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Oxidation–reduction signalling components in regulatory pathways of state transitions and photosystem stoichiometry adjustment in chloroplasts

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ABSTRACT

State transitions and photosystem stoichiometry adjustment are two oxidation–reduction (redox)-regulated acclimatory responses in photosynthesis. State transitions are short-term adaptations that, in chloroplasts, involve reversible post-translational modification by phosphorylation of light-harvesting complex II (LHC II). Photosystem stoichiometry adjustments are long-term responses involving transcriptional regulation of reaction centre genes. Both responses are initiated by changes in light quality and are regulated by the redox state of plastoquinone (PQ). The LHC II kinase involved in the state 2 transition is a serine/threonine kinase known as STT7 in *Chlamydomonas*, and as STN7 in *Arabidopsis*. The phospho-LHC II phosphatase that produces the state 1 transition is a PP2C-type protein phosphatase currently termed both TAP38 and PPH1. In plants and algae, photosystem stoichiometry adjustment is governed by a modified two-component sensor kinase of cyanobacterial origin – chloroplast sensor kinase (CSK). CSK is a sensor of the PQ redox state. Chloroplast sigma factor 1 (SIG1) and plastid transcription kinase (PTK) are the functional partners of CSK in chloroplast gene regulation. We suggest a signalling pathway for photosystem stoichiometry adjustment. The signalling pathways of state transitions and photosystem stoichiometry adjustments are proposed to be distinct, with the two pathways sensing PQ redox state independently of each other.

Key-words: chloroplast genome; chloroplast sensor kinase; CoRR hypothesis; light-harvesting complex II; plastoquinone; protein phosphatase; redox sensor; redox signalling; serine/threonine kinase; two-component systems.

INTRODUCTION

Oxidation–reduction (redox) reactions are a major class of chemical reactions in nature. They involve transfer of electrons between chemical species, with oxidation being loss of electrons by an electron donor and reduction being gain of electrons by an electron acceptor. Redox state refers to the state of oxidation or reduction of an electron carrier. Redox reactions abound in biological systems. They are at the heart of energy conversion, thereby underpinning life on Earth. In a process termed redox control, redox states of atoms or molecules act as signals to regulate a wide variety of biological phenomena (Pfannschmidt, Allen & Oelmuller 2001a; Buchanan & Balmer 2005; Foyer & Noctor 2009). 'Redox signalling' refers to the signalling processes that connect redox signals to redox-regulated responses.

In eukaryotes, redox reactions of photosynthesis and respiration are compartmentalized in chloroplasts and mitochondria, respectively. Chloroplasts are sites of photosynthesis, the conversion of light energy into chemical energy, in plants and algae. Bound by two or more envelope membranes, chloroplasts contain an intricate membranous system of flattened vesicles known as thylakoids. Traversing thylakoid membranes are two photosystems, photosystem II (PSII) and photosystem I (PSI). Each photosystem consists of a peripheral array of light-harvesting antenna pigments and an internal core of its reaction centre, where the light-driven redox chemistry takes place. Because of their differing pigment compositions, the two photosystems have different light absorption properties. Absorption of light by PSII is restricted to shorter wavelengths than is the case for PSI (Myers 1971). A 'red drop' in quantum yield of oxygen by photosynthesis occurs as the wavelength of a single incident light exceeds approximately 660 nm (the precise value depends on the species under investigation) (Emerson & Lewis 1943). The wavelength at which the quantum yield begins to fall is extended to beyond 700 nm when a second source of light of constant wavelength, shorter than 660 nm, is also present – the effect of the shorter-wavelength, second light is to postpone the red drop of the first (Emerson 1958). Between these two wavelengths, there is therefore an enhancement of quantum yield, such that the quantum yield from both light sources acting together is greater than the sum of the separate quantum yields each obtained from one of the wavelengths acting alone (Emerson 1958; Myers 1971; Clayton 1980).

This phenomenon of 'Emerson enhancement' is explained by the requirement for two separate photosystems, I and II, acting in series (Hill & Bendall 1960; Duysens, Amesz & Kamp 1961): the red drop is the long-wave limit of one photosystem, PSII, and it is therefore the long-wave limit of the whole process of photosynthesis if a

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single source of light is present. At wavelengths beyond the red drop, the second, supplementary light source then supplies energy to the otherwise rate-limiting PSII, and so the action spectrum of complete photosynthesis is extended to the greater wavelength at which PSI becomes unable to convert light energy. In simplified terms, PSII is the short-wave photosystem, while PSI is the long-wave photosystem whose action spectrum extends into the far-red. In oxygenic photosynthesis, the two photosystems are connected in series by cytochromes b_6 and f (Hill & Bendall 1960). This series connection enables the photosystems to extract electrons from water, using the energy from sunlight. The electrons are then passed through a chain of electron carriers, and finally via NADPH to CO_2 to yield triose phosphates in the reductive pentose phosphate pathway or 'Calvin cycle'.

Because of optical filtering effects in natural habitats, the light that plants and algae absorb is often enriched in particular wavelengths of light that favour one photosystem over the other. Shading by other plants, for example, produces an enrichment in long-wave components that favour PSI. Conversely, in aquatic ecosystems, longer wavelengths are absorbed preferentially as light passes though water, and so transmitted light favours PSII. There are also ecological and physiological variations between and within species that change metabolic demand for ATP from photophosphorylation. Because the two photosystems are connected in series, their rates of electron transport, or current, must be equal for purely linear, non-cyclic electron transport. Light energy is wasted if it is absorbed and transferred to either reaction centre at a rate in excess of its photochemical conversion in electron transport. Thus, for efficient energy conversion, the two photosystems, transferring electrons at equal rates, should absorb and convert light energy also at equal rates. In fact, the contribution to total ATP synthesis of cyclic electron transport (Arnon, Allen & Whatley 1954), through PSI alone (Heber & Walker 1992; Bendall & Manasse 1995; Allen 2003a), means that the two photosystems should absorb and transfer excitation energy in proportion to their rates of primary photochemistry. The contribution of PSI cyclic photophosphorylation, nominally 15% of ATP synthesis in the steady state in C3 plants (Allen 1984b, 2003a), itself varies according to metabolic demand for ATP relative to NADPH (Allen & Bennett 1981; Allen 1983b; Heber & Walker 1992), while the relative ATP requirement is even higher in C_4 and CAM plants than in C_3 plants. For maximal efficiency, it is necessary for absorbed excitation energy to be capable of redistribution between the photosystems under these circumstances, too (Allen 1984a,b; Finazzi *et al*. 2002). Plants and algae counter any disproportion between the two photosystems by two simple tricks they acquired early in their evolution – state transitions and photosystem stoichiometry adjustment.

State transitions were first proposed to explain light-induced changes in the yield of room-temperature chlorophyll fluorescence and accompanying changes in the quantum yield of oxygen evolution (Bonaventura & Myers 1969; Murata 1969). 'States' in state transitions refer to two light states that photosynthetic systems adopt, with 'state 2' denoting an adaptive state to light that is preferentially absorbed by PSII (light 2), and 'state 1', to a light that is preferentially absorbed by PSI (light 1). Transitions between these two states are called the state 1 and state 2 transitions. The two states, 1 and 2, are acclimatory states in which total absorbed excitation energy tends to become distributed equally between the two photosystems (Myers 1971).

The Z-scheme or two-light reaction model requires that the rates of electron transport at the two reaction centres are equal (Hill & Bendall 1960). However, the added requirement for a varying contribution of PSI-driven cyclic photophosphorylation must be taken into consideration. Thus, the relative quantity of the two photosystems can vary in inverse proportion to their rates of energy conversion. Indeed, it was found that photosystem stoichiometry varies greatly according to species, with values for the ratio of PSI to PSII reaction centres ranging from 0.43 in some cyanobacteria to 3.3 in green algae and plants, while photosystem stoichiometry within a single species also varies, primarily in response to changes in the wavelength of light (Melis, Mullineaux & Allen 1989; Chow, Melis & Anderson 1990; Murakami, Kim & Fujita 1997). Both state transitions and photosystem stoichiometry adjustment increase the quantum efficiency of photosynthesis at limiting light intensity. The nature and significance of these two processes have been reviewed previously (Allen 1983a, 1995, 2003b; Williams & Allen 1987; Wollman 2001; Rochaix 2007; Lemeille & Rochaix 2010). The present review focuses mainly on the associated redox sensory systems and regulatory pathways that have recently become more clearly elucidated.

SIGNALS AND SENSORS IN CHLOROPLASTS

State transitions were first described in 1969, and it took nearly 12 years to discover the signal that regulates this important acclimatory response, which fine-tunes photosynthetic efficiency to different light environments. A light-regulated chloroplast protein kinase (Bennett 1977, 1979a,b) was shown to phosphorylate light-harvesting complex II (LHC II), which in turn changed the light-harvesting property of isolated thylakoids (Bennett, Steinback & Arntzen 1980). Using chemical electron donors, acceptors and electron transport inhibitors, which act at specific points of the photosynthetic electron transport pathway, it was shown that light *per se* is not the direct signal that activates the LHC II kinase, but instead the redox state of an electron carrier located between the two photosystems – plastoquinone (PQ) – was revealed as the actual signal (Allen & Bennett 1981; Allen *et al*. 1981). This implied that different light conditions, which preferentially excite one photosystem over the other, affect the redox state of the PQ pool, as it becomes more reduced when PSII is preferentially excited, and more oxidized when PSI is favoured. Thus, a relatively more reduced PQ pool induces the state 2 transition by activation of an LHC II kinase. As LHC II becomes phosphorylated, phospho-LHC II moves from PSII to PSI, increasing the absorption cross-section of

PSI at the expense of PSII. Conversely, in PQ oxidizing conditions, the LHC II kinase is inactive and the state 1 transition is promoted by the dephosphorylation of LHC II by the phospho-LHC II phosphatase. Dephosphorylated LHC II returns to PSII from PSI, and thereby redistributes absorbed excitation energy in favour of PSII. PQ redox-regulated reversible phosphorylation of LHC II is now generally accepted as an essential component of the molecular mechanism of state transitions in plants.

As soon as LHC II kinase activity was identified, the enzyme itself was sought (Lin, Lucero & Racker 1982; Lucero, Lin & Racker 1982; Coughlan & Hind 1986; Gal *et al*. 1992; Hind, Marshak & Coughlan 1995; Sokolenko *et al*. 1995; Race & Hind 1996; Tullberg, Håkansson & Race 1998; Snyders & Kohorn 1999, 2001). The actual LHC II kinase was ultimately identified by a genetic rather than by a biochemical approach. By screening for state transition mutants in the unicellular green alga *Chlamydomonas reinhardtii*, a serine/threonine protein kinase called STT7 (state transition-deficient mutant 7) was identified (Depege, Bellafiore & Rochaix 2003). *Chlamydomonas* cells deficient in STT7 cannot phosphorylate LHC II, are unable to undergo the state 2 transition and are locked in state 1. Inactivation of the *Arabidopsis* homolog of the *STT7* gene, *STN7*, produces a similar state transition-deficient phenotype (Bellafiore *et al*. 2005). Additional biochemical and genetic analysis of STT7/STN7 demonstrated its role as the LHC II kinase (Bonardi *et al*. 2005; Lemeille *et al*. 2009, 2010). Topological studies of STT7 suggest a single transmembrane domain, a lumen-located N-terminal stretch of residues and a stroma-exposed kinase domain (Lemeille *et al*. 2009). Co-immunoprecipitation analysis revealed direct interaction of STT7 with LHC II, with the Rieske iron–sulphur protein of the cytochrome b_6-*f* complex, and with PSI (Lemeille *et al*. 2009). These results are taken as evidence to suggest that STT7/STN7 alone is sufficient to couple the PQ pool redox state to the phosphorylation of LHC II, and that no other protein kinases are directly involved in state transitions. An earlier study found aberrant chlorophyll fluorescence and chlorophyll *a*/*b* ratios in *stn7* mutants grown under light conditions that favour individual photosystems (Bonardi *et al*. 2005). Based on these results, a role for STN7 in photosystem stoichiometry adjusments has been suggested (Bonardi *et al*. 2005). However, analysis of the *Chlamydomonas* homolog of STN7 did not confirm this phenotype for *stn7* (Rochaix 2007). Moreover, in *stn7* mutants, the expression of chloroplast reaction centre genes and nuclear photosynthetic genes was similar to that in wild type (Bonardi *et al*. 2005; Tikkanen *et al*. 2006). It is therefore likely that the function of STT7/STN7 is confined only to state transitions. In high-light conditions, STN7 is inactive, as it is inhibited by the ferredoxin–thioredoxin system (Rintamaki *et al*. 2000; Bonardi *et al*. 2005). This inhibition of the LHC II kinase in high-light conditions underlines the nature of state transitions as a low-light phenomenon. The impaired growth phenotype and the reduced seed setting of *Arabidopsis stn7* mutants growing under fluctuating light quality are further indicators of the adaptive significance of state transitions (Bellafiore *et al*. 2005; Tikkanen *et al*. 2006; Frenkel *et al*. 2007).

A paralogue of *STN7* was found in both *Chlamydomonas* and *Arabidopsis*, and is known as *STL1*/*STN8* (Depege *et al*. 2003; Bellafiore *et al*. 2005; Bonardi *et al*. 2005). *Stn8* mutants showed normal state transitions, but were affected in PSII core phosphorylation (Bonardi *et al*. 2005). PSII core proteins D1, D2 and CP43 were not phosphorylated in *stn8* mutants in either low-light or high-light conditions, suggesting that the main function of STN8 is phosphorylation of PSII core proteins (Bonardi *et al*. 2005). PSII core proteins appear to become phosphorylated in high light, and are not particularly selective for PSII or PSI-specific light of rate-limiting intensity, as is LHC II phosphorylation in state transitions (Harrison & Allen 1991). Unlike STN7, STN8 seems to be active in high-light conditions (Bonardi *et al*. 2005). The real significance of PSII core phosphorylation is still not understood. However, the originally suggested function in D1 repair cycle has garnered support recently (Tikkanen *et al*. 2008; Fristedt *et al*. 2009). A role for the phosphorylation in decreasing co-operativity among PSII units has also been suggested (Allen & Holmes 1986). The decreased co-operativity may relieve excitation pressure on PSII.

Thylakoid-associated kinases (TAKs) are a family of three serine threonine kinases identified in the course of the search for the LHC II kinase (Snyders & Kohorn 1999, 2001). *Arabidopsis* plants that lack TAKs exhibit a partial deficiency in state transitions and increased sensitivity to high-light conditions (Snyders & Kohorn 2001). However, these kinases do not have any homologs in *Chlamydomonas*; it is therefore likely that TAKs have some plant-specific functions (Lemeille & Rochaix 2010).

Like state transitions, photosystem stoichiometry adjustment is an acclimatory response to changes in light quality, but it is long term in scope, taking hours and days to complete instead of minutes, and it can also result in larger adjustments in light-harvesting antenna size. It was generally considered that photosystem stoichiometry adjustments involve regulation of *de novo* synthesis of chlorophyll *a* and chlorophyll *a*-binding proteins as precursors to new photosystems (Murakami *et al*. 1997), but how this was done remained unknown for nearly a decade. By using photosystem-specific light conditions and electron transport inhibitors that limit the rate of photosynthetic electron transport at specific points of the electron transport pathway, it was found that the redox state of PQ pool, the point of regulatory control of the LHC II kinase, also controls the transcription of chloroplast genes that encode the core reaction centre apoproteins of the photosystems (Pfannschmidt, Nilsson & Allen 1999a; Pfannschmidt *et al*. 1999b; Tullberg *et al*. 2000; Puthiyaveetil & Allen 2008). This PQ-regulated chloroplast reaction centre gene transcription is the initiation step of the overall molecular mechanism of photosystem stoichiometry adjustment. In light 1, the PQ pool becomes more oxidized, repressing PSI gene transcription and/or activating PSII gene transcription. In

the complementary light 2 conditions, the transcription of PSI genes is activated and/or PSII genes are repressed. These differential effects on chloroplast reaction centre gene transcription, coupled with their translation, influence the rate of *de novo* synthesis of photosystems, and therefore their relative abundance or stoichiometry in light 1 and light 2 (Pfannschmidt *et al*. 1999a,b). The demonstration that state transitions and photosystem stoichiometry adjustment are controlled by the same regulatory signal – PQ pool redox state – revealed the analogous nature of these two responses. State transitions operate primarily at the post-translational level and stoichiometry adjustments at the transcriptional level of gene regulation, with both responses serving to compensate for imbalance in excitation energy caused by changes in the quality of light (Allen 1992, 1995; Allen & Pfannschmidt 2000; Pfannschmidt 2003; Rochaix 2011).

A two-component system was proposed as the basis of PQ-controlled gene regulation in chloroplasts. This proposal was a prediction of the hypothesis that redox regulation of gene expression has been retained from the endosymbiont ancestors of chloroplasts and mitochondria, and accounts for the retention, in evolution, of their distinct cytoplasmic genomes (Allen 1992, 1993a). This hypothesis has been termed the 'CoRR' hypothesis, an abbreviation of co-location for redox regulation (Allen 2003c,d). Two-component systems are a ubiquitous family of two cognate signal transduction proteins found in prokaryotes, including cyanobacteria (Stock, Robinson & Goudreau 2000; Wuichet, Cantwell & Zhulin 2010). The two components are a sensor histidine kinase, which becomes autophosphorylated on histidine when a specific environmental change occurs, and a response regulator that contains an aspartate residue to which the phosphate group is transferred, thereby activating an appropriate response.

Chloroplast sensor kinase (CSK) is a two-component sensor kinase of chloroplasts, first described in 2008 as a soluble stromal protein and the product of a nuclear gene (Puthiyaveetil *et al*. 2008). CSK has homologs in all major lineages of photosynthetic eukaryotes. In accordance with the CoRR hypothesis, a CSK homolog is also found in cyanobacteria, indicating the cyanobacterial ancestry of this chloroplast protein (Puthiyaveetil *et al*. 2008; Puthiyaveetil & Allen 2009), and, by implication, conservation of its regulatory function through the transition from endosymbiont to cytoplasmic organelle. CSK contains a sensor domain and a kinase domain, features typical of all sensor histidine kinases. The sensor domain is a GAF domain (named after vertebrate cGMP-specific phosphodiesterase, cyanobacterial adenylate cyclase and the bacterial formate hydrogen lyase transcription activator FhlA). GAF sensor domains are structurally related to the PAS (named after the PER, ARNT and SIM proteins) redox sensor domains (Taylor & Zhulin 1999; Ho, Burden & Hurley 2000), and are also known for their redox-sensing function (Kumar *et al*. 2007). The kinase domain of CSK shows variation with respect to the conserved histidine residue that undergoes autophosphorylation (Puthiyaveetil *et al*. 2008; Puthiyaveetil & Allen 2009). Plant and green algal CSKs have lost the conserved histidine residue, while red algal, diatom, brown algal and cyanobacterial CSKs still possess it. The loss of histidine residue correlates with an altered autokinase activity in plant CSK (Puthiyaveetil & Allen 2008; Puthiyaveetil *et al*. 2008).

The inactivation of the *CSK* gene in *Arabidopsis* causes impaired reaction centre gene transcription and photosystem stoichiometry adjustment (Puthiyaveetil *et al*. 2008). More specifically, *CSK* knock-out mutants cannot repress PSI transcription in light 1. Concerning the nature of the signal sensed by CSK, it is found that oxidized PQ promotes autophosphorylation activity in over-expressed and purified CSK (Ibrahim, Puthiyaveetil & Allen, unpublished data). It thus becomes evident that although LHC II kinase and CSK act on the same PQ regulatory signal, two distinct forms of PQ – reduced and oxidized – activate these kinases, respectively. A pleiotropic effect of CSK inactivation on room temperature fluorescence is observed in an *Arabidopsis CSK* mutant, while direct involvement of CSK in state transitions is unlikely as judged by low-temperature chlorophyll fluorescence emission spectra and ^{32}P-labelling of LHC II in isolated thylakoids (Puthiyaveetil *et al*., submitted). These results suggest that STN7 and CSK are two independent sensors of the redox state of the PQ pool, initiating two distinct, but analogous, acclimatory responses – state transitions and photosystem stoichiometry adjustments.

GETTING SIGNALS OUT OF THE THYLAKOID MEMBRANE

The signal that initiates state transitions and photosystem stoichiometry adjustment is the redox state of the PQ pool. PQ is a mobile electron carrier that carries electrons from PSII to Cyt b_6f complex within the thylakoid membrane. As the quinone signal originates within the membrane, how might STT7/STN7 and CSK sense PQ redox state and convey this information to post-translational or transcriptional levels of gene expression? Because quinones are the only rapidly mobile electron carriers within the lipid bilayer, they seemingly form suitable signalling molecules for the membrane-anchored redox sensors. The mobility imparted by quinones' isoprenoid tails allows their reactive aromatic rings to interact with sensor domains anchored close to the membrane surface.

One model for the activation of STT7/STN7 by plastoquinol (PQH_2) suggests a two-step mechanism (Zito *et al*. 1999). Upon binding of PQH_2 at the quinol oxidation site (Qo) site of Cyt b_6f complex, the Rieske iron–sulphur protein moves from a distal position to a proximal position. This movement causes a conformational change that activates the LHC II kinase. It has been suggested that the two conserved cysteine residues on the lumenal side of STT7/STN7 are involved in the inactivation of the kinase by the thioredoxin system (Depege *et al*. 2003; Lemeille & Rochaix 2010). According to this model, these two cysteines interspersed with four amino acids represent a thioredoxin

target site. The ferredoxin–thioredoxin system is known to deactivate the LHC II kinase in high-light conditions (Rintamaki *et al*. 2000). It is further proposed that thylakoid proteins, such as CcdA and Hcf164, transmit the thioredoxin signals from the stroma to the lumenal cysteine residues of the STT7/STN7 (Dietzel, Brautigam & Pfannschmidt 2008; Lemeille & Rochaix 2010).

There are several limitations with this model of STT7/STN7 regulation (Puthiyaveetil 2011). First of all, the model hinges on conformational change rather than redox poise of the PQH_2 pool as the ultimate source of the signal. It does not distinguish the movement of Rieske protein as part of the activation mechanism from its regular movement in the protonmotive Q-cycle. Furthermore, the finding that site-directed mutagenesis of the conserved lumenal cysteine residues renders the STT7/STN7 inactive cannot be reconciled with this model (Lemeille *et al*. 2009). If the cysteines were indeed sites of thioredoxin-mediated inhibition, one would expect a constitutively active kinase, which is insensitive to thioredoxin inhibition. Additionally, this model posits that thylakoid proteins, such as CcdA and Hcf164, are involved in the transduction of thioredoxin signals from the stroma to the lumen; however, these proteins have well-defined functions in the biogenesis and assembly of the Cyt b_6f complex (Lennartz *et al*. 2001; Page *et al*. 2004). Therefore, their signalling functions in state transitions are expected to be secondary to their established role in biogenesis of chloroplasts.

This brings us to STN8, the paralogue of STN7, which is responsible for phosphorylation of PSII core proteins (Bonardi *et al*. 2005). How does STN8 sense signals and how is it activated? Like STN7, STN8 has a tripartite structure with a stromaly exposed kinase domain, a single membrane-spanning helix and an N-terminal stretch of amino acids exposed to the lumen. However, unlike STN7, STN8 does not possess conserved lumenal cysteine residues (Depege *et al*. 2003). Because PSII core phosphorylation is not selectively activated by PSII-specific light (Harrison, Tsinoremas & Allen 1991; Silverstein, Cheng & Allen 1993b), a direct activation of STN8 from the Qo site is unlikely. Furthermore, activation of STN8 by STN7 in a phosphorylation cascade is unlikely, as PSII core phosphorylation is unaffected in *stn7* mutants (Bonardi *et al*. 2005).

If sensing quinone signals is challenging enough for a membrane-intrinsic redox sensor such as STN7, one might ask how does the soluble redox sensor CSK function? Soluble redox sensors of quinones are known; in fact, examples exist in bacteria to suggest that it is possible. The light input component of cyanobacterial circadian oscillator, CikA, is a soluble protein sensing PQ redox state (Ivleva *et al*. 2006). The NifL sensor, which regulates the expression of nitrogen-fixing genes in *Azotobacter vinelandii*, is another example of a soluble redox sensor of quinone (Grabbe & Schmitz 2003). A third example is the anti-repressor protein AppA, which regulates photosynthesis gene regulation in *Rhodobacter sphaeroides* (Oh & Kaplan 2000). Additionally, the occurrences of membrane-extrinsic, electron carrier proteins that exchange electrons with the quinone pool also support quinone redox sensing by soluble sensors. An example for this is the respiratory protein sulphide : quinone oxidoreductase (SQR), found in many bacteria, archaea and eukaryotes (Theissen *et al*. 2003). SQR is a flavoprotein enzyme that catalyses electron transfer from the sulphide to the quinone.

Although the redox-sensing mechanism of CSK is yet to be revealed, three possibilities exist as to how CSK could sense the oxidized PQ. The first possibility is that CSK contain a redox-responsive cofactor such as flavin or haem within its GAF sensor domain. The NifL sensor of *A. vinelandii* and the AppA sensor of *R. sphaeroides* use flavin group for their redox-sensing function (Grabbe & Schmitz 2003). A haem prosthetic group confers the redox-sensing ability of the *Mycobacterium tuberculosis* DosS GAF domain (Kumar *et al*. 2007). A second possibility is that CSK uses redox-sensitive cysteine residues. Redox sensing by cysteine residues is a recurring theme in biological systems (Paget & Buttner 2003; Antelmann & Helmann 2011). Two conserved cysteines are found in CSK, with one of them present in the GAF sensor domain, and the other in the kinase domain. The role of these cysteines in CSK function remains to be understood. A third possibility is that CSK uses a redox-responsive cofactor, as well as conserved cysteines for its sensing function. In SQR, a Rossmann-fold binds a flavin adenine dinucleotide (FAD) molecule, and a pair of conserved cysteine residues transfer electrons from the sulphide to the quinone via FAD (Cherney *et al*. 2010). In this enzyme, a hydrophobic channel binds quinone, with the quinone aromatic ring stacked between two conserved phenylalanine residues in the hydrophobic core (Cherney *et al*. 2010). Hydrophobic regions resembling Rossmann-fold and quinone-binding pocket are present in CSK, and conserved phenylalanine residues are also found in the GAF sensor domain of CSK. Two amphipathic alpha-helices help SQR to anchor with the respiratory membrane during its electron transport reactions (Cherney *et al*. 2010). The presence of such amphipathic helices in CSK could help it associate with the thylakoid membrane as part of its redox-sensing function.

STATE TRANSITIONS – DUAL REGULATORS AT WORK

In addition to the LHC II kinase that initiates the state 2 transition, a second component – a phospho-LHC II phosphatase – is required to trigger the transition to state 1 (Bennett 1980; Allen *et al*. 1981). This phosphatase is thought to drive state 2 to state 1 transition in light 1 by dephosphorylating the phospho-LHC II, as the dephosphorylated LHC II returns to PSII from PSI. Until recently, the identity of the phospho-LHC II phosphatase has remained unclear. Two recent papers describe the identification and characterization of the long-sought phospho-LHC II phosphatase (Pribil *et al*. 2010; Shapiguzov *et al*. 2010). Named as thylakoid-associated phosphatase of 38 kDa (TAP38) or as PPH1, this PP2C-type protein phosphatase is a 38 kDa thylakoid protein with a predicted transmembrane helix

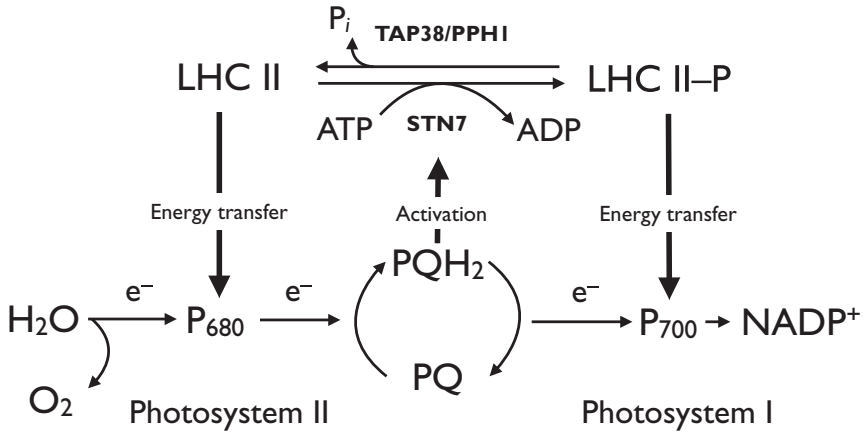

Figure 1. A regulatory scheme depicting the interactions of photosynthetic electron carriers with redox-signalling components of state transitions. Adapted from Allen (1992). Light reactions of photosynthesis are represented as electron transport from H_2O to $NADP^+$ via two photosystems connected by a cytochrome b_6f complex that oxidizes plastoquinol (PQH_2) to plastoquinone (PQ). In light absorbed preferentially by photosystem II (PSII), the light-harvesting complex II (LHC II) kinase STN7 responds to PQH_2 and initiates the state 2 transition, in which phosphorylation of LHC II leads to decreased light-harvesting antenna size of PSII and increased antenna size of photosystem I (PSI). When the PQ pool becomes largely oxidized, for example, when light quality favours excitation of PSI, the light- and redox-independent phospho-LHC II phosphatase TAP38/PPH1 catalyses phospho-LHC II dephosphorylation, resulting in a decrease in the light-harvesting antenna size of PSI and an increase of that of PSII.

at its C-terminus (Pribil *et al*. 2010; Shapiguzov *et al*. 2010). *Arabidopsis* plants with an inactivated *TAP38*/*PPH1* gene show hyperphosphorylation of LHC II and are arrested in state 2. Over-expressed and purified TAP38/PPH1 protein catalyses dephosphorylation of phospho-LHC II *in vitro*.

While the TAP38/PPH1 has all the properties required of the phospho-LHC II phosphatase, it is interesting to ask how it might fit in the regulatory scheme of state transitions (Fig. 1). Thylakoid phosphoprotein phosphatases are known to be redox insensitive and kinetically heterogenous (Silverstein, Cheng & Allen 1993a), with markedly different time courses of dephosphorylation. This surely rules out redox regulation of TAP38/PPH1; however, at this stage, there is no reason to eliminate other forms of regulation. If we assume that TAP38/PPH1 is not regulated at all on the timescale of state transitions, then this raises the question how might state transitions be achieved under seemingly opposite actions of a kinase and a phosphatase? The answer may lie in the kinetics of phosphorylation and dephosphorylation reactions, as well as in substrate availability for the LHC II phosphatase. The phosphorylation of LHC II and transition to state 2 from state 1 is faster than dephosphorylation of phospho-LHC II and transition to state 1. With the state 2 transition taking only 1–2 min to complete and the state 1 transition, as much as 15 min, it appears that the phosphorylation reaction is at least sevenfold faster than the dephosphorylation reaction. The reason for this may be that the free energy of phosphoryl transfer in the kinase reaction makes phosphorylation thermodynamically more favourable than the dephosphorylation reaction when both kinase and phosphatase are active. In the state 2 transition, therefore, the kinase outcompetes the phosphatase; however, in the state 1 transition, because the kinase is inactive, the slow but constant action of the phosphatase tips the reaction in favour of dephosphorylation of phospho-LHC II. This scenario thus supports the hypothesis that LHC II kinase is the most important determinant of the phosphorylation level of LHC II. A second possibility is that TAP38/PPH1 is usually associated with PSI, and therefore it dephosphorylates its substrate only when the latter is part of the PSI antenna, and not part of the PSII antenna.

Dephosphorylation of PSII core proteins was not affected in TAP38/PPH1 knock-out mutants (Pribil *et al*. 2010), indicating that more than one thylakoid phosphoprotein phosphatase is likely to be present in chloroplasts. This possibility is also supported by the finding that different thylakoid phosphoprotein phosphatase reactions proceed at different rates (Silverstein *et al*. 1993a).

TRANSCRIPTIONAL CONTROL – FUNCTIONAL PARTNERS, OLD AND NEW

In a two-component system, the sensor kinase comes paired with its response regulator partner, and it is the response regulator that brings about a physiological response to the signal sensed by the kinase (Stock *et al*. 2000). It is therefore interesting to ask: which is the response regulator partner of CSK? Studies with the cyanobacterial homolog of CSK suggest that the NarL-type response regulator ycf29 is the partner of CSK (Paithoonrangsarid *et al*. 2004; Sato *et al*. 2007). In red algae, diatoms and brown algae, ycf29 co-occurs with CSK, while in green algae and plants, neither ycf29 nor any other chloroplast response regulator is found (Puthiyaveetil & Allen 2009). The apparent loss of chloroplast response regulator in the green lineage correlates with

a modified kinase activity of CSK (Puthiyaveetil *et al*. 2008; Puthiyaveetil & Allen 2009).

In green algae and plants, non-response regulator proteins appear to have replaced the original ycf29 response regulator partner of CSK. In support of this, two known chloroplast transcriptional regulators are found to interact with CSK in a yeast two-hybrid study (Puthiyaveetil *et al*. 2010). One is the chloroplast sigma factor 1 (SIG1) (Shimizu *et al*. 2010). Chloroplast sigma factors, like CSK, originated from cyanobacteria (Tiller, Eisermann & Link 1991). Sigma factors are transcriptional initiation factors that enable the RNA polymerase to bind at specific gene promoters. *Arabidopsis* chloroplasts contain as many as six sigma factors (SIG1–6), with distinct but sometimes overlapping functional roles in transcribing the whole chloroplast genome or subsets of chloroplast genes in a developmental stage-specific or environmentally regulated fashion (Schweer *et al*. 2009). The important observation that connected sigma factors to photosystem stoichiometry adjustment is that the SIG1 is phosphorylated in response to oxidized PQ in light 1 (Shimizu *et al*. 2010). Phospho-SIG1 suppresses transcription of PSI genes in light 1, while the PSII transcription is unaffected by the phosphorylation of SIG1. This differential transcription of chloroplast photosystem genes results in a decrease of PSI amount relative to PSII, and thus adjustment of photosystem stoichiometry to the light 1 condition (Shimizu *et al*. 2010). However, for this SIG-1-mediated regulation to work, a protein kinase that phosphorylates SIG1 in response to oxidized PQ is required. This is where CSK fits in (Fig. 2a). As mentioned earlier, oxidized PQ promotes the autophosphorylation of CSK, and CSK interacts with SIG1 in a yeast two-hybrid screen. This interaction of CSK and SIG1 has been additionally confirmed by an *in vitro* pull-down assay; also, a truncated SIG1 protein, which contains the threonine 170 (T170) residue critical for the stoichiometry control, can be phosphorylated by CSK *in vitro* (Puthiyaveetil, Ibrahim & Allen, unpublished data). The presumed ability of CSK to phosphorylate its substrates on serine or threonine residues, using its autophosphorylated residue as a catalytic intermediate, is comparable to similar activities in some modified histidine kinases such as phytochromes and plant pyruvate dehydrogenase kinase (Yeh & Lagarias 1998; Thelen, Miernyk & Randall 2000).

It is therefore proposed that CSK represses PSI transcription in light 1 by acting as the SIG1 kinase, thereby decreasing the stoichiometry of PSI relative to PSII (Fig. 2a). However, the observation that CSK also interacts with a second chloroplast protein known as plastid transcription kinase (PTK) potentially introduces an additional layer of complexity in the mechanism of photosystem stoichiometry adjustment (Puthiyaveetil *et al*. 2010). PTK is a eukaryotic serine/threonine kinase that belongs to the casein kinase family of protein kinases (Baginsky *et al*. 1999). PTK is usually found associated with the plastid-encoded RNA polymerase (abbreviated PEP), where it phosphorylates PEP subunits to repress chloroplast transcription in low light (Baginsky *et al*. 1999; Baena-Gonzalez *et al*. 2001). PTK is thus regarded as a general repressor of chloroplast transcription in low light.

Phosphorylation inactivates PTK, and phospho-PTK cannot repress chloroplast transcription (Link 2003). However, the physiological relevance of PTK phosphorylation and the identity of the PTK kinase remain unclear. The observation that CSK interacts with PTK in the yeast two-hybrid assay seems to resolve these two issues (Puthiyaveetil *et al*. 2010). Firstly, the inference from the yeast two-hybrid study is that CSK could be the elusive PTK kinase. The interaction between CSK and PTK has been further confirmed using a pull-down assay, and PTK phosphorylation by CSK has also been demonstrated *in vitro* (Puthiyaveetil *et al*., unpublished data). Secondly, in order to understand the physiological relevance of PTK phosphorylation by CSK, two additional factors have to be taken into consideration. These are the light conditions in which these two kinases operate and their differing functional roles as general or specific regulators of chloroplast transcription. PTK operates in low-light conditions (~50 μE m^{-2} s^{-1}) to repress chloroplast transcription non-specifically (Baena-Gonzalez *et al*. 2001), while the CSK-mediated-specific chloroplast reaction centre gene regulation also occurs in low light (in the order of 12 μE m^{-2} s^{-1}), which affects the redox state of PQ – light 1 and 2 (Puthiyaveetil *et al*. 2008). This overlap in light conditions in which PTK and CSK function introduces a problem to chloroplasts. In order to achieve specific down-regulation of PSI genes in light 1 condition, the PTK-mediated general repression of chloroplast transcription has to be removed. This is where the CSK-mediated phosphorylation of PTK makes sense (Fig. 2a). CSK by acting as the PTK kinase will release general repression on chloroplast transcription, as phospho-PTK is inactive as a PEP kinase. CSK then represses only PSI transcription by acting as the SIG1 kinase, as phospho-SIG1 cannot efficiently transcribe at PSI gene promoters (Shimizu *et al*. 2010).

So far, we have only discussed photosystem stoichiometry adjustment in light 1. In light 2, when the PQ pool becomes reduced, PSI reaction centre genes are up-regulated. This requires a phospho-PEP phosphatase, as yet unidentified, that dephosphorylates phospho-SIG1 so that the repression on PSI transcription occurred during light 1 is now released (Fig. 2b). Under this light condition, CSK is inactive as a SIG1 kinase. What about PTK? Because CSK is inactive as a protein kinase, it cannot inhibit PTK, so PTK must be active as a PEP kinase. An earlier model of gene regulation assumed that the phospho-PEP phosphatase overrides PTK action by being kinetically faster or more numerous or by some other mechanism (Puthiyaveetil *et al*. 2010). In the light of new data (Brautigam *et al*. 2009), it appears that PTK may not be very active in light 2. Glutathione (GSH) inhibits PTK as a PEP kinase (Baginsky *et al*. 1999; Baena-Gonzalez *et al*. 2001), and it is possible that an increase in the GSH/GSSG ratio ensures that non-specific repression of chloroplast transcription does not occur, or occurs only to a limited extent, in light 2 (Fig. 2b).

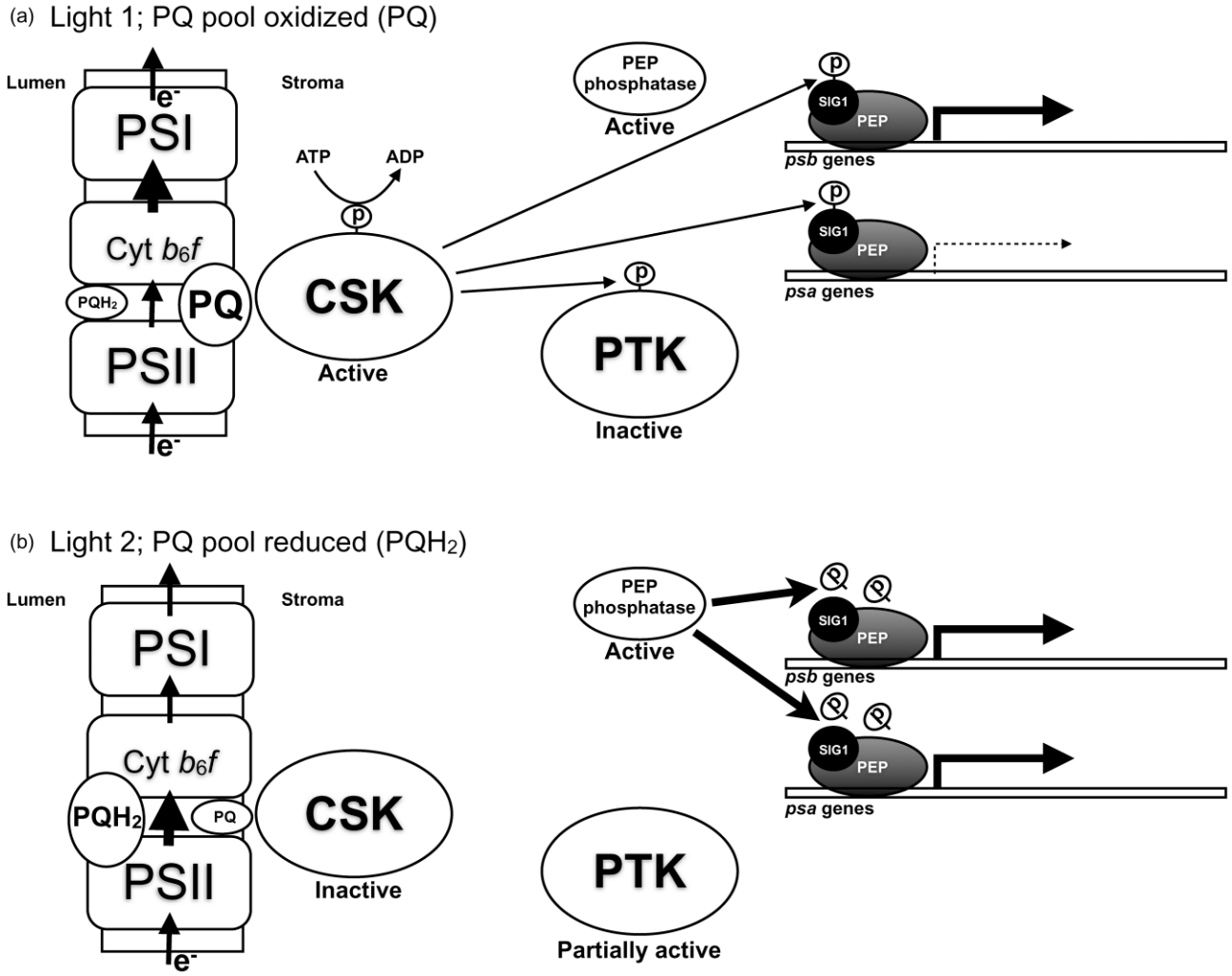
(a) Light 1; PQ pool oxidized (PQ)
Lumen
Stroma
PSI
Cyt b6f
PQH2
PQ
PSII
e-
ATP
ADP
P
CSK
Active
PEP phosphatase
Active
SIG1
PEP
psb genes
psa genes
PTK
Inactive
(b) Light 2; PQ pool reduced (PQH2)
Lumen
Stroma
PSI
Cyt b6f
PQH2
PQ
PSII
e-
CSK
Inactive
PEP phosphatase
Active
SIG1
PEP
psb genes
psa genes
PTK
Partially active
(c) High light; PQ pool reduced (PQH2); stromal reductants (Trx and GSH) reduced
Lumen
Stroma
PSI
Cyt b6f
PQH2
PQ
PSII
e-
CSK
Inactive
PEP phosphatase
Active
SIG1
PEP
psb genes
psa genes
PTK
Inactive

Figure 2. The proposed signal transduction pathway for photosystem stoichiometry adjustment. Photosynthetic electron transport is depicted diagrammatically, with electrons flowing from photosystem II (PSII) to photosystem I (PSI) via cytochrome b_6f complex (cyt b_6f). The redox state of plastoquinone (PQ) pool, and stromal electron carriers thioredoxin (Trx) and glutathione (GSH) control transcription of PSI genes (*psa* genes) and PSII genes (*psb* genes) through the redox sensory system formed by chloroplast sensor kinase (CSK), plastid transcription kinase (PTK) and sigma factor 1 (SIG1). 'PEP' stands for plastid-encoded RNA polymerase, and PEP phosphatase is the protein phosphatase involved in dephosphorylating phosphorylated PEP. Adapted from (Puthiyaveetil *et al*. 2010). (a) Under light 1, red- and far-red enriched illumination at rate-limiting intensity, the PQ pool is maintained in its oxidized form (PQ). CSK is autophosphorylated and active as a protein kinase using both SIG1 and PTK as substrates: SIG1 and PTK are thus maintained in their phosphorylated forms. Phospho-SIG1 represses transcription at the *psa* promoter while allowing transcription of *psb* genes. Phospho-PTK is the inactive form of PTK; therefore, it cannot suppress chloroplast transcription non-specifically, and under this circumstance, only CSK-mediated – via phospho-SIG1 – specific repression of *psa* genes occurs. PEP phosphatase is active in this light condition. However, the phosphorylation of SIG1 by CSK is faster than the dephosphorylation of SIG1 by PEP phosphatase. Therefore, in light 1, the action of CSK predominates over the action of the PEP phosphatase, resulting in an increase in the stoichiometry of PSII relative to PSI. (b) Under light 2, low-intensity illumination at wavelengths below the red drop in quantum yield and thus driving PSII, the PQ pool is largely in its reduced form (PQH_2). CSK is not autophosphorylated and is inactive as a protein kinase. The repression of *psa* genes, occurred during light 1, is now relieved by the slow but constant action of a PEP phosphatase that catalyses dephosphorylation of phospho-SIG1. As a result, PSI transcription increases. Under this light condition, PTK is only partially active as a PEP kinase, as it becomes increasingly inhibited by the rise in reduced glutathione (GSH) concentration. The action of PEP phosphatase overrides the residual PTK activity by dephosphorylating both SIG1, as well as PEP subunits, so that non-specific repression of reaction centre genes is counteracted. The increase in PSI transcription in light 2, therefore, leads to an increase in PSI units relative to PSII. (c) At higher light intensities, typically exceeding 100 μE m^{-2} s^{-1}, CSK and PTK are both made inactive by reduced GSH, by thioredoxin or by both. The inactivation of CSK and PTK protein kinases, combined with the phosphatase activity of PEP phosphatase, serves to increase chloroplast transcription globally, possibly as a stress response that helps chloroplasts to cope with the high photosynthetic activity in high-light conditions. Adapted in part from Puthiyaveetil *et al*. (2010).

Another physiological condition where the GSH-mediated repression of PTK operates is high light (Fig. 2c). At high-light intensities (~1000 μE m^{-2} s^{-1}), sufficient for reduction of GSH (Karpinski *et al*. 1997), and when regulation of photosystem stoichiometry adjustment is no longer a requirement, a global increase in chloroplast transcription is seen. In this stress response, which helps chloroplasts to cope with the high photosynthetic activity, the kinase activity of PTK is inhibited by GSH, releasing repression on chloroplast transcription (Baena-Gonzalez *et al*. 2001). Interestingly, CSK activity is also inhibited by thiol reductants such as GSH (Ibrahim, Puthiyaveetil & Allen, in preparation), suggesting the existence of a physiologically relevant regulatory mechanism operating in high light. An especially active phospho-PEP phosphatase may also work towards removing any inhibition of chloroplast transcription at high light (Fig. 2c).

Additional experiments are necessary to test and refine this new model of a chloroplast redox signalling pathway composed of CSK, PTK, SIG1 and phospho-PEP phosphatase (Fig. 2). For reasons that are not yet clear, it seems that, in evolution, non-response regulator partners, such as PTK and SIG1, have displaced the ycf29 as the functional partner(s) of CSK in plant photosystem stoichiometry adjustment.

LESSONS FROM CYANOBACTERIA

Chloroplasts are descendents of cyanobacteria (Gray 1992; Martin *et al*. 2002; Raven & Allen 2003; Bjorn & Govindjee 2009). Cyanobacteria too deploy state transitions and photosystem stoichiometry adjustments for correcting excitation energy imbalances in different time domains. Like that in plants, an altered redox state of the PQ pool is the signal that initiates state transitions and photosystem stoichiometry adjustments in cyanobacteria (Mullineaux & Allen 1986, 1990; Murakami *et al*. 1997). Understanding cyanobacterial redox signalling pathways, therefore, offers tremendous predictive value in elucidating plastid redox signalling pathways. In this context, it is interesting to see how far we have succeeded in dissecting out cyanobacterial redox regulatory units. It was suggested that the chloroplast LHC II model can accommodate cyanobacterial and red algal state transitions (Allen *et al*. 1981; Allen, Sanders & Holmes 1985; Allen 1992). In particular, the state 2 transition in cyanobacteria is characterized by a decreased absorption cross-section of PSII (Mullineaux & Allen 1988) accompanied by an increased absorption cross-section of PSI (Tsinoremas *et al*. 1989). However, cyanobacteria and red algae possess a different light-harvesting system from plants, and therefore the participation of a homolog of STT7/STN7 in their state transitions is unlikely. Moreover, the role of reversible phosphorylation is questionable in cyanobacterial state transitions (Mullineaux 1999), and it is likely that post-translational modifications other than phosphorylation are involved.

In cyanobacteria, a two-component system known as RppAB has been implicated in photosystem stoichiometry adjustment (Li & Sherman 2000). This system regulates both reaction centre and light-harvesting genes as part of the stoichiometry control. The RppAB system consists of the sensor histidine kinase RppB and the response regulator RppA. These two proteins, however, do not seem to be involved equally in photosystem stoichiometry control. A photosynthetic gene regulatory phenotype was discernable only in the response regulator mutants, and not in the sensor kinase mutants, which showed a wild-type response (Li & Sherman 2000). This has led to the suggestion that sensor kinases other than RppB could form partners of RppA in stoichiometry adjustment. Along these lines, a yeast

two-hybrid study found that the histidine kinase 2 (Hik2), the cyanobacterial homolog of CSK, interacts with RppA and thus forms its functional partner (Sato *et al*. 2007). Hik2 shows similar domain composition to CSK, including the putative redox-responsive cysteine residues. Even though a direct demonstration of the function of Hik2 in redox sensing is lacking in cyanobacteria, a few studies provide indirect evidence for this function (Mikami *et al*. 2003, Sato *et al*. 2007). As discussed earlier, in cyanobacteria, Hik2 also has a second response regulator partner known as response regulator 1 (Rre1). Through Rre1, Hik2 controls some important acclimatory responses (Mikami, Suzuki & Murata 2004; Paithoonrangsarid *et al*. 2004). These observations favour the idea that there may exist a functional crosstalk between RppAB and Hik2–Rre1 two-component systems. To complicate the matters further, RppAB system has also been shown to be involved in nickel sensing (Lopez-Maury *et al*. 2002). It is interesting to note that the RppAB system of cyanobacteria leaves no homologs in chloroplasts, whereas the Hik2–Rre1 system still survives as the CSK–Ycf29 pair. Although the details of the signalling pathways underpinning photosystem stoichiometry adjustments in cyanobacteria remain to be worked out, cyanobacteria and chloroplasts are very likely to use similar components and pathways in controlling reaction centre transcription.

CONCLUSION

While state transitions and photosystem stoichiometry adjustment share the same regulatory signal, the redox state of PQ, they seem to rely on two distinct signalling properties of PQ. For their signal, state transitions sense the reduced form of the quinone (Fig. 1), while photosystem stoichiometry adjustment is initiated by the oxidized form of quinone (Fig. 2). This possibly explains why these two responses do not have to compete for the same regulatory signal. We propose that state transitions and photosystem stoichiometry adjustment arising from two separate regulatory pathways, with each pathway sensing quinone signals independently. This proposal differs from other published schemes, which postulate shared regulatory components for these two processes (Allen 1995; Pesaresi *et al*. 2009). Because state transitions and photosystem stoichiometry adjustment are initiated by the same regulatory signal, they may represent coupled post-translational and transcriptional responses to the same environmental challenge, a coupling common in bacteria (Allen 1993b; Bauer *et al*. 2003; Allen, Allen & Puthiyaveetil 2008). As part of photosystem stoichiometry adjustment and more generally in response to changes in chloroplast homeostasis, expression of nuclear photosynthetic genes is also regulated (Pfannschmidt *et al*. 2001b; Gray *et al*. 2003; Woodson & Chory 2008), but how plastid signals reach the nucleus is still an open question.

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