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Review

Regulation of photosynthetic electron transport ☆

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ABSTRACT

The photosynthetic electron transport chain consists of photosystem II, the cytochrome b_6f complex, photosystem I, and the free electron carriers plastoquinone and plastocyanin. Light-driven charge separation events occur at the level of photosystem II and photosystem I, which are associated at one end of the chain with the oxidation of water followed by electron flow along the electron transport chain and concomitant pumping of protons into the thylakoid lumen, which is used by the ATP synthase to generate ATP. At the other end of the chain reducing power is generated, which together with ATP is used for CO_2 assimilation. A remarkable feature of the photosynthetic apparatus is its ability to adapt to changes in environmental conditions by sensing light quality and quantity, CO_2 levels, temperature, and nutrient availability. These acclimation responses involve a complex signaling network in the chloroplasts comprising the thylakoid protein kinases Stt7/STN7 and Stl1/STN7 and the phosphatase PPH1/TAP38, which play important roles in state transitions and in the regulation of electron flow as well as in thylakoid membrane folding. The activity of some of these enzymes is closely connected to the redox state of the plastoquinone pool, and they appear to be involved both in short-term and long-term acclimation. This article is part of a Special Issue entitled "Regulation of Electron Transport in Chloroplasts".

1. Introduction

The primary light-driven reactions of photosynthesis occur in the thylakoid membranes and are mediated by photosystem II (PSII) and photosystem I (PSI). The existence of these two distinct light reactions was first inferred from studies on the enhancement of the photosynthetic yield observed by illuminating algae with two light sources of far red and short-wavelength light [1]. The coupling of the two light reactions in a linear electron transfer chain forms the basis of the Z-scheme, which was proposed by Hill and Bendall 50 years ago and which still prevails today [2]. In this scheme, the two light reactions operate in series whereby electrons extracted from water by PSII are transferred through the plastoquinone pool, the cytochrome b_6f complex (Cytb_6f), and plastocyanin to PSI and ultimately to ferredoxin and $NADP^+$ to produce NADPH. These electron transfer reactions are coupled with proton pumping into the thylakoid lumen, and the resulting proton gradient is harnessed to produce ATP. Both ATP and NADPH fuel the Calvin-Benson cycle for CO_2 fixation and other assimilatory processes.

In recent years, significant advances have been made in our understanding of the composition, structure, assembly, and regulation of the major photosynthetic complexes PSII, PSI, with their associated antenna systems, Cytb_6f and ATP synthase. The structures of PSII [3], PSI [4] and Cyt b_6f [5,6] have been determined at atomic resolution providing new insights into the electron transfer routes within these complexes. They contain multiple subunits, pigments, and redox cofactors and are synthesized through the coordinate action of the nuclear and chloroplast genetic systems (for review, see [7,8]). Thus, some of the photosynthetic subunits are encoded by chloroplast genes and translated on chloroplast ribosomes while others are encoded by nuclear genes, synthesized on cytoplasmic ribosomes and imported into the chloroplast where they are assembled, together with their chloroplast-encoded partners, into functional complexes. Such a dual genetic origin of photosynthetic proteins necessitates a complex regulatory network for their coordinated expression and raises questions on why the plastid genetic systems have been maintained during evolution. Among several possible reasons, one hypothesis proposed by John Allen [9] is that plastid genomes have been maintained because of the dependence of their expression on the redox state of the electron transport chain. The localization of these two systems within the same cellular compartment would allow for rapid adjustment of gene expression to changes in environmental cues. Expression of the genes involved in photosynthesis is indeed regulated by several factors among which light is particularly important. Besides its role in light energy capture and conversion, the photosynthetic apparatus also acts as a sensor for changes in the light environment. In particular, the redox state of various photosynthetic electron transport components and photosynthesis-dependent

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redox-active compounds are involved, at least in some cases, in the coupling of photosynthetic electron flow with gene expression (for review, see [10]).

A remarkable feature of the photosynthetic apparatus is its ability to adjust rapidly to changes in environmental and metabolic conditions and in micronutrient availability. While light is essential for photosynthesis, too much light can be harmful when the absorbed light energy exceeds the capacity of the photosynthetic machinery. Under these conditions, the excess photons and electrons need to be dissipated to protect the photosynthetic apparatus from light-induced damage. This occurs through a rapidly inducible non-photochemical quenching process, called ΔpH-dependent quenching in which the excess absorbed light energy is dissipated as heat [11]. In addition, several photoprotective mechanisms exist such as plastid antioxidant enzymes and molecules [11], repair processes for lipid peroxidation [12], and damaged PSII [13]. At the other extreme, under low light conditions, the photosynthetic machinery adapts to optimize its photosynthetic yield. In nature photosynthetic organisms are subjected to constant changes in light quantity and quality and need to adjust their photosynthetic electron transport system accordingly. The molecular mechanisms underlying the responses of the photosynthetic electron transport chain to these environmental changes and their regulation have been intensively studied in recent years and will be discussed in this review.

2. Photosynthetic electron transport pathways

Photosynthetic electron flow is driven by two photochemical reactions catalyzed by PSII and PSI, which are linked in series by the electron transport chain (Fig. 1). At one end of this chain, the photochemical activity of PSII creates a charge separation across the thylakoid membrane with a strong oxidant on the donor side capable of oxidizing water with the concomitant release of protons and molecular oxygen in the thylakoid lumen and the reduction of the primary electron acceptors of PSII, Q_A and Q_B, on the stromal side of the membrane. Once it has accepted two electrons, Q_B is released from PSII into the plastoquinone pool and reduced plastoquinol docks to the Qo site of Cytb_6f. This complex acts as a proton pump in a Q-cycle-like process [14]. Oxidation of plastoquinol releases two protons in the lumen and two electrons, one of which is transferred through the high potential chain to the Rieske protein and cytochrome *f* and subsequently to plastocyanin and PSI. The other electron is transferred through the low potential chain to cyt_L and cyt_H within the Cytb_6f complex and finally to a quinone at the Qi site to form a semiquinone. Upon oxidation of a second plastoquinol at the Qo site, this process is repeated and semiquinone is reduced to quinol and released from Qi. It can enter the Q-cycle again as described above. Electron transfer from Cytb_6f to PSI is mediated by the copper protein plastocyanin in the thylakoid lumen. PSI acts as a light-driven oxidoreductase by oxidizing plastocyanin and transferring electrons through its three internal 4Fe-4S centers F_X, F_A, and F_B to ferredoxin. Ultimately these electrons can be used by ferredoxin $NADP^+$ reductase (FNR) to produce NADPH, which together with the ATP generated by the ATP synthase will drive the Calvin–Benson cycle for CO_2 assimilation. In addition, ferredoxin donates electrons to other pathways such as cyclic electron transfer, sulfur and nitrogen assimilation, and to thioredoxins, which regulate carbon assimilation [15]. In some green algae, upon a transition from the dark to the light under anaerobic conditions, ferredoxin transfers transiently electrons to chloroplast hydrogenases, which catalyze the formation of hydrogen [16]. In these organisms, this process appears to act as an electron safety valve to dissipate excess reducing power when the Calvin–Benson cycle is not yet fully activated.

Besides the linear electron transfer mode (LEF), the system can also perform cyclic electron transport (CEF) [14,17]. In this case, reduced ferredoxin, the terminal acceptor of PSI, transfers its electrons back to the plastoquinone pool through NADPH or directly to the Cytb_6f complex, giving rise to cyclic electron flow around PSI, which is coupled with proton translocation and ATP formation. One cycle of the Calvin–Benson pathway, i.e., the fixation of one CO_2 molecule consumes 3 ATP and 2 NADPH molecules. However, estimates of the ATP/NADPH ratio arising from LEF are about 1.28, which is clearly not sufficient for driving the Calvin cycle [18]. This can be estimated from the fact that the transfer of 4 electrons from water to $NADP^+$ by the linear electron transfer chain produces 2 NADPH molecules and is coupled to the pumping of 12 protons into the thylakoid lumen

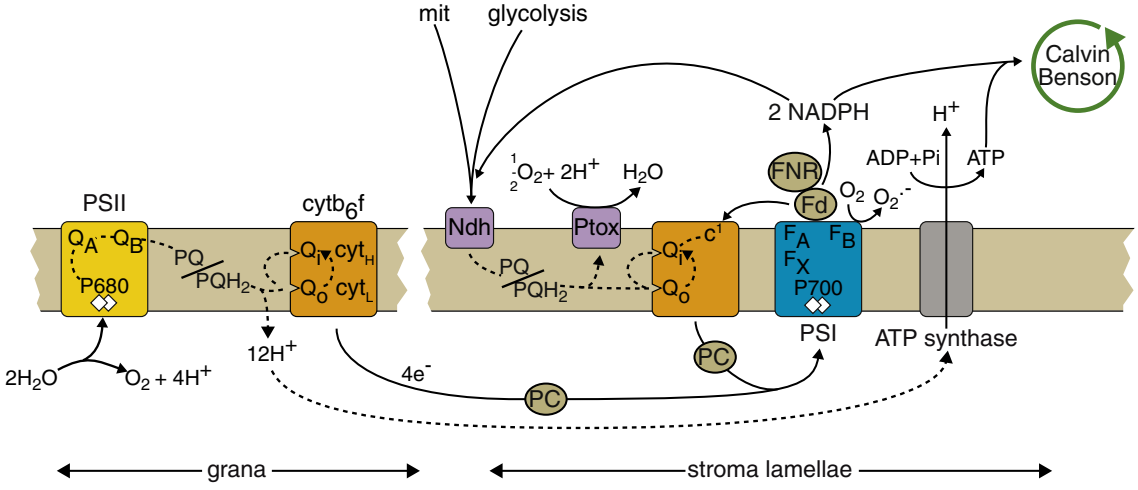

Fig. 1. Electron transport pathways of oxygenic photosynthesis. The thylakoid membrane with PSII, Cytb_6f, PSI, and the ATP synthase is shown. Electron transport pathways are shown by dotted lines with arrows to indicate the direction of electron flow. Linear electron transport (LEF) starts with the photo-induced water oxidation catalyzed by PSII. The stoichiometry of the reactions is indicated for 4 photons absorbed by PSII and 4 photons absorbed by PSI. Electrons are transferred from PSII through the PQ pool to Cytb_6f, which acts as proton pump through the Q cycle. Electrons are transferred from Cytb_6f to the soluble electron carrier plastocyanin (PC) and then to PSI, which acts as light-driven plastocyanin ferredoxin oxidoreductase. Ultimately FNR reduces NADP + to NADPH at the expense of reduced ferredoxin. Cyclic electron flow (CEF) involves PSI and Cytb_6f and occurs mostly in the stroma lamellae of thylakoids. Two different routes are indicated. The first uses ferredoxin, NADPH, and the Ndh complex. In the second pathway, ferredoxin transfers electrons directly to cyt *c′* in the Cytb_6f complex. Electron transport is coupled to proton translocation into the thylakoid lumen, and the resulting pH gradient drives ATP synthase to produce ATP. In addition pseudocyclic electron flow occurs in which molecular oxygen is reduced by PSI (Mehler reaction) to produce superoxide ($O_2^{\cdot-}$), which is converted to H_2O_2 by superoxide dismutase and to H_2O and O_2 by peroxidases and catalases (water–water cycle). Stromal reducing equivalents originating from mitochondria or from glycolysis are channeled into the PQ pool through the chlororespiratory chain, which includes the Ndh complex and PTOX as final electron acceptor.

assuming there is no leakage of electrons or protons. The membrane embedded chloroplast ATP synthase CFo consists of 14 CFo subunits arranged in a circular structure [19]. Each of these subunits allows for the transfer of one proton per turn of the ATP synthase. Thus 14 protons are required for a full turn of the ATP synthase to generate 3 ATP molecules. Hence, two additional protons, in addition to the 12 protons produced by LEF, need to be pumped across the thylakoid membrane through the cyclic electron transfer pathway to accommodate the requirements of the Calvin–Benson cycle. Optimal operation of the Calvin–Benson cycle therefore requires both linear and cyclic electron flow. Moreover, by tuning the levels of LEF and CEF, the ATP/NADPH can be adjusted to meet the cellular demands. This is particularly striking in *Chlamydomonas reinhardtii* in which intracellular ATP depletion induces a switch from linear to cyclic electron transport (see below).

While CEF is well documented in cyanobacteria, unicellular algae, and C4 plants, it is only recently that its importance has been recognized and studied in C3 plants [14]. In C3 plants, CEF is especially important under specific stress conditions such as low CO_2, high light, drought, or during dark to light transitions. Under these conditions, cyclic electron flow allows for acidification of the thylakoid lumen, which is required for inducing ATP synthesis and for triggering non-photochemical quenching, which in turn downregulates PSII. CEF is also important when photorespiration is active because more ATP is required for CO_2 fixation under these conditions [20]. Several routes for cyclic electron flow have been proposed. In the first, ferredoxin transfers its electrons through FNR to $NADP^+$ and the NAD(P)H dehydrogenase complex and ultimately to the plastoquinone pool. However, in plants, the level of this complex is probably too low for mediating sufficient cyclic electron flow required for ATP production under steady state conditions [21]. FNR, which is found both in the stroma and associated with thylakoid membranes, has been proposed to modulate partitioning between the cyclic and linear electron pathways. In the second pathway, ferredoxin is thought to transfer electrons to the plastoquinone pool through a ferredoxin–plastoquinone oxidoreductase [22], an enzyme that has however not yet been identified. In the third, ferredoxin may interact directly with Cytb_6f and transfer its electrons to cytc′, a new component identified in the crystal structure of the Cytb_6f complex, using a Q-cycle derived mechanism (reviewed in [14,23]).

A deficiency in cyclic electron flow is expected to lower the acidification of the lumen and to diminish NPQ. A genetic screen for mutants of *Arabidopsis* deficient in NPQ indeed led to the identification of *pgr5*, a mutant deficient in the thylakoid protein PGR5 [24]. In this mutant, both ferredoxin-dependent reduction of plastoquinone and NPQ are decreased. Moreover, when this mutant was crossed with *crr2*, a mutant deficient in the NADH dehydrogenase (Ndh), the double mutant was severely retarded in growth, suggesting that PGR5 plays some role in cyclic electron flow. However, other studies indicate that PGR5 is not directly involved in cyclic electron flow. It may regulate the switch between linear and cyclic electron flow as loss of PGR5 limits the ability of cyclic electron flow to compete for electrons with linear electron flow [25]. A second protein, PGRL1, involved in cyclic electron flow was identified through a reverse genetic screen [26]. As in *pgr5*, loss of this protein leads to perturbations in cyclic electron flow. PGRL1 and PGR5 interact physically with each other and bind to PSI. At this time, it is not yet clear whether the PGR5–PGRL1 complex is involved directly or indirectly in the ferredoxin-dependent cyclic electron flow.

Interestingly, a large supercomplex has been detected in *Chlamydomonas*, which comprises PSI with its light-harvesting system LHCI, the major trimeric LHCII and the minor monomeric LHCII CP29 and CP26, Cytb_6f including PetO, a subunit that has only been detected in *Chlamydomonas*, and the proteins PGRL1 and FNR [27]. This complex is capable of driving cyclic electron flow as shown by spectroscopic measurements, which indicate that upon illumination reducing equivalents generated by PSI are transferred to Cytb_6f and that oxidized PSI can be reduced by reducing equivalents from Cytb_6f. The experimental identification of this complex is compatible with the view that the Cytb_6f complex accepts electrons directly from ferredoxin and that the hypothetical action of the ferredoxin–plastoquinone reductase may be mediated through the concerted action of PGRL1, PGR5, and FNR. Thus it is possible that there is only one single route of ferredoxin-dependent cyclic electron flow that involves Cytb_6f. Although the location of the PSI-Cytb_6f supercomplex in the thylakoid membrane has not yet been determined, it could be separated from PSII [27]. This supercomplex must localize with a fraction of the mobile electron carriers plastoquinone, plastocyanin, and ferredoxin in its vicinity in such a way that cyclic electron flow can operate independently from linear electron flow. Such an organization would avoid competition between cyclic and linear electron flow and avoid disturbance of the redox poise of CEF components by reduced LEF components (see Fig. 1).

Over-reduction of the electron transport chain can stall electron flow because under these conditions, there is no way for the electrons to go [17]. Under these conditions, pseudo-cyclic electron flow occurs in which electron transfer proceeds from water to PSI with molecular oxygen as alternative electron acceptor. This process called Mehler reaction leads to the formation of superoxide, which is converted subsequently by superoxide dismutase and catalase to form water and oxygen. This water–water cycle has been proposed to restore the redox poise when the electron transport chain is over-reduced [28], thereby allowing cyclic electron transfer to function and to generate ATP for the Calvin cycle, which will in turn oxidize NADPH and restore LEF.

The PQ pool of the photosynthetic linear and cyclic electron transport chain is shared with the chlororespiratory chain, which is involved in electron transfer reactions from stromal reductants to dioxygen through the PQ pool. Although this chlororespiratory chain was discovered in 1982 by Bennoun [29] in *Chlamydomonas*, it is only recently that some of the components involved in non-photochemical reduction and oxidation of the PQ pool have been identified. The first is a NAD(P) dehydrogenase complex, which also appears to be involved, in part, in cyclic electron flow. Several subunits of this complex are encoded by the chloroplast genome, and other nucleus-encoded subunits have been identified through genetic approaches. However, none of the identified subunits carry any known motif involved in pyridine nucleotide binding or catalysis [30]. As for other photosynthetic complexes, the biosynthesis of the NADH complex requires the participation of many nucleus-encoded factors, which act at transcriptional and post-transcriptional steps. In this regard, genetic mutant screens based on altered fluorescence properties of mutants with defects in the NDH complex have identified several novel factors mostly involved in the editing of chloroplast NDH transcripts [31]. In some cases, editing appears to depend on developmental and environmental conditions. Other factors identified in this screen are involved in the processing of polycistronic transcripts and in the regulation of their expression [32]. It is noticeable that the NADH dehydrogenase complex is not present in all photosynthetic organisms and in particular is absent in the green alga *C. reinhardtii* in which plastoquinone reduction by NAD(P)H is mediated instead by the NDH-2 enzyme [33].

The other identified component of the chlororespiratory chain is a plastid quinol terminal oxidase called PTOX [34,35]. The level of this enzyme increases in response to environmental stresses. Together with the NDH complex, PTOX could have a regulatory role by poising the redox state of intersystem electron carriers in thylakoid domains in which cyclic electron flow is prevalent. Moreover, it could act as a safety valve by preventing over-reduction of the electron transport chain under high light [36]. Indeed overexpression of PTOX lowers the PQ reduction state during the induction phase of photosynthesis when the Calvin–Benson cycle is not yet activated [37,38]. However,

the same studies indicate that there is only a minor effect during steady-state photosynthesis and overexpression of PTOX does not convey increased protection against photoinhibition in tobacco and *Arabidopsis* [37,38]. Loss of PTOX expression in the *immutans* mutant of *Arabidopsis* leads to a variegation phenotype, which is characterized by the presence of white sectors with abnormal plastids, lacking lamellar structures and unable to produce chlorophyll and carotenoids, in normally green organs [34]. Analysis of this mutant has revealed that PTOX plays an important role during the initial early stages of chloroplast biogenesis in protection of photooxidation by lowering the excitation pressure of PSII [39] and it also has a crucial role in carotenoid biosynthesis [36]. Further insights into the possible role of PTOX come from studies of marine photosynthetic organisms, which are confronted with iron limitation in the oceans. They adapt to this particular environment by decreasing the level of Cytb_6f and PSI, which contain several iron sulfur centers and are thus an important sink for iron in the chloroplast [40]. This however could lead to perturbations in electron transfer. These organisms appear to use an alternative electron pathway from water to O_2 by extracting electrons from the intersystem electron transport chain prior to photosystem I [40]. Treatment with propyl-gallate, a specific inhibitor of PTOX, was shown to lead to an increased PSII excitation pressure suggesting that this oxidase mediates electron flow from the PQ pool to dioxygen under these conditions. This suggests that PTOX lowers the PSII excitation pressure and prevents formation of ROS, which could otherwise occur at low irradiance [40]. It is not yet known whether this mechanism is also used by other photosynthetic organisms for satisfying their energetic requirements when PSI levels are low, when the PSII excitation pressure is high due to changes in environmental conditions, or when PSII is disconnected from the remaining electron transport chain.

3. State transitions

The light-harvesting systems of PSII and PSI have different protein and pigment composition and hence different light absorption properties. Thus changes in light quality lead to unequal excitation of PSII and PSI and create an imbalance in the electron transport chain, which is corrected through state transitions [41–43] (Fig. 2). Under changing light conditions, the redox state of the plastoquinone pool becomes reduced or oxidized depending on whether PSII or PSI is preferentially excited. A reduced redox state of the plastoquinone pool favors the binding of plastoquinol to the Qo site of the Cytb_6f complex and leads to the activation of a protein kinase, which phosphorylates LHCII [44,45]. This phosphorylation event causes the migration of the phosphorylated LHCII to PSI thus increasing the cross-section of the PSI antenna and rebalancing the excitation energy between PSII and PSI (state 2). This process is reversible as overexcitation of PSI leads to the oxidation of the plastoquinone pool, inactivation of the LHCII kinase, dephosphorylation of LHCII through a phosphatase and to the return of LHCII to PSII (state 1). In plants considerable thylakoid membrane remodeling occurs during state transitions with breakage and fusion of grana layers [46]. In *C. reinhardtii*, a major role of state transitions is to maintain ATP homeostasis rather than antennae size adjustment [41]. Transition from state 2 to state 1 occurs readily when the cellular ATP level decreases. As much as 80% of the LHCII antenna is mobile during state transitions in this alga, whereas in land plants, it is only 15–20% [47].

The best candidate for the LHCII protein kinase is the Stt7/STN7 kinase, which was initially identified through a genetic screen for mutants of *C. reinhardtii* deficient in state transitions [48]. The Stt7 kinase has an ortholog in *Arabidopsis* called STN7 and is conserved in other land plants and in eukaryotic photosynthetic organisms [49]. The Stt7/STN7 kinase contains a single transmembrane domain that separates the small N-terminal region in the thylakoid lumen from the catalytic kinase domain on the stromal side of the membrane [50]. Besides its role in state transitions, which represents a short-term response occurring in the minute range, STN7 is also involved in a long-term response [51]. This response involves a readjustment of the stoichiometry of the two photosystems to allow for optimal photosynthesis under conditions, which favor either one of the two photosystems. The Stt7/STN7 kinase forms a small family with Stl1/STN8, another thylakoid protein kinase, which is also conserved in *Chlamydomonas* and *Arabidopsis* [48] and required for the phosphorylation of the PSII core proteins [51,52]. Besides its activation through the reduced redox state of the plastoquinone pool, the LHCII kinase is

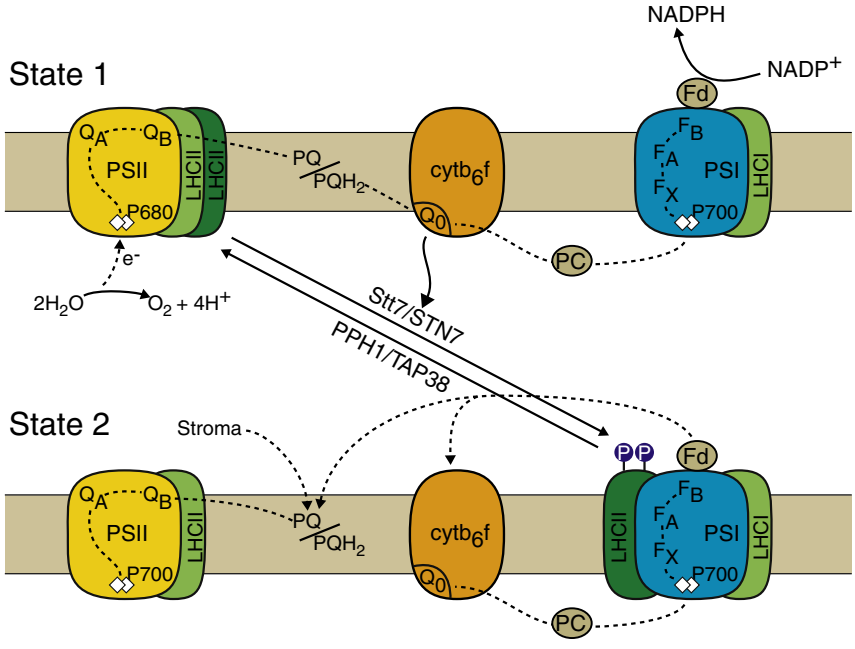

Fig. 2. State transitions. Transition from state 1 to state 2 occurs when the redox state of the plastoquinone pool (PQ) is reduced, for example, as a result of preferential excitation of PSII relative to PSI. Docking of plastoquinol (PQH_2) to the Qo site of Cytb_6f leads to the activation of the protein kinase Stt7/STN7, which phosphorylates LHCII either directly or indirectly. Phosphorylated LHCII dissociates from PSII and binds to PSI. In *C. reinhardtii*, this state 1 to state 2 transition leads to a switch from linear electron flow to cyclic electron flow. Upon preferential excitation of PSI relative to PSII, the kinase is inactivated and the PPHI/TAP38 phosphatase dephosphorylates LHCII, which moves back to PSII.

inactivated at high light when ferredoxin and thioredoxin are reduced [53,54]. Possible target sites include two conserved Cys residues in the N-terminal domain. Site-directed mutagenesis of either of these Cys residues abolishes kinase activity [50]. Although these two Cys are located on the lumen side of the membrane and the ferredoxin/thioredoxin system operates on the stromal side, one interesting possibility is that it controls the redox state of the two Cys through a trans- membrane thiol pathway mediated by the CcdA and Hcf164 proteins, which are known to be essential for plastid cytochrome *c* maturation and Cyt b_6f assembly [55–57].

A LHCII-specific phosphatase, called PPH1/TAP38, which dephosphorylates LHCII upon a transition from state 2 to state 1, has recently been identified [58,59]. This phosphatase is specifically required for the dephosphorylation of the major trimeric Lhcb1 and Lhcb2 proteins but not of the PSII core proteins D1, D2, and CP43. PPH1 belongs to the family of monomeric PP2C type phosphatases. It is a chloroplast protein that is mainly associated with the stromal membranes of the thylakoid membranes. Loss of PPHI/TAP38 gives rise to an increase in the antenna size of PSI and strongly impairs state transitions. The phosphatase appears to act directly on LHCII as recombinant PPH1/TAP38 is able to dephosphorylate LHCII *in vitro* [58]. Thus phosphorylation and dephosphorylation of LHCII during state transitions are specifically controlled by the Stt7/STN7 kinase and PPH1 phosphatase pair.

Although Stt7/STN7 mediated phosphorylation of LHCII is clearly required for state transitions, i.e., for the reversible transfer of the mobile LHCII between PSII and PSI, in other cases, phosphorylation of LHCII is not accompanied by state transitions. This occurs when reversible LHCII protein phosphorylation is induced by subjecting *Arabidopsis* plants to alternative periods of low light and high light irradiance [60]. Under this fluctuating light regime, LHCII is phosphorylated during the low light and dephosphorylated during the high light phases, but these changes in phosphorylation do not significantly change the distribution of excitation energy between PSII and PSI. In the absence of STN7, a high excitation pressure of PSII is observed under low light suggesting that this kinase ensures a balanced distribution of excitation energy to both photosystems and thereby optimizes electron transfer under fluctuating light. Loss of STN7 in plants subjected to these fluctuating light conditions leads to a severe decrease in growth [60].

In *C. reinhardtii*, state transitions act as a switch between linear (state 1) and cyclic electron flow (state 2) [61,62]. Although transitions between state 1 and state 2 can be induced by exposure to light preferentially absorbed by PSI and PSII, respectively, the switch between linear and cyclic electron flow is mainly controlled by the cellular ATP level. A drop in ATP triggers a transition from state 1 to state 2. The analysis of state transitions has provided new insights into the interdependence of mitochondrial respiratory and chloroplast photosynthetic electron flow. When oxidative phosphorylation is inhibited by anaerobiosis or by addition of uncouplers or inhibitors of mitochondrial electron transport, a rapid transition to state 2 occurs [63]. Similarly, mutants deficient in mitochondrial respiration tend to stabilize state 2 [64]. In these mutants, light specific for PSI fails to promote transition to state 1, their plastoquinone pool is more reduced, and the rate of cyclic electron flow is increased (Fig. 3A). In these mutants non-photochemical reduction of the plastoquinone pool occurs by stimulated NADH production through glycolysis induced by lower ATP levels [65] and also because of lower rates of mitochondrial NADH oxidation [64]. Thus the ATP deficiency in these mitochondrial mutants is partly compensated by increased cyclic photophosphorylation in the chloroplast. This shows that photosynthetic electron transport is tightly controlled by respiration in *C. reinhardtii*. In particular, the redox poise of the plastoquinone pool is determined to a large extent by the efficiency of respiration. Surprisingly, the importance of state transitions as a regulatory process for modulating cyclic and linear electron transport and for meeting intracellular demands for ATP is not apparent from the phenotype of the *stt7* mutant, which is nearly indistinguishable from that of wild type in terms of phototrophic growth properties [66]. However, if one takes into account that mitochondria are the main energy producer besides chloroplasts, it is conceivable that there might be some energetic compensation when either of the two systems is perturbed. This was indeed elegantly illustrated by a genetic approach in which a double mutant deficient both in state transitions (*stt7*) and mitochondrial respiration (*dum22*) was shown to be severely affected in phototrophic growth [67] indicating that state transitions have an essential role in the maintenance of the intracellular ATP level when mitochondrial respiration is impaired. The reduced phototrophic growth of this double mutant clearly indicates that in the absence of ATP generated through respiration,

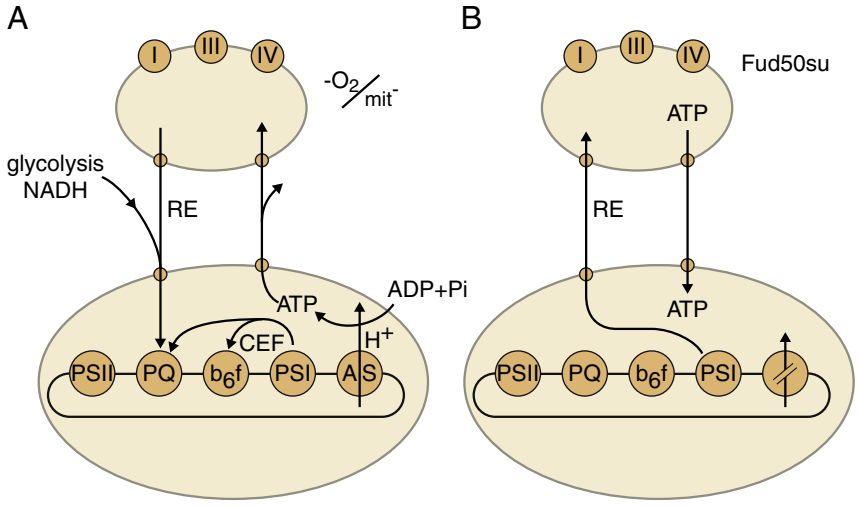

Fig. 3. Interactions between the chloroplast photosynthetic and mitochondrial respiratory electron transport chains in *C. reinhardtii*. Complexes I (NADH dehydrogenase), III (*bc*1 complex), and IV (cytochrome oxidase complex) from the mitochondrial respiratory chain (upper part) and photosynthetic complexes PSII, Cytb_6f, PSI, and ATP synthase (AS) in the chloroplast thylakoid membrane (lower part) are indicated. A. Loss of mitochondrial respiration under anaerobic conditions ($-O_2$) or because of a mutation affecting the respiratory chain (mit$^-$) leads to increased influx of reducing equivalents (RE) into the chloroplast through the malate shuttle. As a consequence, the more reduced redox state of the plastoquinone pool triggers a transition from state 1 to state 2 and a switch from LEF to CEF with increased ATP synthesis. ATP is exported from the chloroplast through the ATP/ADP transporter to the cytosol and mitochondria. B. In the *Fud50su* suppressor strain, which lacks chloroplast ATP synthase [68], ATP generated in the mitochondria is transferred to the chloroplast through the ATP/ADP transporter. Excess reducing equivalents produced in the chloroplast are probably exported to the cytosol and mitochondria.

cyclic electron flow becomes crucial for maintaining photosynthetic activity. In the mitochondrial mutant, the increased influx of electrons into the plastoquinone pool through the photochemical activity of PSII on one hand and the non-photochemical chlororespiratory processes on the other is counterbalanced by the enhanced PSI activity due to the 2-fold increase in PSI antenna in state 2 with concomitant increase in CEF [67]. In contrast in the *stt7dum22* double mutant, which is blocked in state 1, the plastoquinone pool is over-reduced and because of the failure of this strain to enhance CEF, the requirement of the Calvin–Benson cycle for an ATP/NADPH ratio of 1.5 cannot be satisfied. Thus efficient carbon assimilation in this alga requires the combined energetic contribution of both respiration and state transitions through their ability to modulate the absorption cross-section of PSI and cyclic photophosphorylation.

Another example of the dynamic metabolic interactions between the mitochondrial and chloroplast compartments is provided by the analysis of *Fud50*, a chloroplast mutant of *C. reinhardtii* deficient in ATP synthase and unable to grow phototrophically. A second site suppressor, *Fud50su*, was isolated in which phototrophic growth is restored although the chloroplast ATP synthase is still absent [68]. However, the observation that phototrophic growth is sensitive to inhibitors of mitochondrial electron transport led to the proposal that in this suppressor, exchange of NADPH reducing equivalents from the chloroplast to the mitochondria on one hand and transport of ATP from the mitochondria to the chloroplast on the other are activated in such a way that the ATP derived from mitochondrial respiration is sufficient for carbon assimilation in chloroplasts that lack ATP synthase (Fig. 3B) [68]. It would clearly be interesting to unravel the molecular mechanisms that are altered in this suppressor mutant.

4. Redox state of the plastoquinone pool

In the intricate chloroplast electron transport network, the plastoquinone pool acts as a major hub in electron trafficking. Transfer of electrons from the plastoquinone pool to the Cytb_6f complex is the rate-limiting step in linear electron flow (see below). Thus the redox state of the plastoquinone pool plays a critical role in this process. Because the plastoquinone pool is at the crossroad of the photosynthetic and chlororespiratory electron transfer chains, its redox state is influenced by both of these pathways and by several other factors. Reducing equivalents arising from starch breakdown can reduce the plastoquinone pool in the dark through a nucleus-encoded type II NAD(P)H dehydrogenase in *C. reinhardtii* [33,69].

The plastoquinone pool can be oxidized through the plastid terminal oxidase whose functional role is not yet fully understood. Alternatively, plastoquinone oxidation could also be mediated by cytochrome *b559* of PSII as shown by its ability to mediate PQH_2 oxidation *in vitro* and by the fact that the plastoquinone pool is over-reduced in a mutant deficient in cytochrome *b559* [70]. The redox state of plastoquinone is further influenced by the relative excitation of PSII and PSI, which are influenced by changes in the wavelength and intensity of light, the ATP/ADP ratio, CO_2 concentration, the level of PTOX, and the operation of the Calvin–Benson cycle.

Is there a link between photosynthetic electron transfer and plastid gene expression? Two-component systems of signal transduction consisting of a sensor kinase and a response regulator are well documented in bacteria and cyanobacteria [71]. Although chloroplast-encoded sensor kinases are unknown in plants and algae, a nucleus-encoded sensor kinase has recently been identified in *Arabidopsis* chloroplasts [72]. This kinase, named CSK, is well conserved in most lineages of green algae and plants. It contains a GAF domain that is implicated in redox sensing [72]. Although chloroplast transcript accumulation is altered in the *csk* mutant relative to the wild type upon irradiation with light of wavelengths, which stimulate preferentially PSII or PSI, it is not yet clear whether CSK acts as a redox regulatory coupling device between photosynthesis and chloroplast gene expression.

Besides triggering chloroplast responses such as state transitions, changes in the redox state of the plastoquinone pool can also influence nuclear gene expression through retrograde signaling. However, the cytosolic components of this signaling chain, which appears to act both at the transcriptional and post-transcriptional level, are still largely unknown [73,74].

5. Role of plastoquinone diffusion in the control of photosynthetic electron transport

The PSII–LHCII complexes and most of the mobile LHCII are present in grana stacks, whereas PSI–LHCI and ATP synthase are excluded from this region because of steric hindrance due to their bulky stromal domains. In contrast to the other complexes, the Cytb_6f complexes are distributed throughout all the domains of the thylakoid membranes [75] (Fig. 1). Electron flow between the photosynthetic complexes is mediated by two mobile carriers, plastoquinone and plastocyanin in the lipid bilayer and lumenal space, respectively. This lateral separation of the photosynthetic complexes within the membrane compartments raises the question of how electrons are transferred between them and especially on the importance of plastoquinone diffusion.

The oxidation of plastoquinol at the Qo site of the Cyt b_6f complex with a turnover time of 3.3 to 5 ms is the slowest step in the electron transport chain [76]. In comparison, upstream electron transport occurs with turnover times of 1 ms for the transfer from H_2O to PSII centers [77] and of 2 ms for the transfer from Q_A to diffusible plastoquinone [78]. Downstream transfer from Cytb_6f to PSI through the mobile carrier plastocyanin is significantly faster with half times ranging between 150 and 550 μs (see [79]).

The question is whether diffusion of plastoquinone between PSII and Cytb_6f complexes within the grana discs, which have diameters of 400 to 500 nm, is fast enough for plastoquinone to act as a non-rate-limiting carrier in electron flow throughout the membrane. While diffusion of plastoquinone in lipid vesicles was measured to be sufficiently high for plastoquinone to act as a non-rate-limiting carrier [80], this is not the case in the protein crowded environment of the thylakoid membrane in which plastoquinone diffusion has been estimated to be one order of magnitude smaller than the value estimated for non-diffusionally controlled plastquinol oxidation [81]. To reconcile the apparent discrepancy between the observed slow plastoquinone diffusion and the notion that plastoquinol oxidation at the Qo site is a non-diffusion-limited process, a microdomain organization has been proposed in which rapid plastoquinone diffusion is limited to small lipid microdomains in the vicinity of active PSII centers [82]. Plastoquinone movement is rapid within these microdomains, which include only a few PSII and Cytb_6f complexes. In contrast, long distance plastoquinone migration is slow because of the crossing of many domain boundaries formed by protein transmembrane regions in the thylakoid membrane. A more elaborate version of the microdomain model has been proposed, which takes into account specific PSII–LHCII supercomplexes and LHCII–LHCII interactions with a heterogeneity in the microdomain organization [83]. In the grana regions, about 70% of PSII is located in small domains with only 1 to 2 PSII, and 30% of PSII is located in larger domains with more than 10 PSII. It is very likely that phosphorylation/dephosphorylation of LHCII and PSII proteins plays an important role in the control of assembly and organization of this microdomain structure. In this respect, a recent study of the *Arabidopsis stn8* mutant deficient in the thylakoid protein kinase STN8 [51,52] has revealed that the folding of the thyalkoid membranes is affected [84]. In this mutant, the loss of light-induced PSII core protein phosphorylation leads to an enlargement of the grana and restricts the lateral migration of the PSII reaction center protein D1 between the grana and stromal membrane

domains during the PSII repair cycle and thereby slows down the turnover of damaged D1 in plants exposed to high light.

6. Impacts of micronutrient limitation on electron flow

Deprivation of micronutrients can profoundly affect photosynthetic electron transfer. Under sulfur deprivation, green algae downregulate PSII activity and oxygen evolution, whereas starch production and respiratory activity are enhanced [85]. This leads to an anaerobic state in which the activities of Cytb_6f and PSI do not decline significantly thus allowing the algal cells to maintain ATP synthesis via cyclic electron transport through Cytb_6f and PSI. Moreover, sulfur deprivation enhances the degradation of starch, proteins, or lipids leading to the release of electrons, which are directed to the plastoquinone pool through the Ndh-2 complex. Because the Calvin–Benson cycle, which acts as a major electron sink, is downregulated under sulfur deprivation, this could generate excess reducing power and oxidative damage. However, in some green algae, the hydrogenase that is induced under anaerobiosis acts as powerful electron valve and disposes of the excess electrons by producing hydrogen [86].

Iron depletion has also drastic effects on the photosynthetic electron transport chain. This is because iron plays a central role in electron transfer reactions due to its ability to donate and accept electrons in heme and iron–sulfur proteins and to act as a cofactor. Iron is involved in all the major photosynthetic complexes PSII, Cytb_6f and PSI. The level of these complexes is reduced under iron limitation with a concomitant decrease in electron transport and reduced carbon fixation rates. With its three 4Fe-4S centers, PSI is particularly sensitive to iron deficiency.

In *C. reinhardtii*, mild iron deficiency leads to a pronounced degradation of PSI and to a remodeling of LHCI reducing thereby the transfer of excitation energy from LHCI to PSI and thus diminishing photooxidative damage [87,88]. Moreover, the functional antenna size of PSII is increased, which is accompanied by increased photoinhibition and inactivation of PSII [89]. This inactivation of PSII centers has been proposed to act as a quencher of excitation energy under severe light stress or iron deprivation and to protect neighboring PSII centers from damage by acting as effective energy sinks thus avoiding over-reduction of the electron transport chain. In cyanobacteria, the ratio of PSI:PSII changes from 4:1 to 1:1 under iron limitation (see [90]). Several adaptive responses occur under these conditions. PSI is modified to enhance light harvesting while limiting photooxidative damage. Iron deficiency induces the degradation of the light-harvesting phycobilisomes [91] and the expression of the "iron stress induced" *isiA* protein, which resembles the chlorophyll *a* binding protein CP43 of PSII [92,93]. This protein forms a ring of 18 molecules around PSI [94,95]. Also, iron-containing electron acceptors such as ferredoxin are replaced by flavodoxin, an iron-independent substitute.

Besides the photosynthetic apparatus, the respiratory electron transport chain is also a major user of iron. Iron limitation has therefore a major impact and leads to a decrease in iron-containing respiratory complexes. It is interesting that for *Chlamydomonas* cells, the impact of iron deficiency depends on the presence or absence of a source of reduced carbon such as acetate in the growth medium. Photoheterotrophically grown cells maintain high respiratory activity at the expense of photosynthesis, whereas phototrophically grown cells keep both photosynthetic and respiratory function under conditions where photoheterotrophic cells lose their photosynthetic capacity [89]. However, phototrophic cells lose their photosynthetic activity when iron becomes limiting [96].

7. Conclusions and perspectives

The past decade has witnessed remarkable advances in the structure determination at the atomic level of the major photosynthetic complexes PSII [3], PSI [4], and their associated light-harvesting systems, the Cytb_6f complex [5,6] and the mitochondrial F1 ATP synthase [97], which is very similar to the chloroplast CF1 ATP synthase. These structures have provided invaluable insights into the function of these complex molecular machines, particularly how they capture light energy and how they mediate electron transfer. These structural studies have been complemented by biochemical, biophysical, and genetic approaches, which have given a more comprehensive picture of the photosynthetic system.

A striking feature of photosynthetic organisms is their ability to adapt to a variety of environmental changes and stress conditions. This is particularly true for plants that are sessile. The picture that emerges from recent studies is that the photosynthetic apparatus does not only provide energy for the cellular metabolism but it also acts as a sensitive sensor. This ability allows the system to adapt to changes in the light environment, to temperature variations and to the availability of carbon sources. In this respect, both forward and reverse genetic approaches have been very rewarding for the identification of key components involved in non-photochemical quenching, in state transitions, in CEF, in chloroplast gene expression, and in chloroplast signaling. A number of protein kinases and phosphatases closely associated with the thylakoid membranes have been identified that orchestrate dynamic changes in the organization of the photosynthetic complexes within these membranes. We still know little about the molecular basis of this dynamics. Some of these kinases such as Stt7/STN7 are activated through changes in the redox state of the plastoquinone pool and their major substrates have been identified. A particularly interesting feature is that the Stt7/STN7 kinase is involved both in short-term responses within the chloroplast, which occur in the second to minute range, and also in long-term responses, in particular retrograde signaling, which occur within the hour to day range. Other chloroplast kinases have been identified such as Stl1/STN8 [49,51,52], CSK [72], TAK [98], and casein kinase CKII [99]. A challenging task will be to identify the components of their respective signaling chains in the chloroplast as well in the other cell compartments and to establish a complete picture of the chloroplast signaling network, which would link these different kinases in a comprehensive scheme.

Finally, powerful global approaches have been developed and used for studying the acclimation of the photosynthetic apparatus. We have entered the omics age with transcriptomics, proteomics, and metabolomics all of which provide large data sets that are not always easy to analyze but convey a global picture of cellular responses. Synthetic biology also offers very promising possibilities to reengineer the photosynthetic apparatus. This will lead to a better understanding of the regulation of photosynthesis and opens a new area in photosynthetic research, the ability to create photosynthetic organisms with novel properties suitable for producing large amounts of compounds of economic interests such as food, pharmaceuticals, and biofuels.

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