemen JFL van, Roon H van, Dekker JP (2000a) Conformational changes in photosystem II supercomplexes upon removal of extrinsic subunits. Biochemistry 39:12907–12915

Boekema EJ, Breemen JFL van, Roon H van, Dekker JP (2000b) Arrangement of photosystem II supercomplexes in crystalline macrodomains within the thylakoid membrane of green plant chloroplasts. J Mol Biol 301:1123–1133

Bonaventura C, Myers J (1969) Fluorescence and oxygen evolution *from Chlorella pyrenoidosa*. Biochim Biophys Acta 189:366–386

Brill JA, Hime GR, Scharer-Schuksz M, Fuller MT (2000) A phospholipid kinase regulates actin organization and intercellular bridge formation during germline cytokinesis. Development 127:3855–3864

Carlberg I, Andersson B (1996) Phosphatase activities in spinach thylakoid membranes – effectors, regulation and location. Photosynth Res 47:145–156

Danon A, Mayfield SP (1994) Light-regulated translation of chloroplast messenger RNAs through redox potential. Science 266:1717–1719

Dubertret G, Mirshahi A, Mirshahi M, Gerard-Hirne C, Tremolieres A (1994) Evidence from in vivo manipulations of lipid composition in mutants that the delta 3-trans-hexadecaenoic acid-containing phosphatidylglycerol is involved in the biogenesis of the light-harvesting chlorophyll *a*/*b*-protein complex of *Chlamydomonas reinhardtii*. Eur J Biochem 226:473–482

Ebbert V, Godde D (1994) Regulation of thylakoid protein phosphorylation in intact chloroplasts by the activity of kinases and phosphatases. Biochim Biophys Acta 1187:335–346

Emlyn-Jones D, Ashby MK, Mullineaux CW (1999) A gene required for the regulation of photosynthetic light-harvesting in the cyanobacterium *Synechocystis 6803*. Mol Microbiol 33:1050–1058

Fleischmann MM, Ravanel S, Delosme R, Olive J, Zito F, Wollman FA, Rochaix JD (1999) Isolation and characterization of photoautotrophic mutants of *Chlamydomonas reinhardtii* deficient in state transition. J Biol Chem 274:30987–94

Fork DC, Satoh K (1986) The control by state transitions of the distribution of excitation energy in photosynthesis. Annu Rev Plant Physiol Plant Mol Biol 37:335–361

Foyer CH (1996) Oxygen processing in photosynthesis. Biochem Soc Trans 24:427–433

Fragata M, Strzalka K, Nénonéné EK (1991) Magnesium chloride-induced reversal of oxygen evolution decay in photosystem II particles incubated with phosphatidylglycerol. Vesicles at high lipid photosystem II ratio. J Photochem Photobiol 11:329–342

Fragata M, Nenonene EK, Maire V, Gabashvili IS (1997) Structure of the phosphatidylglycerol-photosystem II complex studied by FT-IR spectroscopy. Mg(II) effect on the polar head group of phosphatidylglycerol. J Mol Struct 405:151–158

Gal A, Zer H, Ohad I (1997) Redox-controlled thylakoid protein phosphorylation. News and views. Physiol Plant 100:869–885

Garnier J, Wu B, Maroc J, Guyon D, Trémolières A (1990) Restoration of both an oligomeric form of the light-harvesting antenna CPII and a fluorescence state II-state I transition by Δ^3-trans-hexadecanoic acid-containing phosphatidylglycerol, in cells of a mutant of *Chlamydomonas reinhardtii*. Biochim Biophys Acta 1020:153–162

Hagio M, Gombos Z, Varkonyi Z, Masamoto K, Sato N, Tsuzuki M, Wada H (2000) Direct evidence for requirement of phosphatidylglycerol in photosystem II of photosynthesis. Plant Physiol 124:795–804

Hamel P, Olive J, Pierre Y, Wollman FA, Vitry C de (2000) A new subunit of cytochrome b6f complex undergoes reversible phosphorylation upon state transition. J Biol Chem 275:17072–17079

Hankamer B, Nield J, Zheleva D, Boekema EJ, Barber J (1997) Isolation and biochemical characterisation of monomeric and dimeric PS II complexes from spinach and their relevance to the organisation of photosystem II in vivo. Eur J Biochem 243:422–429

Hartwig H-D, Allen JF, Paulsen H, Race HL (1998) Truncated recombinant light harvesting complex II proteins are substrates for a protein kinase associated with photosystem II core complexes. FEBS Lett 435:101–104

Hobe S, Prytulla S, Kühlbrandt W, Paulsen H (1994) Trimerization and crystallization of reconstituted chlorophyll *a*/*b* complex. EMBO J 13:3423–3429

Horton P, Allen JF, Black MT, Bennett J (1981) Regulation of phosphorylation of chloroplast membrane polypeptides by the redox state of plastoquinone. FEBS Lett 125:193–196

Horton P, Ruban AV, Walters RG (1994) Regulation of light harvesting in green plants: indication by non-photochemical quenching of chlorophyll fluorescence. Plant Physiol 106:415–420

Ikeuchi M, Plumley, FG, Inoue Y, Schmidt GW (1987) Phosphorylation of photosystem II components, CP43 apoprotein, D1, D2 and 10–11 kilodalton protein in chloroplast thylakoids of higher plants. Plant Physiol 85:638–642

Keren N, Ohad I (1999) State transition and photoinhibition. In: Rochaix JD; Goldschmidt-Clermont M, Merchant S (eds) Molecular biology of the chloroplast and mitochondria in *Chlamydomonas*. Kluwer, Dordrecht, pp 569–596

Koivuniemi A, Aro EM, Andersson B (1995) Degradation of the D1- and D2- proteins of photosystem II in higher plants is regulated by reversible phosphorylation. Biochemistry 34:16022–16029

Krause GH, Weiss E (1991) Chlorophyll fluorescence and photosynthesis: the basics. Annu Rev Plant Phsyiol Plant Mol Biol 42:313–349

Krebs E (1994) The growth of research on protein phosphorylation. Trends Biochem Sci 19:439–440

Krupa Z, Williams JP, Khan MU, Huner NPA (1992) The role of acyl lipids in reconstitution of lipid-depleted light-harvesting complex II from cold-hardened and nonhardened rye. Plant Physiol 100:931–938

Kruse O, Schmid GH (1995) The role of phosphatidylglycerol as a functional effector and membrane anchor of the D1-core peptide from photosystem II-particles of the cyanobacterium *Oscillatoria chalybea*. Z Naturforsch 50c:380–390

Kruse O, Radunz A, Schmid GH (1994) Phosphatidylglycerol and β-carotene bound onto the D1-core peptide of photosystem II in the filamentous cyanobacterium *Oscillatoria chalybea*. Z Naturforsch 49c:115–124

Kruse O, Zheleva D, Barber J (1997) Stabilization of photosystem II dimers by phosphorylation: implication for the regulation of the turnover of D1 protein. FEBS Lett 408:276–280

Kruse O, Nixon PJ, Schmid GH, Mullineaux CW (1999) Isolation of state transition mutants of *Chlamydomonas reinhardtii* by fluorescence video imaging. Photosynth Res 61:43–51

Kruse O, Hankamer B, Konczak C, Gerle C, Morris E, Radunz A, Schmid GH, Barber J (2000) Phosphatidylglycerol is involved in the dimerization of photosystem II. J Biol Chem 275:6509–6514

Kühlbrandt W, Downing KH (1989) Two-dimensional structure of plant light-harvesting complex at 3.7 A resolution by electron crystallography. J Mol Biol 207:823–828

Kühlbrandt W, Wang DN (1991) Three-dimensional structure of plant light-harvesting complex determined by electron crystallography. Nature 350:130–134

Li XP, Björkman O, Shih C, Grossman AR, Rosenquist M, Jansson S, Niyogi KK (2000) A pigment-binding protein essential for regulation of photosynthetic light harvesting. Nature 403:391–395

Link G (1999) Redox regulation of photosynthetic genes. In: Govindjee (ed) Advances in photosynthesis, vol 7. Kluwer, Dordrecht, pp 1–52

Lunde C, Jensen PE, Haldrup A, Knoetzel J, Scheller HV (2000) The PSI-H subunit of photosystem I is essential for state transitions in plant photosynthesis. Nature 408:613–615

Marder JB, Telfer A, Barber J (1988) The D1 polypeptide subunit of photosystem II reaction centre has a phosphorylation site at its amino-terminus. Biochim Biophys Acta 932:362–365

Markwell JP, Baker NR, Bradbury M, Thornber JP (1984) Use of zinc ions to study thylakoid protein phosphorylation and the state 1-state 2 transition in vitro. Plant Physiol 74:348–354

Melis A (1991) Dynamics of photosynthetic membrane composition and function. Biochim Biophys Acta 1058:87–101

Michel HP, Hunt DF, Shabarkowitz J, Bennett J (1988) Tandem mass spectroscopy reveals that three photosystem II proteins of spinach chloroplasts contain *N*-acetyl-*O*-phosphothreonine at their NH_2-termini. J Biol Chem 263:1123–1130

Mullineaux CW, Tobin M J, Jones GR (1997) Mobility of photosynthetic complexes in thylakoid membranes. Nature 390:421–424

Murata N (1969) Control of excitation transfer in photosynthesis. I. Light-induced change of chlorophyll *a* fluorescence in *Porphyridium cruentum*. Biochim Biophys Acta 172:242–251

Nield J, Orlova EV, Morris EP, Gowen B, Heel M van, Barber J (2000) 3D map of the plant photosystem II supercomplex obtained by cryoelectron microscopy and single particle analysis. Nat Struct Biol 7:44–47

Nilsson A, Stys D, Drakenberg T, Spangfort MD, Forsen S, Allen, JF (1997) Phosphorylation controls the three-dimensional structure of plant light harvesting complex II. J Biol Chem 272:18350–18357

Niyogi KK, Björkman O, Grossman AR (1997) *Chlamydomonas* xanthophyll cycle mutants identified by video imaging of chlorophyll fluorescence quenching. Plant Cell 9:1369–1380

Nussberger S, Dörr K, Wang DN, Kühlbrandt W (1993) Lipid–protein interactions in crystals of plant light-harvesting complex. J Mol Biol 234:347–356

Paulsen H (1995) Chlorophyll *a*/*b*-binding proteins. Photochem Photobiol 62:367–382

Pfannschmidt T, Nilsson A, Allen J (1999) Photosynthetic control of chloroplast gene expression. Nature 397:625–628

Pursiheimo S, Rintamäki E, Baena-Gonzalez E, Aro E-M (1998) Thylakoid protein phosphorylation in evolutionally divergent species with oxygenic photosynthesis. FEBS Lett 423:178–182

Race HL, Hind G (1996) A protein kinase in the core of photosystem. II. Biochemistry 35:13006–13010

Rando RR (1998) Regulation of protein kinase C activity by lipids. FASEB J 2:2348–2355

Reilly PL, Woska JR Jr, Jeanfavre DD, McNally E, Rothlein R, Bormann BJ (1995) The native structure of intercellular adhesion molecule-1 (ICAM-1) is a dimer: correlation with binding to LFA-1. J Immunol 155:529–532

Reinbothe S, Reinbothe C (1996) The regulation of enzymes involved in chlorophyll biosynthesis. Eur J Biochem 237:323–343

Remy R, Tremolieres A, Ambard-Bretteville F (1984) Formation of oligomeric light-harvesting chlorophyll *a*/*b* protein by interaction between its monomeric form and liposomes. Photobiochem Photobiophys 7:267–276

Rintamäki E, Kettunen R, Aro EM (1996) Differential D1 dephosphorylation in functional and photodamaged photosystem-II centers: dephosphorylation is a prerequisite for degradation of damaged D1. J Biol Chem 271:14870–14875

Rintamäki E, Martinsuo P, Pursiheimo S, Aro EM (2000) Cooperative regulation of light-harvesting complex II phosphorylation via the plastoquinol and ferredoxin-thioredoxin system in chloroplasts. Proc Natl Acad Sci USA 97:11644–11649

Rögner M, Boekema EJ, Barber J (1995) How does photosystem 2 split water? The structural basis of efficient energy conversion. Trends Biochem Sci 21:44–49

Schaller GE, Ladd AN, Lanahan MB, Spanbauer JM, Bleecker AB (1995) The ethylene response mediator ETR1 from *Arabidopsis* forms a disulfide-linked dimer. J Biol Chem 270:12526–12530

Scheibe R (1991) Redox-modulation of chloroplast enzymes: a common principle for individual control. Plant Physiol 96:1–3

Schuster G, Dewit M, Staehelin LA, Ohad I (1986) Transient inactivation of the thylakoid photosystem II light-harvesting protein kinase and concomitant changes in intramembrane particle sizes during photoinhibition of *Chlamydomonas reinhardtii*. J Cell Biol 103:71–80

Silverstein T, Cheng L, Allen JF (1993) Chloroplast thylakoid protein phosphatase reactions are redox-independent and kinetically heterogeneous. FEBS Lett 334:101–105

Smith TA, Kohorn BD (1991) Direct selection for sequences encoding proteases of known specificity. Proc Natl Acad Sci USA 88:10989–10992

Snyders S, Kohorn BD (1999) TAKs: thylakoid membrane protein kinases associated with energy tranduction. J Biol Chem 274:9137–9140

Staehelin LA, Arntzen CJ (1983) Regulation of chloroplast membrane function: protein phosphorylation changes the spatial organization of membrane components. J Cell Biol 97:1327–1337

Stys D, Veverka V, Motejl M, Hrabal R (1998) Identification and role of the lipid binding site on light-harvesting complex II. In: Garab G (ed) Photosynthesis: mechanisms and effects. Kluwer, Dordrecht

Sundby C, Larson UK, Henrysson T (1989) Effects of bicarbonate on thylakoid protein phosphorylation. Biochim Biophys Acta 975:277–282

Telfer A, Allen JF, Barber J, Bennett J (1983) Thylakoid protein phosphorylation during state1-state2 transitions in osmotically shocked pea chloroplasts. Biochim Biophys Acta 722:176–181

Vener AV, Kan PJM van, Gal A, Andersson B, Ohad I (1995) Activation/deactivation cycle of redox-controlled thylakoid protein phosphorylation. J Biol Chem 270:25225–25232

Vener AV, Kan PJM van, Rich PR, Ohad I, Andersson B (1997) Plastoquinol at the Qo-site of reduced cytochrome bf mediates signal transduction between light and protein phosphorylation: thylakoid protein kinase deactivation by a single turnover flash. Proc Natl Acad Sci USA 94:1585–1590

Vener AV, Rokka A, Fulgosi H, Andersson B, Herrmann RG (1999) A clophilin-regulated PP2A-like protein phosphatase in thylakoid membranes of plant chloroplasts. Biochemistry 38:14955–14965

Vitry C de, Finazzi G, Baymann F, Kallas T (1999) Analysis of the nucleus-encoded and chloroplast-targeted Rieske protein by classic and site-directed mutagenesis of *Chlamydomonas*. Plant Cell 11:2031–44

Yu CA, Xia JZ, Kachurin AM, Yu L, Xia D, Kim H, Deisenhofer J (1996) Crystallization and preliminary structure of beef heart mitochondrial cytochrome-bc-1 complex. Biochim Biophys Acta 1275:47–53

Zito F, Finazzi G, Delosme R, Nitschke W, Picot D, Wollman FA (1999) The Qo site of cytochrome b6f complexes controls the activation of the LHCII kinase. EMBO J 18:2961–2969