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Review

Diverse mechanisms for photoprotection in photosynthesis. Dynamic regulation of photosystem II excitation in response to rapid environmental change

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ABSTRACT

Photosystem II (PSII) of photosynthesis catalyzes one of the most challenging reactions in nature, the light driven oxidation of water and release of molecular oxygen. PSII couples the sequential four step oxidation of water and two step reduction of plastoquinone to single photon photochemistry with charge accumulation centers on both its electron donor and acceptor sides. Photon capture, excitation energy transfer, and trapping occur on a much faster time scale than the subsequent electron transfer and charge accumulation steps. A balance between excitation of PSII and the use of the absorbed energy to drive electron transport is essential. If the absorption of light energy increases and/or the sink capacity for photosynthetically derived electrons decreases, potentially deleterious side reactions may occur, including the production of reactive oxygen species. In response, a myriad of fast (second to minutes timescale) and reversible photoprotective mechanisms are observed to regulate PSII excitation when the environment changes more quickly than can be acclimated to by gene expression. This review compares the diverse photoprotective mechanisms that are used to dissipate (quench) PSII excitation within the antenna systems of higher land plants, green algae, diatoms, and cyanobacteria. The molecular bases of how PSII excitation pressure is sensed by the antenna system and how the antenna then reconfigures itself from a light harvesting to an energy dissipative mode are discussed.

1. Introduction

Photosystem (PS) II is the photosynthetic enzyme found in plants, eukaryotic algae, and cyanobacteria that uses sunlight energy to extract electrons from water. PSII has three functional domains: (i) the *antenna* of chlorophyll (Chl) and other pigments which absorb and transfer photon energy to (ii) the *reaction center* where the excited state electron from a special pair of Chl *a* molecules (P680) is transferred to a series of electron acceptors, the exceptionally strong oxidizing power of $P680^+$ drives (iii) the extraction of electrons from water within the *oxygen evolving complex* (OEC). PSII ultimately supplies biology with the electrons required for the conversion of inorganic molecules into the organic molecules that serve as the building blocks for life. The waste product of PSII activity, oxygen, oxidized the pre-PSII earth, thereafter changing the chemical composition of the planet through newly available geochemical and biochemical reactions. The appearance of oxygen-evolving photosynthesis some 2.5 billion years ago has been described as the "big bang of evolution" because for the first time in earth's history life now had access to an inexhaustible supply of energy in the form of sunlight; biology had solved its energy crisis and PSII established itself as the "engine of life" [1]. However, the PSII engine readily breaks down (rather ironically, via oxidative damage) if the sunlight powering PSII exceeds the engine's turnover capacity for electron processing. Thus, a major obstacle to the evolutionary success of photosynthesis has been the ability of PSII to rapidly balance its excitation by the sun's rays with its de-excitation by electron transport within fluctuating light environments.

Adjustments brought about by gene regulation in the stoichiometries of light harvesting pigments, reaction center proteins, electron transport components, carbon dioxide import/fixation enzymes, and sink capacity for photosynthetic sugar production all offer long-term acclimation to excess light, but operate too slowly to rapidly combat the strong changes in incident light intensity that plants and algae can encounter in nature (i.e. sunflecks, fast vertical movement of aquatic species through the water column, even diurnal changes in solar flux). Higher plants do have a limited ability to quickly regulate light absorption through changes in leaf angle, leaf area (curling), and chloroplast streaming. Non-motile phytoplankton have even fewer strategies at

Abbreviations: APC, allophycocyanin; Chl, chlorophyll; Cyt, cytochrome; ΔpH, trans-thylakoid proton gradient; FCP, fucoxanthin chlorophyll protein; FRP, fluorescence recovery protein; L_{cm}, PBS core-membrane linker polypeptide; LHC, light harvesting complex; LHCII, integral membrane PSII accessory light harvesting protein; MGDG, monogalactosyldiacylglycerol; NPQ, non-photochemical quenching; OEC, oxygen evolving complex; OCP, orange carotenoid protein; PBS, phycobilisome; Pheo, pheophytin; PS, photosystem; PQ, plastoquinone; PQH_2, plastoquinol; PSI, photosystem I; PSII, photosystem II; Q_A, quinone A; Q_B, quinone B; ROS, reactive oxygen species

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their disposal to rapidly decrease light absorption. Two principal strategies have arisen within the chloroplast at the post-translational, molecular level for dissipating (quenching) excess absorbed light energy for the safeguard of PSII. One strategy is to increase photochemical de-excitation of reaction center Chl by either recirculating electrons within the PSII reaction center [2,3], and/or by passing electrons to alternate electron acceptors within the chloroplast [4]. The other strategy is to downregulate PSII excitation at its source within the antenna system, typically via the harmless conversion of absorbed light energy into heat [5,6]. This top-down approach towards regulating PSII excitation is the subject of this communication.

The goal of the following literature review is to offer a summary of the current state of understanding for the "fast" and reversible (second to minutes timeframe) antenna-based PSII photoprotective mechanisms (collectively described as non-photochemical quenching) that are found in higher land plants, green algae, diatoms, and cyanobacteria. Both the molecular mechanisms of how over-excitation of PSII is sensed and how excitation energy is dissipated are discussed in detail. Emphasis is on the diversity of the flexible, post-translational energy dissipative pathways that have evolved in response to the natural fluctuating light environs that PSII inhabits.

2. Photosystem II

2.1. Basics of the core complex and reaction center

PSII is a large multisubunit protein complex situated within the thylakoid membrane. The most recent crystal structure of PSII has been resolved to 1.9 Å [7]. The PSII core complex is composed of the membrane intrinsic D1/D2/CP43/CP47 core proteins, plus the lumen-side extrinsic polypeptides of the OEC, and ten or so small membrane intrinsic polypeptides. D1 and D2 form the skeleton for the heterodimeric reaction center containing the photochemically active Chl *a* molecules and electron transfer cofactors. CP43 and CP47 contain additional Chl *a* molecules and serve as the proximal antenna for transferring excitation energy to the reaction center. There are six reaction center pigments in the D1/D2 dimer: the four central Chl *a* molecules (ChlD1, ChlD2, PD1, PD2; where the PD1 and PD2 form the P680 Chl dimer of the reaction center) and the two pheophytins (PheoD1, PheoD2) [2]. The first charge separation event involves the radical pair $ChlD1^{\cdot+}$ $PheoD1^{\cdot-}$, followed by formation of the more stable $P680^{\cdot+}$ $PheoD1^{\cdot-}$ [2,8]. After electron transfer to Pheo, the photochemically derived electrons are passed along a series of lower energy, spatially separated electron acceptors (from Pheo to Q_A, from Q_A via a bicarbonate associated non-heme iron to the lipid soluble mobile electron carrier PQ within the Q_B binding pocket). Two sequential photon absorption events are required to doubly reduce PQ before electrons can leave PSII carried by PQH_2. $P680^{\cdot+}$ is an exceptionally strong oxidizing agent and is used to power water splitting by the Mn_4CaO cluster of the OEC. During the S-state cycle of the Mn_4Ca cluster, the absorption of four photons by P680 drives the splitting of two water molecules and formation of molecular O_2 through a consecutive series of five intermediates (S_0, S_1, S_2, S_3, and S_4) (for review of Mn_4Ca cluster and water splitting see [9]). Electron donation from the Mn_4Ca cluster to $P680^{\cdot+}$ is aided by the redox active tyrosine Y_Z. The protons released into the lumen by the S cycle and the protons removed from the stroma (or cytosol in cyanobacteria) during the reduction of PQ build the trans-thylakoid proton gradient that powers ATP production. Reviews on the form and function of the PSII reaction center include [2,10–12].

2.2. An introduction to PSII light harvesting complexes and PSII macro-organization

The capture of photon energy is aided by large, pigment-protein light harvesting complexes (LHCs) that associate with the PSII core complex. LHCs greatly enhance the absorbance cross section of PSII over that achieved by the Chls inherent to the PSII core alone. Highly efficient energy transfer among the LHC pigments permits a rapid thermal equilibrium of energy, with excitons settling on the lower energy excited states of the PSII reaction center. The LHC apoproteins must bind pigments at appropriate distances and orientations, and provide localized environments to tune pigment energy levels, so as to promote efficient energy transfer within and between light harvesting complexes and to the PSII core. The light harvesting functions of LHCs are dynamic, switching between an antenna mode for collecting and funneling light energy towards PSII and a photoprotective mode for excitation energy dissipation. LHCs differ among lineages (see [13,14]).

The "auxiliary" antenna of cyanobacteria and red algae is the phycobilisome (PBS). The PBS is associated with the cytosolic face of the thylakoid and is composed of water soluble phycobiliproteins containing covalently attached bilin pigments. The PBS structure is composed of a series of rods containing the phycobiliprotein phycocyanin (and often additional phycobiliproteins) radiating from a core of cylinders composed of the phycobiliprotein allophycocyanin (APC). Linker proteins interconnect the phycobiliproteins and anchor the PBS to the thylakoid. See [15] for details on PBS structure and function.

The LHCs of eukaryotic algae and plants are composed of integral membrane proteins with transmembrane helices (the extended light-harvesting complex superfamily) that contain non-covalently bound Chl *a* plus accessory Chls and xanthophylls (there are important algal exceptions, including the red algae which retain PBS but have photosystem I specific integral membrane light harvesting proteins). The LHCs of the green lineage contain Chl *b*. LHCII are the principal light harvesting complexes for PSII and are encoded by genes of the Lhcb multi-genic family. The apoproteins of LHCII contain three membrane-spanning regions. Binding approximately 80% of the total PSII Chl, trimeric LHCII is the major LHCII (hereafter simply referred to as LHCII) composed of three apoprotein subunits in higher plants (Lhcb1, Lhcb2, and Lhcb3). Additional monomeric antenna complexes form the minor LHCIIs (CP29, CP26, and CP24, the products of the *lhcb4*, *lhcb5*, and *lhcb6* genes in higher plants, respectively). In higher plants, PSII is found in vivo within large PSII—LHCII supercomplexes composed of a dimer of PSII cores (C_2) surrounded by two copies of CP26 and CP29 and two strongly bound LHCII trimers (S_2) forming the C_2S_2 supercomplex. Two CP24, two moderate strongly bound LHCII trimers (M_2), and a varying number of loosely bound LHCII trimers (L_x) associate with C_2S_2 to form $C_2S_2M_2L_x$ megacomplexes, especially in low light grown chloroplasts. For a comprehensive review of the form and function of the major and minor LHCII refer to [16]. The 2-D organization of PSII—LHCII supercomplexes can become 3-D due to contacts between the stromal-facing surfaces of PSII, LHCII, and the minor LHCII within adjacent membranes of grana, resulting in semi-crystalline arrays of PSII—LHCII [17–19]. PSII—LHCII macrostructure and grana stacking are dependent on the presence of Mg^{2+} for masking electrostatic charges [17].

The LHCs of diatoms are composed of FCPs (fucoxanthin Chl proteins). FCPs are heavily enriched in the xanthophyll fucoxanthin and contain Chl *c* as a substitute for Chl *b*. LhcF proteins are the primary constituents of the PSII light harvesting FCP complexes. Analogous to LHCII, the functional unit of FCPs is a trimer ([20]. FCP trimers are structurally and functionally heterogeneous, composed of different combinations of FCP proteins [20], and likely exist in the thylakoid within higher order aggregates [21]. How LHCs are arranged with the photosystems in the diatom thylakoid, though, is largely unknown.

3. The precarious existence of PSII: excitation pressure, photodamage, and NPQ

3.1. Excitation pressure

Environmental and physiological conditions favoring a higher rate of excitation energy reaching P680 than can be dissipated via

photochemistry and forward electron transport leads to heightened "excitation pressure" on PSII. Excitation pressure is formally defined as the relative measure of the reduction state of Q_A, representing the proportion of PSII reaction centers in a closed state (Tyr_Z $P680^+$ Pheo Q_A^- Q_B), and can be measured non-invasively using the saturating light pulse method of Chl fluorometry [22]. Elevated ΔpH (over acidification of the lumen) and over reduction in the redox status of the photosynthetic electron carriers are physiological indicators of high PSII excitation pressure within the chloroplast.

3.2. Photodamage

The PSII proteins (particularly D1) and surrounding lipid milieu are prone to photodamage by reactive oxygen species (ROS) under conditions of heightened excitation pressure. Electron transfer can either be donor side inhibited, as when electrons are not available from water to re-reduce the $P680^+$ radical, or acceptor side inhibited, when electrons are trapped on Q_A or Q_B due to saturated forward electron transfer away from excited state P680 (see reviews by [3,23]). The very high redox potential ($^+$1.2–1.4 V) of the long lived $P680^+$ radical can drive detrimental oxidation reactions in the surrounding photosynthetic apparatus when the donor side is inhibited. Incomplete oxidation of water by the OEC is associated with the formation of H_2O_2, which can be oxidized to the superoxide radical ($O_2^{\bullet-}$) by Tyr_Z or reduced to the hydroxyl radical ($HO^{\bullet}$) by manganese released from the Mn_4Ca cluster. Acceptor side inhibition promotes reverse electron transfer from Q_A to Pheo for charge recombination of the $Pheo^-$ $P680^+$ radical pair. This back reaction results in a radical pair in the singlet state $^1Pheo^-$ $^1P680^+$, which can be converted to the triplet radical pair $^3Pheo^-$ $^3P680^+$. Under conditions of a more negative Q_A/Q_A^- midpoint redox potential, charge recombination of the triplet radical pair ($^3Pheo^-$ $^3P680^+$) results in the formation of triplet excited state P680 ($^3P680^*$). The triplet excitation energy from $^3P680^*$ is readily transferred to the triplet ground state of molecular oxygen ($^3O_2$) forming damaging singlet oxygen ($^1O_2$), which can ultimately lead to the production of other ROS. Singlet oxygen can also form in the PSII antenna complex if there is intersystem crossing of singlet excited state Chl ($^1Chl^*$) to triplet excited state chlorophyll ($^3Chl^*$). The strong coupling between Chl and carotenoids typically found in the membrane integral LHC complexes, such as LHCII, results in excitation energy transfer from $^3Chl^*$ to carotenoid forming the triplet excited carotenoid which relaxes harmlessly emitting heat. For further examination of PSII ROS formation, one is encouraged to refer in detail to the works by Pospíšil [3,23].

Production of ROS by PSII leads not only to PSII- and thylakoid-specific damage (recently reviewed in [24,25]), but also oxidative degradation and damage on a whole cell scale [26]. Irreversible photodamage to PSII leads to the formation of a photoinactivated PSII, requiring lengthy and costly repair processes, principally via the degradation and de novo synthesis of the D1 protein [27–31]. The deleterious effects of ROS are stopped by either dissipating the excess excitation energy before it is transferred to molecular oxygen, or by cleaning up ROS species before extensive damage can incur, via the activity of ROS scavengers and antioxidants [25,32] (Fig. 1). High excitation pressure can stem from daily and seasonal transitions in incident sunlight intensity, and from other environmental factors that affect photosynthetic electron sink capacity such as salt, nutrient, temperature, pathogen, or desiccation stress. If the rate of D1 turnover can't keep up with the rate of PSII photoinactivation, there is a net loss in functional PSII, a condition described as photoinhibition [33] (see Fig. 1).

3.3. Photoprotective, electron-transfer driven mechanisms built into the reaction center

When the PSII reaction center is closed, electrons generated from the PSII acceptor side can be re-circulated by secondary electron transfer (PSII cyclic electron transfer) as a photoprotective mechanism.

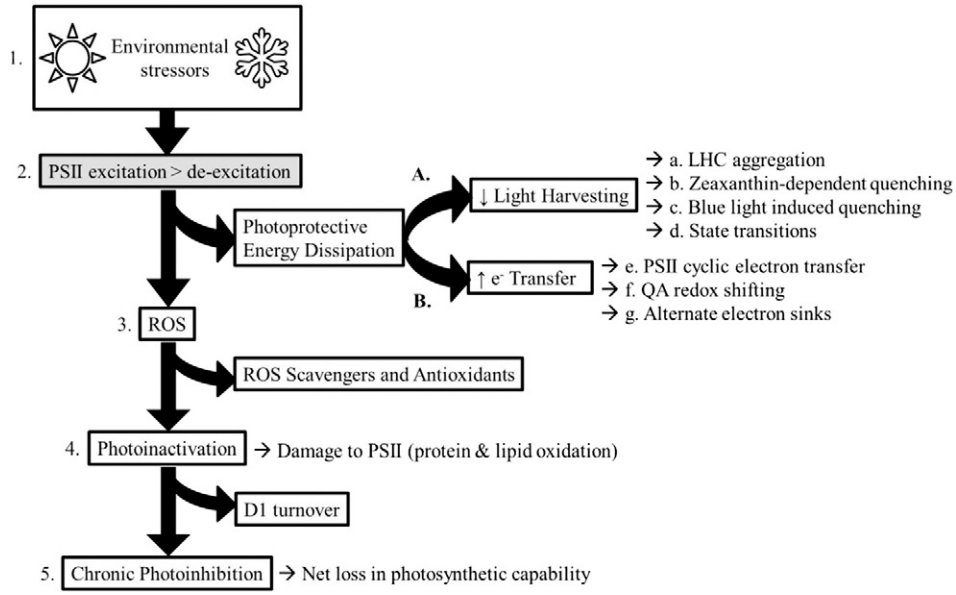

Fig. 1. The fate of PSII under high excitation pressure. Environmental stressors which perturb photostasis (such as high light and cold) (1.) cause the redox state of the PSII quinone electron acceptors to become overly reduced, thereby impeding the photochemical relaxation of P680*, resulting in high PSII excitation pressure (2.). Further excitation of long-lived chlorophyll singlet excited states leads to chlorophyll triplet state formation and ROS production (3.). PSII can become photoinactivated when ROS initiate deleterious oxidative reactions within the PSII protein and lipid milieu (4.). The PSII reaction center is continuously repaired via D1 protein turnover; however, if the rate of PSII photodamage exceeds the rate of PSII repair then there is a net loss in PSII activity (photoinhibition) (5.). Photosynthetic organisms have evolved an assemblage of photoprotective mechanisms to rapidly relieve PSII excitation pressure and steer away from the (2.) to (5.) pathway. In the "photon safety valve" branch of photoprotective mechanisms (A.) the rate of PSII excitation is downregulated at the antenna level through a variety of mechanisms which transiently decrease the PSII effective antenna size. Excitation energy can be converted directly to heat within the antenna system (a., b., c.) or rerouted to PSI (d.). In the "electron safety valve" approach to photoprotection (B.), the rate of PSII de-excitation can be up-regulated within the reaction center by recirculating electrons from the donor side via $Cytb_{559}$ mediated cyclic electron transfer (e.), in effect harmlessly re-reducing $P680^+$ with its own electrons, or by promoting forward electron transfer away from P680* (f.). Outside of PSII, electrons can be shuttled to alternate electron acceptors but with no gain in linear photosynthetic electron transport (g.). Here O_2 can safely act as an electron acceptor within the water–water cycle, chlororespiration, and photorespiration pathways (see [4]; in cyanobacteria, bidirectional Hox hydrogenases and several flavodiiron proteins serve as electron valves (see [160]). See text for further details.

Electrons are shuttled from quinones, through the D2 subunit's $Cytb_{559}$, β-carotene, and the redox active Chl, Chl_{Dz}, so as to reversibly connect the PSII electron acceptor and donor sides, in such providing a source of electrons to reduce oxidized Chl radicals or quench the formation of singlet oxygen [2,10,11,34,35]. Oxidation of the D2 β-carotene may be the initial step in secondary electron transfer [36], and this β-carotene has also been shown to quench triplet Chl *a* [37]. $Cytb_{559}$ exists in three redox forms, high potential (Em 370–400 mV), intermediate potential (Em 170–250 mV), and low potential (Em 50–140 mV) [10]. Regulation of $Cytb_{559}$ redox potential was hypothesized to be controlled by quinone binding at the QC site, but more recently, another quinone binding site, QD, has been discovered and is now thought to be the site of $Cytb_{559}$ oxidation [10].

The redox potential of Q_A is known to shift depending on the PSII protein environment, as via loss in Ca^{2+} from the Mn_4Ca cluster [38,39] or PsbA isoform substitution in cyanobacteria [40] (reviewed in [2,10]). Q_A exists in a low potential (−80 mV) and a high potential form (+65 mV), with some variation between species, and the high potential form being present in PSII prior to the assembly of the Mn_4Ca cluster. The high potential Q_A increases the energy gap between the $P680^+$ Q_A^- and $P680^+$ $Pheo^-$ radical pairs, thereby disfavoring the back reaction, subsequent charge recombination, P680 triplet chlorophyll formation, singlet oxygen generation, and ultimately photodamage (refer back to Section 3.2). Structure based theories for how the Q_A redox potential can shift include: a change in H-bonding to Q_A [41], the bicarbonate ion associated with the reaction center non-heme Fe deprotonating to form carbonate, and PSII protein conformational changes induced by Ca^{2+} binding to the Mn_4Ca cluster (see discussion by [2]). The high potential Q_A is rationalized in terms of a mechanism for protecting PSII from photodamage under conditions where electron donation from water is missing, as during PSII assembly, or inadequate to match $P680^+$ formation, as during times of excessive PSII excitation. The high ΔpH (and acidified lumen) generated during conditions of high PSII excitation pressure would activate the high potential Q_A via low pH stimulated release of Ca^{2+} from the Mn_4Ca cluster [38]. The high potential Q_A form would favor forward electron transport under physiological conditions when the acceptor (stromal) side of PSII is exposed to pH > 7.5 (as in the presence of elevated ΔpH), because Q_A to Q_B electron transfer is proton coupled, whereas Pheo to Q_A electron transfer is not proton coupled [2].

3.4. NPQ

Non-photochemical quenching (NPQ) is a process whereby "excess" excitation energy reaching PSII is not used for photochemistry, but is safely dissipated non-radiatively as heat. Fig. 1 outlines the relationship between PSII excitation pressure, photodamage, and NPQ. NPQ is readily monitored by a decrease in photochemistry and maximal Chl *a* fluorescence emission when measured using the PAM fluorometry with saturating light pulse method (reviewed by [42]). The physiological benefit of NPQ is a lowering of PSII excitation pressure and photodamage, yet how much NPQ, particularly the post-translational mechanisms, prevents photodamage and aids in the survival of plants, algae, and cyanobacteria has been difficult to quantify in vivo. Ruban and Belgio have recently presented a new parameter for quantifying the "protective" NPQ component (pNPQ), and found a near linear relationship between pNPQ and the tolerated light intensity in *Arabidopsis thaliana* plants [43].

There are different subtypes of NPQ that can generally be distinguished based on the time-scale of their induction and relaxation and the mechanisms involved: (i) Fast, reversible energy dissipation mediated through buildup of the trans-thylakoid ΔpH gradient on a seconds timescale (qE), (ii) state transitions on a seconds/minutes time scale (qT), (iii) fast inducing but slower, minutes to hours, to relax xanthophyll (zeaxanthin) dependent energy dissipation (qZ), and (iv) photoinhibition, many minutes to hours (qI) [44,45]. The proposed mechanisms behind qE, qT, and qZ principally involve dynamic changes to the light harvesting function of the PSII antenna. qI involves a loss in the number of active PSII reaction centers from photodamage. The contribution of each type of NPQ towards total non-photochemical quenching capacity differs between taxonomic group, stress conditions, and light history of the individual photosynthetic organism [6,46–48]. As qI provides no photoprotective benefit to active PSII cores [49], is not rapidly reversible, and requires protein synthesis for PSII repair, it will not be considered within the context of the post-translational photoprotective quenching mechanisms discussed in the current communication. Also not included are the longer term "locked in" quenching mechanisms associated with desiccation ("dNPQ") and cold induced dormancy (e.g. desiccation in lichens [50]; over-wintering evergreens [51]). The antenna-based NPQ mechanisms that will be the subject of further discussion are summarized in Table 1.

4. Non-photochemical quenching dependent upon energization across the thylakoid (qE)

qE is the fastest antenna based response to excess high light conditions and is defined as NPQ that is strictly dependent on energization of the thylakoid (i.e. ΔpH). Since qE is the dominant form of NPQ in higher plants, the terms NPQ and qE are sometimes used interchangeably. qE involves a fast (seconds to a few minutes time scale) and reversible macrostructural reorganization of the PSII antenna system in response to a rise in ΔpH, converting the antenna from a light harvesting mode to a light energy dissipative mode [6,52–54]. This conversion is facilitated and modulated by proton sensing amino acid residues and xanthophyll pigments. There are four constituents to the qE "scenario" as defined by [53]: 1) the trigger (protons), 2) site of action (major or minor antenna complexes), 3) mechanics (antenna aggregation/ protein conformational changes), and 4) the quenchers (Chl or xanthophylls). The research literature on NPQ and qE is rich and controversial, and full coverage is beyond the scope of this communication. For more detailed reviews the reader is directed to a recent comprehensive book on non-photochemical quenching [55], which has excellent coverage of a wide range of topics, mechanisms, and perspectives.

4.1. qE in higher plants

4.1.1. ΔpH controlled modulation of the PSII antenna light harvesting/energy dissipation "switch". An overview of the factors involved

Higher plant qE continues to be the subject of intense research, yet much debate remains. A number of mechanisms of ever increasing detail have been proposed. The relative importance of a number of contributing factors varies between models and no consensus has been reached. The process is complex and may well involve more than one of the currently proposed mechanisms [48,56–58].

During low light conditions the conformation of minor and/or major LHCII and the supramolecular arrangement of PSII–LHCII supercomplexes are optimized for the highly efficient collection and transfer of light energy to the PSII cores. The buildup of ΔpH during high light is believed to trigger changes in the conformation of minor and/or major LHCII and the supramolecular arrangement of PSII–LHCII supercomplexes that promote the non-radiative trapping of excitons. Energy dissipation likely occurs at multiple sites which may include the major and minor antenna as well as PSII. The xanthophyll cycle and the Lhcb family-belonging minor PSII polypeptide PsbS are both heavily involved in quenching. Fig. 2A summarizes the qE constituents in higher plants.

Although the presence of PsbS is essential for "fast" qE and the relative amount of PsbS correlates well with the extent of qE, the mechanism is not well understood [59]. Due to its similarities to LHC proteins, PsbS was originally thought to bind pigments, possibly responsible for quenching, although it has become clear that PsbS is likely pigment

Table 1
Summary of strategies for quick-acting and reversible antenna-centered PSII energy dissipation. All quenching mechanisms result in the dissipation of excitation energy *en route* to the PSII core. Question marks (?) denote uncertainty/inconclusiveness. The photosynthetic groups (higher plants, green algae, diatoms, or cyanobacteria) in which a quenching mechanism has been shown to be important are indicated in the far-right column. See text for details.

Quenching mechanism	Antenna target	Quenching procedure	Triggering stimulus	Modulators	Photosynthetic groups
Fast, trans-thylakoid energization dependent (qE)	(major) PSII light harvesting complexes	Protonation induced conformational changes in LHC initiate quencher pigment interactions; quenched LHC may form aggregates and decouple from PSII	ΔpH	Xanthophyll cycle[a], PsbS/LhcsR/LhcX	Higher plants, green algae, diatoms
LHC xanthophyll substitution dependent (qZ)	(minor) PSII light harvesting complexes	Xanthophyll de-epoxidation initiates quencher pigment interactions	ΔpH	Xanthophyll cycle	Higher plants, green algae?, diatoms?
Blue light-mediated	PBS core	PBS terminal emitters quenched by a photo-activated carotenoid	Intense blue-green irradiation	OCP, FRP	Cyanobacteria
State transition LHCII (qT)	LHCII	Reversible phosphorylation of LHCII regulates LHCII migration between PSII and PSI providing balance between PSII and PSI excitation; phosphor-LHCII that remain uncoupled to photosystems may be in a quenched state	PQ pool redox status	Stt7/STN7 kinase, PPH1/TAP38 phosphatase	Higher plants, green algae
State transition PBS (qT)	PBS	Photosystem antenna size regulated by transient PBS docking to PSII and PSI AND/OR the spillover of excitation energy from PSII to PSI	PQ pool redox status	PSI monomerization?, PSII particle arrangement?	Cyanobacteria

[a] The role of a xanthophyll cycle in modulating qE in green algae is not clear.

free [60]. It has been shown that protonation of its two lumen exposed glutamate residues is essential for quenching and, thus, that PsbS operates as a pH sensor for qE [61]. PsbS protonation has been observed to affect thylakoid stacking [62], membrane fluidity [63], and associations between CP29, CP24 and LHCII trimers [64]; hence, it has been proposed that protonation of PsbS triggers thylakoid organizational changes.

The xanthophyll cycle of higher plants has been heavily described (e.g. see reviews of [65,66]). In brief: high light results in acidification of the lumen and the activation of violaxanthin de-epoxidase, which in two steps de-epoxidises violaxanthin (less hydrophobic) to antheraxanthin to zeaxanthin (more hydrophobic) using the co-substrate ascorbate within MGDG enriched regions of the thylakoid; transition to lower light conditions raises stromal pH and preferentially activates zeaxanthin epoxidase for the epoxidation of zeaxanthin back to violaxanthin using NADPH. Zeaxanthin has been proposed to act as an allosteric regulator of the PSII antenna, "priming" the system for protonation activated qE [53,67]. The xanthophyll cycle modulates the extent and speed of qE quenching as well as being involved in a slower, longer lasting zeaxanthin dependent quenching qZ (discussed in Section 5).

The xanthophyll cycle and PsbS appear to work in a complementary fashion to regulate qE over different time scales in response to fluctuations in excitation pressure. A high de-epoxidation state of the xanthophyll cycle pigment pool gives *A. thaliana* plants a faster inducing NPQ, but can slow the subsequent relaxation of NPQ [68]. Such kinetic properties of NPQ provide the light harvesting apparati with a molecular memory (hysteresis) for the "average" high light exposure during the previous minutes or hour, particularly pertinent for plants in natural canopies where light intensity is rather randomly distributed in space and time, so that photoprotection is not lost during temporary drops in ΔpH and antenna de-protonation [46,53,66,69]. This approach gives land plants the adaptive advantage of being able to rapidly regulate the light reactions of photosynthesis in response to fast irradiance fluctuations, such as sunflecks, within the background of diurnal/seasonal rhythms in sunlight. [70] has recently addressed diurnal and seasonal changes in PSII energy allocation under non-steady state photosynthesis in rice. See [71] for an excellent ecophysiological perspective on the central role of the xanthophyll cycle in non-photochemical quenching.

4.1.2. Molecular mechanisms for quenching

As energy transfer efficiencies are high within PSII antenna systems, an increase in an energy loss pathway affecting a single antenna Chl could be sufficient to quench excitation within an entire PSII antenna system. Therefore, relatively minimal changes in the organization of the photosynthetic apparatus could conceptually invoke the large scale quenching observed during qE, and in accordance, quenching mechanisms could act by increasing de-excitation pathways only at specific "quenching sites" within the PSII antenna system. Changes in the efficiency of energy transfer from the bulk antenna pigments to specific quenching sites and/or formation of such sites are thus a common theme in proposed mechanisms for controlling quenching. Quenching in the antenna could be induced either by (i) connecting a Chl molecule to a molecular species with a high rate of internal conversion to the ground state (i.e. a xanthophyll) or by (ii) modifying the molecular configuration and/or environment of the Chl molecule so as to increase the rate of internal conversion. An effective way to accomplish the latter is to increase the energetic coupling between a pair or group of pigments which could generate a variety of different kinds of quenching centers.

4.1.2.1. Quenching by xanthophylls. Since the low energy S_1 state of xanthophylls possess an extremely short lifetime (~10 ps) and lie at a similar energy level as that of the lowest excited state of the Chl Q_y band, xanthophylls are expected to be effective quenchers for excited state Chl. Energy gap calculations showed that the energy level of the violaxanthin S1 state lies above Chl *a* Q_y, whereas zeaxanthin S_1 lies below Chl *a* Q_y, thus in theory allowing the xanthophyll cycle to act as a switch ("molecular gearshift") for converting xanthophylls from a light harvesting (excitation energy donor) function to a quencher (excitation energy acceptor and dissipater) [72]. Yet when recombinant LHCII monomers were measured with femtosecond time-resolved absorption spectroscopy, the lutein, zeaxanthin, and violaxanthin S_1 states were all found to lie well below Chl *a* Q_y [73]. Nevertheless, both incoherent (weak) and coherent (strong) xanthophyll–Chl interactions have been implicated as quenchers.

Coherent Chl–xanthophyll coupling, witnessed as an instantaneous population of xanthophyll S_1 upon Chl excitation, also provides access to energy dissipative states. In an excitonic coupled Chl–xanthophyll dimer, the low lying excited states can have more of a carotenoid than Chl character providing enhanced coupling to the ground state. Evidence for Chl–xanthophyll excitonic interactions during qE has been provided using two-photon excitation in LHCII aggregates and leaves [74–76]. Two-photon excitation measurements on liposomes containing LHCII, PsbS, and zeaxanthin showed an increase of electronic interactions between carotenoid S_1 and Chl states that correlated directly with Chl fluorescence quenching [77]. The disassociation of

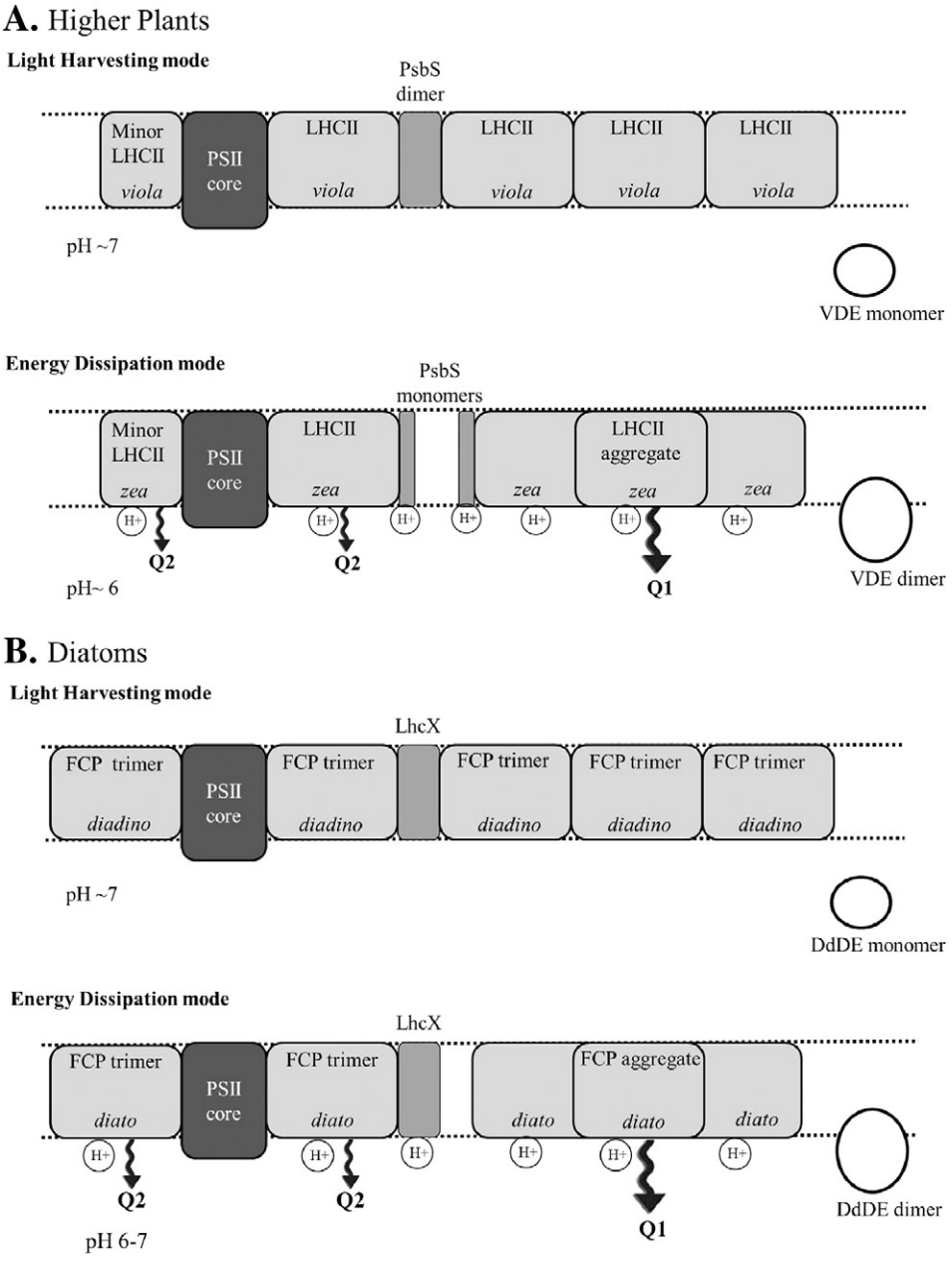

Fig. 2. Simplified cartoon models for antenna-based qE in higher plants (A) and diatoms (B). A. Acidification of the lumen triggers: (i) activation of violaxanthin de-epoxidase (VDE) by dimerization and binding to MDGD-rich regions of the thylakoid, and the two-step de-epoxidation of viola (violaxanthin) into zea (zeaxanthin); (ii) monomerization of PsbS; (iii) protonation of LHCII. The accumulation of zeaxanthin and PsbS monomerization facilitate and modulate the aggregation of LHCII complexes and decoupling of the LHCII aggregates from PSII. Chlorophyll *a*–chlorophyll *a* quenching interactions at the Q2 quenching site are activated by conformational changes within the LHCII aggregates. If there is a pre-accumulation of zeaxanthin, as from previous high light exposure, then quenching can be activated by ΔpH without additional zeaxanthin. Q1 quenching can relax quickly upon a rise in lumenal pH (irrespective of zeaxanthin epoxidation), due to PsbS-mediated dissipation of the LHCII aggregates. Zeaxanthin-dependent quenching (Q2) occurs within the minor LHCII (and possible within LHCII that remain coupled to PSII) and relaxes slowly upon rise in lumenal pH. B. The qE mechanism in diatoms is activated at lower lumenal pHs than in higher plants. Acidification of the lumen triggers: (i) activation of diadinoxanthin de-epoxidase (DdDE) (presumably, as in VDE, by dimerization and binding to MDGD-rich regions of the thylakoid), and the one-step de-epoxidation of diadinoxanthin (diadino) into diatoxanthin (diato); (ii) protonation of FCP antenna proteins (LhcF and/or LhcX?). Diatoxanthin accumulation initiatives the formation of FCP aggregates that become detached from PSII. Analogous to Q1 quenching in higher plants, chlorophyll *a*–chlorophyll *a* quenching interactions are predicted to form from aggregation induced conformational changes in FCP. Q2 quenching in diatoms is assigned to FCP complexes that do not de-couple from PSII. *All* antenna-based qE in diatoms seems to be obligatory on the concurrent accumulation of diatoxanthin. The relaxation of qE is slow in diatoms and can have a strong dependency on diatoxanthin epoxidation (dashed lines represent aqueous interfaces of the thylakoid membrane; H+ represents protonation of lumenal loop exposed residues in integral membrane proteins). See text for details.

the charge transfer state of a coherently coupled xanthophyll–Chl dimer into an anion and cation and subsequent recombination of the charges, provide another pathway for dissipating excitation energy as heat. Monitoring the near infrared absorbance of the xanthophyll cation radical has been used to show that a qE scalable xanthophyll cation (assigned to zeaxanthin) forms upon Chl excitation in isolated thylakoids and PSII minor antenna complexes, but not necessarily in LHCII [53,78–80]. The specificity of the zeaxanthin cation over that of the violaxanthin and lutein cation in fluorescence quenching within LHCII or minor PSII antenna is not yet conclusive [80,81]. Chl–xanthophyll interactions have thus been shown to quench excitation energy in the PSII antenna (ostensibly localized to within the minor antenna), but the relative importance of such quenching pathways towards total energy dissipation during qE is still uncertain. Nevertheless, recently the extent of quenching has been shown to be strongly correlated to the extent of carotenoid–Chl interactions in grana thylakoids [82]. The

mechanistic involvement of carotenoids in qE is currently a very hot topic and more detailed summaries of possible and proposed roles can be found in [59,73,83].

4.1.2.2. Quenching by chlorophyll. Although xanthophylls are innately effective quencher molecules, are they principally responsible for the main dissipation of excitation energy during qE? When in organic solvent or thin films, fluorescence at Chl *a* concentrations equivalent to those found in LHCII (~0.6 M) is heavily quenched due to concentration quenching effects [84]. The protein scaffold within the light harvesting complexes controls concentration quenching. The challenge of how to manage Chl concentration quenching must have been fundamental to the early evolution of photosynthetic light harvesting complexes. Is the qE mechanism a response by natural selection to provide a means of regulating the extent of concentration quenching in response to excitation pressure?

Isolated LHCII trimers self-quench upon aggregation/oligomerization and exhibit spectroscopic properties characteristic of a poorly fluorescing Chl *a*–Chl *a* mixed exciton charge transfer state. In aggregated LHCII (as compared to trimers), there is a relative loss in short wavelength emission (loss in F680 at 77 K) coupled to a relative increase in long wavelength emissions (gain in F700 and a far-red shoulder at 77 K), and a red shift in the Chl *a* Q_y absorption band (see [85,86]). The strong electron–phonon (vibronic) coupling of the Chl *a*–Chl *a* mixed charge transfer state enhances coupling to the ground state and could provide a route for quenching [86]. LHCII has a number of Chls that are located at or close to the surface of the protein [87] that may support the formation of Chl *a*–Chl *a* coherent interactions between adjoining trimers during LHCII aggregation [86]. Fluorescence from the mixed exciton charge transfer Chl *a* terminal emitter in aggregated LHCII has a characteristic lifetime of ~400 ps; a very similar far-red enhanced emission with a lifetime of 400 ps was also observed in intact *Arabidopsis* leaves only under NPQ conditions [86]. Recently, [88] provided evidence using single molecule spectroscopy of isolated LHCII complexes under in vitro qE conditions that the F700 fluorescence band is the result of a conformational shift of the LHCII to access lower-energy states, and that F700 itself is not involved in non-radiative dissipation of excitation energy. Instead, the emissive states at 680–685 nm and >760 nm showed the two largest amounts of energy dissipation [88]. For more on single molecule approaches see [89].

4.1.2.3. LHCII aggregation. LHCII aggregation is one of the earliest proposed mechanisms of quenching [90] and remains a popular model [53]. When LHCII trimer preparations are aggregated in vitro, as via low detergent concentrations, fluorescence of the LHCII oligomers is heavily quenched, up to a ten-fold decrease in fluorescence yield at 77 K and a comparable shortening of fluorescence lifetime (e.g. [86,91]). Quenching is also observed in LHCII aggregates that are directly isolated from thylakoid membranes containing LHCII aggregated in vivo [92] and in LHCII crystals [5]. Quenching in LHCII aggregates was further shown to be sensitive to low pH [93]. These observations led investigators to postulate that qE also involves the aggregation of LHCII. Theoretical calculations for energy transfer in the PSII antenna and reaction center landscapes have suggested that the major LHCII would act as the most effective site for energy quenching [94]. The Horton LHCII aggregation model (as updated in [53] describes four different structural/functional states of the LHCII antenna corresponding to different degrees of energy dissipation. Freeze fracture electron microscopy of intact spinach thylakoids has shown that different qE conditions correspond to differing aggregation states of LHCII particles [95]. qE activation is correlated with a characteristic change in absorbance (Δ535 nm) that has been proposed to result from a red shift in the energy level of a specific sub-population of xanthophyll molecules, and attributed to a geometric change in zeaxanthin [96,97]. Theoretical calculations based on the LHCII structure have suggested that this qE-characteristic absorbance change may stem from the interaction of xanthophyll molecules between adjacent LHCII trimers and thus be a "marker of aggregation" between LHCII trimers upon qE formation [98]. In the LHCII aggregation model, PsbS and xanthophyll de-epoxidation state are proposed to regulate qE by modulating the sensitivity of LHCII to protons. The most recent version of the aggregation model hypothesizes that protonation of acid residues effectively screens charges on the lumenal side of the thylakoid allowing for increased associations between LHCII proteins within the grana. "Condensed" LHCII states are proposed to facilitate the energy dissipative pathways (especially Chl *a*–Chl *a* charge transfer) as discussed above. See [57] for more detail.

4.1.2.4. Location of quenching. Is quenching centered within the minor or major LHCII? The minor LHCIIs (CP29, CP26, and CP24) have been implicated in qE based on their proton and xanthophyll binding sites and their strong quenching properties in vitro (see [53]). The Holzwarth group, using picosecond Chl fluorescence decay spectroscopy of intact leaves, has advocated that two distinct quenching sites exist in the PSII antenna of higher plants: the Q1 quenching site located within aggregated LHCII that become detached from PSII during high light conditions, and the Q2 quenching site located in the antenna that remains coupled to PSII including the minor PSII LHCs (as recently reviewed in [58,66]. Q1 quenching is PsbS-dependent and has been implicated as performing the fast forming and fast reversing ΔpH dependent qE, while Q2 has been assigned to a slower, zeaxanthin-dependent qE that remains in the presence of low ΔpH [66] (also see Section 5).

4.2. qE in green algae

Like higher plants, qE in green algae typically requires the de-epoxidation of zeaxanthin into violaxanthin, a proton gradient, and changes in photosynthetic complex organization [6]; however, there are a few important differences between these members of the green lineage. In many green algae PsbS is replaced by ancient members of the LHC protein superfamily, the stress related LhcsR proteins [99–101]. Furthermore in contrast to higher plants, significant amounts of quenching are induced only after high light acclimation and are not considered constitutive [99,100,102].

In the model green alga *Chlamydomonas reinhardtii*, pigment binding LhcsR has pH sensing abilities, is a strong quencher of Chl fluorescence, and is active in the formation of a carotenoid cation with quenching implications [100]. A conformational change induced by protonation of the C-terminus subdomain has been proposed as a reversible switch for converting LhcR from a light harvesting state to a dissipative state during acidification of the lumen [103]. Dissipative PSII centers isolated from *C. reinhardtii* have been found to contain LhcsR [104].

The role of the violaxanthin–zeaxanthin cycle is not clear in this algal group. No de-epoxidase gene has been located in *C. reinhardtii* [105] and not all green algae exhibit a zeaxanthin dependent NPQ [102]. The Prasinophyte, *Mantoniella squamata*, exhibits a modified violaxanthin–zeaxanthin cycle in which antheraxanthin, not zeaxanthin, accumulates during high light exposure, due to the slow de-epoxidation of antheraxanthin combined with a fast epoxidation of zeaxanthin [106,107].

4.3. qE in mosses

An evolutionary intermediate between the LhcsR modulated qE of the green algae (see Section 4.4) and the PsbS and zeaxanthin modulated qE found in higher land plants (see Section 4.1.1) has been identified in the moss *Physcomitrella patens*. This moss contains both PsbS and LhcsR proteins [108]. The development of PsbS and LhcsR mutants has been used to show that the two proteins are involved in different NPQ mechanisms [108,109]. Violaxanthin de-epoxidase mutants have more recently shown the importance of zeaxanthin during qE in *P. patens* [110]. These findings have led to the proposition that the PsbS and

zeaxanthin mediated qE response gave a fitness advantage to early land plants, thereby the non-constituent, LhcsR based qE mechanism was lost during the colonization of land by higher plants [6,108–110].

4.4. qE in diatoms

Diatoms are capable of performing a high amplitude qE ("super NPQ") with Stern–Volmer *NPQ* quenching values approaching 5× that of a typical higher plant. Diatoms do not have a state transition [111] (see review by [112]), so they rely on qE for regulating the light phase of photosynthesis. qE in diatoms is less understood as compared to higher plants, but has been heavily investigated in recent years. Diatoms acquired their chloroplast by secondary endosymbiosis from a red algal ancestor bestowing them a thylakoid arrangement distinctive from that of the green lineage, most notably there being no grana stacking [112]. "Super NPQ" is only found in diatom species originating from fluctuating environments [113] or when cells are cultured under intermittent high light/low light regimes [114]. qE is induced on a similar time scale as plant qE but takes much longer to relax fully. As opposed to the xanthophyll cycle in plants, the xanthophyll cycle in diatoms involves the one-step de-epoxidation of diadinoxanthin to diatoxanthin. Diadinoxanthin is more soluble in MDGD than violaxanthin [115], diadinoxanthin de-epoxidase is active at higher pHs and has a higher binding coefficient for ascorbate than violaxanthin de-epoxidase [116,117], plus diatoxanthin epoxidase is strongly inhibited by ΔpH [118]. The strength of diatom NPQ could be due to the xanthophyll cycle enzyme activation/deactivation and turnover kinetics, and/or the diatom antenna system may contain more quenching sites or the individual quenchers may be stronger than that in higher plants. qE can be induced in the dark in diatoms via the generation of a weak ΔpH by a chlororespiratory pathway utilizing the plastoquinol pool [119,120].

Although the LhcsR-like LhcX proteins were shown to be pivotal modulators of NPQ amplitude [121], the proton sensor for diatom qE has not been conclusively identified. LhcX proteins can have no glutamate residues exposed in their lumenal loop, yet many LhcF proteins do, leading to the conclusion that FCPs might act as their own proton sensor [122]. However, the one glutamate residue in the lumenal loop of the LhcF and LhcX polypeptides could be masked by negatively charged lipids *in thylakoid* [123]. The expression of LhcX proteins has been shown to react to changes in light intensity [121,124–129], nutrient conditions [130–132], and temperature [133,134]. In *Phaeodactylum tricornutum*, LhcX4 is upregulated in darkness [129] and exhibits a circadian expression pattern [128]. The expression of LhcX proteins does seem to be an important stress response by both centric and pennate diatoms [6]. LhcX proteins have also been shown to incorporate with LhcF proteins within FCP complexes [135,136]. Perhaps the role of LhcX in diatom qE is to promote thylakoid fluidity in inter-antenna complex interactions in a manner which supports the formation of energy dissipative complexes in response to diadinoxanthin de-epoxidation and ΔpH.

The apparent lack of sub-contracting pH sensing to a non-light harvesting protein, such as PsbS, in diatoms has made them heavily dependent on the xanthophyll cycle for modulating qE. Diadinoxanthin de-epoxidation seems to be an obligatory requirement for the induction of qE [137,138], although under certain conditions a rapid NPQ can be induced in the presence of the diadinoxanthin epoxidase inhibitor dithiothreitol [120,139]. The diadinoxanthin/diatoxanthin pigments seem to exist in three distinct pools [6,135]. Pool 1 is bound to LhcF proteins of the FCP (possibly including LhcX) and increases in response to high light with a complex 77 K resonance Raman spectral fingerprint [140]. Pool 2 is found "free" within the MDGD lipid phase of the thylakoid in close association with FCP proteins and may function as an antioxidant in the thylakoid, also increasing in response to high light [135]. Pool 3 is bound to the PSI light harvesting FCP, LhcR, with no apparent response to high light [135].

Quenching has been postulated to involve either direct quenching by diatoxanthin and/or involve Chl *a*–Chl *a* interactions stemming from the aggregation of FCPs [6,114,141,142], much analogous to the pathways described for plant qE (see Section 4.1.2). The energy levels of both diadinoxanthin and diatoxanthin were shown to be lower than the Chl *a* Q_y transition [143]. In such, direct quenching by diatoxanthin would only be possible if qE induced changes in the local protein environment that change the energy level of diatoxanthin or cause configuration changes between diatoxanthin and Chl *a* that promote energy transfer. A diatoxanthin–Chl *a* radical pair might also form, analogous to that discussed for higher plants (see Section 4.1.2), where thermal energy dissipation would stem from the recombination of a Chl a^- radical–diatoxanthin$^+$ radical charge transfer state [6]. Quenching via the formation of a Chl *a*–fucoxanthin charge transfer state in oligomeric FCP complexes has also been proposed [144]. A far red fluorescence component associated with NPQ [120,122,142,145,146] could indicate the presence of a Chl *a*–Chl *a* mixed charge transfer state brought about by inter/intra-FCP complex conformational changes, analogous to that proposed during LHCII aggregation [86].

The picosecond time resolved fluorescence study by Miloslavina et al. assigned two different types of quenching sites (Q1 and Q2) in *P. tricornutum* and *Cyclotella meneghiniana* cells [142]. Parallel to Q1 quenching in higher plants [66] (Section 4.1.2), Q1 quenching from Chl *a*–Chl *a* interactions was hypothesized to occur within aggregated FCP complexes that become decoupled from PSII [142]. Q2 quenching was said to occur within FCP complexes that remain coupled to PSII and be dependent on diatoxanthin [142]. In vitro measurements on trimeric (FCPA) and oligomeric FCP complexes (FCPB) [147] did not exhibit the same time resolved signatures of qE that were observed in vivo by [142]. However, oligomerization of trimeric FCP complexes (FCPA) has recently been shown to give rise to Q1-type red shifted fluorescence signatures in vitro [144]. The time resolved fluorescence study on *C. meneghiniana* by [148] also identified two quenching sites. qE_1 was assigned to quenching within FCPA that remain attached to PSII and within FCPA aggregates that form and become detached from PSII during the buildup of ΔpH; qE_1 rapidly relaxes in the dark [148]. qE_2 was assigned to diatoxanthin dependent quenching within aggregated FCPA and isolated PSII cores that does not relax in the dark [148]. FCP complexes have been shown to self-quench in response to detergent [142,149] and liposome [122] induced aggregation, and low pH [122,149], with quenching enhanced by high diatoxanthin to diadinoxanthin ratios ([122,123,149]) and Mg^{2+} being important in the quenching of FCP from *P. tricornutum* [123]. Based on such experimental results, updated models for Q1 and Q2 quenching in diatoms have been presented [6,150] (Fig. 2B). The heterogeneity observed in qE among diatom species may stem from the different LhcF (and LhcX) protein compositions/combinations in FCPs [20,136,151], and the differing oligomeric organization of FCP complexes in the thylakoid, most notably between the pennate (*P. tricornutum*) and centric (*C. meneghiniana*) clades [21].

A previous high light exposure does not seem to pre-prime qE in diatoms — epoxidation is still required to activate quenching, and reversal of qE can have a strong dependency on diatoxanthin epoxidation especially when the NPQ response is driven to saturation [120]. The qE state appears to be relatively stable as compared to higher plants, requiring diatoxanthin epoxidation or complete dissipation of ΔpH (as via treatments with an uncoupler agent) to "loosen up" the antenna from its quenched state [114,120]. The high amplitude, slow relaxing NPQ in this algal group nevertheless must be highly effective, as diatoms are very successful in environments with turbulent water conditions [152].

5. Zeaxanthin-dependent quenching (qZ) in higher plants

A zeaxanthin-dependent mechanism of NPQ that is PsbS independent and remains after ΔpH has dropped has been shown to contribute to total NPQ in some higher plants such as *A. thaliana* [153]. This zeaxanthin dependent quenching (qZ) both accumulates and relaxes on a time scale of 10–20 min, substantially slower than qE [153]. Crystal

structures have placed violaxanthin binding sites in the major trimeric LHCII complex [87] and in the minor monomeric antenna complex (CP29) [16]. It has been suggested that violaxanthin in the major LHCII is accessible for de-epoxidation, yet in the minor LHCII violaxanthin is tightly bound (maybe too tightly) to be available for de-epoxidation [154,155]. The slow time scale of qZ has led to the hypothesized location of qZ xanthophylls to within CP29/CP26 [153,156–158]. In this hypothesis qE xanthophylls are placed within LHCII, whereas the qZ xanthophylls have been placed within the PSII minor antenna. qZ and Q2 quenching may be describing the same form of quenching — zeaxanthin dependent qE within the PSII minor antenna. A combination of qZ and qE has been proposed to provide long-term PSII photoprotection in over wintering evergreens [51]. Recent work has, however, correlated the slow quenching observed in the *A. thaliana* PsbS-less *npq4* mutant to the presence of the *phot2* gene responsible for chloroplast photorelocation [159], and thus challenged the existence of an independent qZ. The situation is complex, as slow relaxing qE components are also observed in diatoms and other algae suggesting that they may follow a qZ type of mechanism, albeit the xanthophyll pigments can be different (i.e. diatoxanthin accumulation in diatoms as opposed to zeaxanthin accumulation, see Section 4.4).

6. Orange carotenoid protein (OCP) mediated quenching in cyanobacteria

Antenna mediated NPQ in cyanobacteria is not triggered via ΔpH. Orange carotenoid protein (OCP) mediated quenching is directly photo-activated by intense illumination. The lack of a NPQ ΔpH feedback loop for sensing the redox balance of photosynthetic electron transport in cyanobacteria may be a consequence of the thylakoid being shared in both cellular respiration and photosynthetic electron transport and proton translocation pathways (see [160]). Remarkably, the photoprotective role of OCP has been "discovered" and revealed in detail only within the last decade (reviewed by [161]). Details of the quenching mechanism have importantly been elucidated by in vitro reconstitution studies of cyanobacterial PBS quenching (firstly by [162]).

Quenching of the PBS is mediated through OCP, and reversal of quenching is facilitated by the fluorescence recovery protein (FRP) [161,163]. OCP is a water soluble, photoactive protein with the N- and C-termini bridged by the carotenoid 3′-hydroxyechinenone (hECN) [164,165]. In the dark, the inactivated orange colored form of OCP (OCP^O) takes on a "closed" conformation stabilized by the presence of the Arg155–Glu244 salt bridge [163]. Upon absorption of blue-green light, the conjugation length of hECN increases by about one conjugated bond and takes a more planar structure [166], breaking the salt bridge, and converting OCP^O to the metastable red colored activated form OCP^R [163]. The more "opened" conformation of OCP^R would aid in attachment to PBS and exposure of hECN to the PBS chromophores [163,167]. Arg155 has been shown to play an essential role in the interaction between OCP^R and the PBS [167]; perhaps the positive charge is attracted to a negative charge of the PBS [163]. Binding of OCP^R to the PBS stabilizes OCP^R [162]. The C-terminal domain of OCP has been hypothesized to be the site of the photo-switch of the protein, dynamically regulating the photoprotective activity of the otherwise constitutively active carotenoid binding N-terminal domain [168]. In darkness, FRP facilitates the deactivation of OCP^R to OCP^O, detachment from the PBS, and fluorescence recovery [169–171].

OCP^R binds to the PBS core and preferentially quenches APC emission [172–178], although the exact binding location and identification of the APC bilin which interacts with hECN are disputed. Zhang and colleagues have recently used protein chemical cross-linking experiments combined with mass spectrometry analyses on in vitro reconstitutions of OCP and PBS from *Synechocystis* sp. PCC 6803, to reveal that the N-terminal domain of the OCP is closely involved in the association with a site formed by two APC trimers (APC_{660} and APC_{680}) of the basal cylinders of the PBS core [178]. In this docking arrangement, the OCP C-terminus would remain solvent accessible for interaction with FRP [178]. The cross-linking study by [178] suggests that excitation energy would be trapped by the OCP quencher from the spatially closer APC_{660}. This is in contrast to the earlier work by [175] from the same organism, which proposed the terminal emitter containing core membrane linker protein L_{cm} as the docking and quenching site for OCP. However, the studies of [174,176] supported APC_{660} as the quenched bilin. [179] using non-linear laser fluorometry proposed that both APC_{660} and APC_{680} can be quenched. Interestingly, the Zhang et al. study [178] reported that OCP^O exists as a dimer and that OCP^R is found as a monomer.

The photo-physical mechanism for quenching by OCP^R may either involve a charge transfer from the quenched APC_{660} bilin to hECN (as advocated by [173] using spectrally resolved picosecond fluorescence measurements from intact cells), or involve excitation energy transfer to the pronounced intramolecular charge transfer (ICT) state of excited hECN (as suggested by the transient absorbance studies on isolated OCP by [180,181]). The excited state potential energy profile of hECN is modified in OCP^R, as compared to OCP^O, showing an ICT/S_1 state with increased ICT character and a S_1/ICT state with increased S_1 character [181]. Since the ICT/S_1 state has a lifetime approximately three orders of magnitude shorter than that of the bilin excited state, activated hECN in OCP^R would be able to serve as a very efficient quencher [181]. This is in line with the fast rate constant for molecular quenching of quenched APC_{660} (at most $240 \pm 60\ fs^{-1}$) and the high effective quenching capacity of the quencher measured in vivo, in which more than 80% of the excitations in the PBS were prevented from reaching PSI and PSII [173]. There is a need for in vitro femtosecond transient absorbance studies on OCP–PBS complexes to resolve the quenching interaction between the APC bilin and hECN. The quenching observed by [182] in mutant *Synechocystis* PCC6803 cells was strongly temperature dependent, perhaps signifying the importance of back energy transfer from APC terminal emitters to the hECN quenching trap. As one OCP quenches one PBS [162], and since both in vitro [162] and in vivo [170,183] experiments have shown OCP^R binding to PBS to be light-independent, the rate and amplitude dependence of PBS quenching on light intensity/duration seems to be due exclusively to the amount of OCP^R that accumulates during the illumination period [163].

An additional role for OCP in photoprotection has been witnessed under orange-red light illumination conditions, whereby OCP does not interact with the PBS, but behaves as an effective singlet oxygen quencher [184]. There has recently been the report of a cyanobacterial qE mechanism that is ΔpH controlled localized within the PSII core complex of *Synechococcus* sp. PCC 7942 [185]. This quenching may have been elusive to past investigators due to the fast kinetics of cyanobacterial state transitions (see Section 7.3) and OCP quenching. Such a type of quenching had previously been suggested in iron starved cells from the aggregation of CP43′ [186].

7. State transitions (qT)

State transitions (qT) redistribute excitation energy between PSII and PSI in response to illumination quality that causes an imbalance in excitation between the two photosystems, limiting the over-reduction or over-oxidation of the intersystem electron carriers and possible photo-oxidative damage. State 1 refers to the antenna arrangement favoring PSII excitation, whereas in state 2 PSI is preferentially excited. In green algae and higher plants, LHCII delivers energy absorbed by Chl *b* (blue-green photons) and Chl *a* to PSII, whereas Chl *a*, including a few far red absorbing Chl *a* dominates the antenna of PSI. In cyanobacteria, the phycobilins of the PBS are the primary antenna for PSII with PSI excitation more exclusively from the Chl *a* of the PSI proteins. For photosynthetic organisms living in the shade of other photosynthesizers, such as plants living under the canopy or algae and cyanobacteria dwelling deep in the water column, their incident light quality may be attenuated by changes in the absorptivity of their neighbors above. Changes in illumination color that favor excitation of

the PSII antenna cause an over-reduction of the intersystem electron carriers; conversely, preferential excitation of PSI can cause over-oxidation of the PQ pool, both conditions affecting the overall efficiency of photosynthesis under light limiting conditions. Over-reduction of intersystem electron carriers may also result in potentially dangerous side reactions with molecular oxygen. The redox status of the PQ pool is the signal for the induction of state transitions. A transition to state 2 upregulates PSI excitation at the expense of PSII excitation, increasing the "pull" of electrons from the PQ pool and restoring redox balance; conversely, a transition to state 1 increases to "push" of electrons into the PQ pool. State transitions in green algae and higher plants occur through LHCII movement in a manner distinct from that used in qE, wherein the reversible phosphorylation of the antenna proteins regulate the migration of a subpopulation of LHCII from PSII enriched grana regions to PSI enriched stroma exposed regions. Protein kinase activity is regulated by the binding of PQH_2 to the Q_o site of the $Cytb_6f$ complex. In cyanobacteria, there is a movement of PBS across the stromal surface of the thylakoid. Antenna arrangement in cyanobacteria is hypothesized to be regulated by the extent of PSI trimerization. In addition to the redistribution of excitation energy via the migration of mobile light harvesting complexes, there can also be a passive transfer of excitation energy from PSII to PSI if the PSII and PSI antenna are connected when excitation energy not trapped by the PSII reaction center "spillovers" to PSI. Recent reviews on state transitions include [187] (cyanobacteria), [45,188–190].

7.1. State transitions in higher plants

The results of biochemical, electron microscopy, and spectroscopic analyses of the state transition in higher plants have led to the development of a two-process model for describing the state 1 to state 2 transition; pivotal was the recent development of artificial microRNA (amiRNA) *A. thaliana* lines deficient in either Lhcb1 or Lhcb2 expression. Antenna remodeling during state transitions in higher plants is outlined in Fig. 3A.

PQH_2 binding to the $Cytb_6f$ Q_o site activates the chloroplast serine–threonine protein kinase STN7, resulting in phosphorylation of LHCII [45,191,192]. Conformational changes to the Rieske protein of $Cytb_6f$ complex upon PQH_2 binding to the Q_o site are suggested to be sensed by the single-membrane spanning domain of STN7 [191,193]. The kinase is predicted to be activated when the Q_o site is bound and the Rieske protein is in its "proximal" position; the kinase is deactivated when the Q_o site is emptied and the Rieske protein moves to the "distal" position [194,195]. STN7 is also predicted to be active as a dimer [196]. STN7 is presumed to be the kinase that directly phosphorylates LHCII, although other kinases have not been formally excluded, including the STN7 homologous kinase STN8, which has much lower activity than STN7 and targets PSII core proteins [192,197–199]. Although LHCII can be phosphorylated at several sites [198,200], phosphorylation at an N-terminus threonine residue in Lhcb1 and Lhcb2 is the site that is engaged in state transitions [191]. Lhcb2 seems to be the vital target for STN7 in land plants, even though Lhcb1 phosphorylation is important during the state transition in *A. thaliana* [199]. STN7 has a higher affinity for phosphorylating Lhcb2 than Lhcb1, and the Lhcb2 phosphorylation site is highly conserved among land plants [192]. Also in many land plant species, the Lhcb1 gene products lack a phosphorylatable threonine residue [192], indicating low selection pressure to maintain Lhcb1 phosphorylation [199]. Lhcb2 and Lhcb1 phosphorylation of the PSII–LHCII $C_2S_2M_2$ supercomplex and $C_2S_2M_2L_x$ megacomplex [201] is predicted to loosen grana packing by charge repulsion, and cause a release of the "extra" LHCII trimers that are held loosely in the megacomplexes. Since migration is predominantly by the loose LHCII pool, the $C_2S_2M_2$ supercomplex and the semi-crystalline PSII arrays are left intact in state 2 [201].

A sub-population of the now detached "extra" LHCII trimers containing $(Lhcb1)_2Lhcb2$ heterotrimers with phosphorylated Lhcb2, bind intimately with PSI and serve as highly efficient antenna for PSI [199,202], with phospho-Lhcb2 binding predicted at the PsaH, PsaL, and PsaO polypeptide domains of PSI in accordance with [203]. Phospho-LHCII trimers not containing phospho-Lhcb2, but only phospho-Lhcb1, do not interact with PSI and would remain as detached antenna [199]. Such an antenna arrangement would decrease the PSII absorbance cross section without increasing PSI antenna size and may describe the slow "state transition" observed in the Lhcb2 deficient mutant by [199]. An updated PSI–LHCII structural model [202] suggests that the low-energy Chls 611 and 612 in LHCII interact with the Chl 11145 at the interface of PSI, and that mobile LHCIIs are, in fact, an important constituent of the PSI antenna system. In seed plants, only ~25% of LHCII is phosphorylated in state 2 [204]. This is in good agreement with the hypothesis that $(Lhcb1)_2$ phospho-Lhcb2 heterotrimers only interact with PSI, since $(Lhcb1)_2Lhcb2$ heterotrimers are believed to be only a minor component of LHCII, perhaps only 20 to 30% of all LHCII trimers [199]. The induction of qT is on the minutes time scale as compared to seconds for qE, entailing that the state transition is involved with longer-term acclimation than qE. It is currently unclear if the LHCII trimer pools involved in qE and qT are shared or distinct. The net movement of the charged LHCII may contribute to the decrease in grana stack layers that occurs in state 2 adapted chloroplasts [199,205], yet nevertheless, grana stacking can also decrease in state 2 conditions in the absence of a functional state transition [199].

State 2 relaxes when phospho-LHCII is dephosphorylated by the TAP38/PPH1 phosphatase (with no preference towards Lhcb1 or Lhcb2) [192]. The extent of LHCII phosphorylation is hypothesized to be modulated by the redox state of the PQ pool via the slow, constitutive action of TAP38/PPH1. Oxidation of the PQ pool deactivates STN7, favoring the net de-phosphorylation of LHCII.

A "state transition" independent of LHCII phosphorylation may occur during the qE response. In isolated spinach thylakoids, protonation of PsbS has been shown to be able to regulate excitation energy distribution via excitation energy spillover from PSII to PSI [206]. This observation was at first attributed to a redistribution of LHCII during qE that resulted in favored PSI excitation [207,208].

Remodeling of the PSII supercomplex from phosphorylation of PSII core proteins can also occur on a similar time scale as the state 1 to state 2 transition in *A. thaliana* plants, but this remodeling has no direct role in LHCII phosphorylation, or LHCII migration, or change in thylakoid membrane topology [205]. Phosphorylation of the D1 and D2 PSII core proteins requires the STN8 kinase, whereas phosphorylation of CP43 seems to require STN7 [205]. To further confound the role of phosphorylation during the state transition, there are a multitude of other molecular pathways that involve the phosphorylation of PSII–LHCII proteins during chloroplast genesis, thylakoid stacking regulation, repair of photodamaged PSII, and stress responses [209–212].

It has been generally accepted that phospho-LHCII is only associated with PSI as a short-term response to the over-excitation of PSII during the state transition; however, phospho-LHCII has recently been shown to serve as an antenna for PSI during long-term acclimation to a variety of natural light environments [213]. In fact, time-resolved fluorescence measurements showed that LHCII is even a more efficient light harvester in *A. thaliana* thylakoids when associated with PSI than it is when associated with PSII [213]. Thus, the dramatic remodeling of the antenna system observed during laboratory induced state transitions (e.g. using far-red illumination) may not accurately represent the true nature of LHCII distribution under natural conditions.

7.2. State transitions in green algae

In *C. reinhardtii*, three forms of PSII complexes have been identified: PSII core complex, PSII–LHCII supercomplex, and a PSII megacomplex containing multiple LHCIIs [214]. The megacomplex forms in state 1, whereas the PSII core predominates in state 2 [214]. PSII remodeling during the transition to state 2 was proposed to arise from the

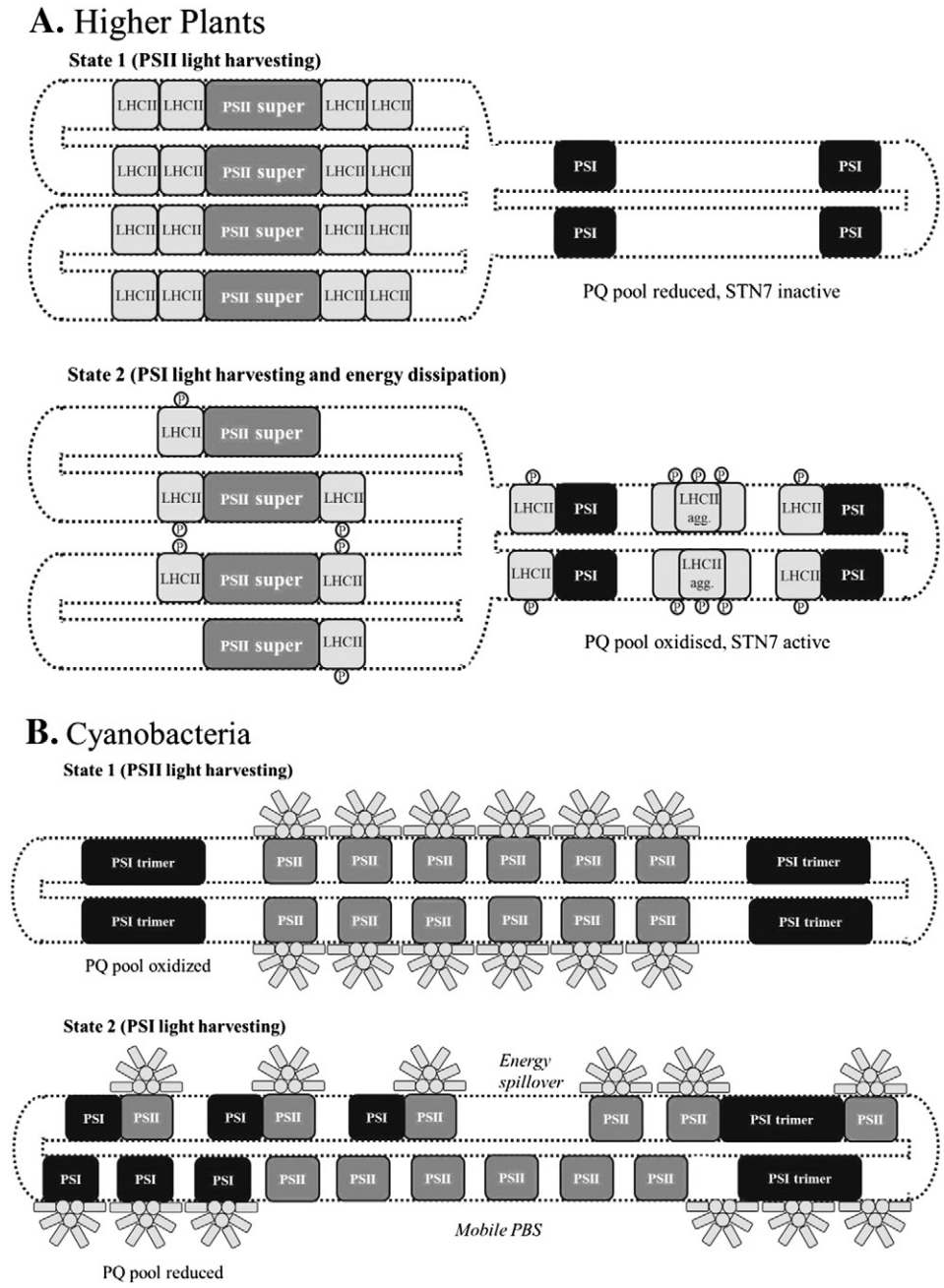

Fig. 3. Simplified cartoon models for antenna rearrangement during the state transition in higher plants (A) and cyanobacteria (B). A. In higher plants, PQH_2 binding to the Q_o site of the $Cytb_6f$ complex activates the STN7 protein kinase during state 2 conditions. STN7 phosphorylates both the Lhcb1 and Lhcb2 proteins of LHCII trimers that are loosely bound to PSII supercomplexes. Phosphorylated LHCII trimers containing Lhcb2 migrate from the PSII enriched grana stacks and then associate with PSI in the stroma lamellae. Not all migrating LHCII bind to PSI, but instead may form into energy dissipative aggregates. Phosphorylation of LHCII also loosens the stacking between grana stacks, likely due to charge repulsion. Release of PQ from the Q_o site inactivates STN7, allowing the (constitutively active?) PPH1/TAP38 phosphatase to dephosphorylate LHCII, thereby stimulating the return to the state 1 antenna configuration. B. In cyanobacteria, accumulation of PQH_2 over that of PQ also simulates the transition to state 2, although the signaling mechanism is unknown. PBSs (shown in light gray) associate with the cytosolic face of the thylakoid. In state 1, PSII particles are organized into lattice-like rows and PSI is trimeric. Upon the transition to state 2, a myriad of new associations may form between PSII, PSI, and PBS. If state 2 is induced by dark adaptation then PSI trimers can disassociate into monomers. State 2 may also be accompanied by a randomization of the PSII particles. In the "energy spillover" method of increasing PSI absorbance cross section, excess excitation energy from PSII core chlorophyll *a* can transfer to the PSI chlorophyll *a*, and this PSII-to-PSI interaction maybe favored by PSI monomerization. In the "mobile PBS" method of increasing PSI absorbance cross section, PBS disassociate from PSII and then associate with PSI. The PBS-to-PSI connection can be from the PBS core or from the PBS rod. If PBS remain associated with PSII, any enhanced proximity of PSII to PSI occurring during state 2 would facilitate energy transfer from PBS rod ends to PSI. The arrangement illustrated in the upper thylakoid represents "energy spillover" supporting interactions; the arrangement illustrated in the lower thylakoid represents "mobile PBS" supporting interactions. Since long-range diffusion of photosystems in the thylakoid is unlikely in vivo, the photosystem re-arrangements shown *here* exaggerate more subtle changes in the interaction of the photosystem proteins that would occur over a small-range scale in vivo. However, PBS are likely capable of long-range movement across the cytosolic surface of the thylakoid and thus are truly "mobile" (dashed lines represent aqueous interfaces of the thylakoid membrane). See text for details.

conversion of the megacomplex to the supercomplex via the phosphorylation of LHCII, and the conversion of the supercomplex to the PSII core via the phosphorylation of the CP26 and CP29 minor LHCII proteins and PSII core proteins [214], reviewed in [45]. In isolated thylakoids, phospho-LHCII was shown to have a nearly two-fold greater diffusion coefficient over that of the unphosphorylated form [215]. Phospho-LHCII associates with PSI [216]. Analysis of isolated PSI supercomplexes from *C. reinhardtii* indicates that in state 2, PSI is able to bind 2 LHCII trimers containing all four types of LHCII and one LHCII monomer (most likely CP29) in addition to the 9 PSI LhcAs [217]. Similar to higher

plants, the state transition also modifies the chloroplast structure, affecting the periodicity of thylakoid stacking [218].

The LHCII serine/threonine kinase Stt7 is required for the phosphorylation of LHCII and the transition from state 1 to state 2 [193]. A comparative phospho-proteomic study of *C. reinhardtii* thylakoids from wild-type and *stt7* mutant cells found Stt7-dependent phosphorylation at the N-terminal stromal regions of many of the major LHCII proteins (Lhcbm1/Lhcbm10, Lhcbm4/Lhcbm6/Lhcbm8/Lhcbm9, Lhcbm3, Lhcbm5) and CP29 [188]. Stt7 was also involved in phosphorylation of the PSII core kinase Stl1, and Stt7 itself could be found phosphorylated [188]. Co-immunoprecipitation studies revealed that Stt7 is firmly associated with $Cytb_6f$ but also interacts with LHCII and PSI, but not PSII [219].

The Stt7 is a transmembrane protein [219]. How PQH_2 binding to the $Cytb_6f$ Q_o site activates the Stt7 catalytic domain is unknown [220], although the similar inhibition of the transition to state 2 in both higher plants and green algae by the Reiske protein targeting effects of stigmatellin and DBMIB (2,5-dibromo-3-methyl-6-isopropyl-1,4, benzoquinone) suggests similar mechanisms for the STN7/ Stt7 activation [195]. The state 2 to state 1 transition is less understood, but is predicted to proceed when oxidation of the PQ pool inactivates the Stt7 kinase, LHCII is dephosphorylated by the Pph1/TAP38 phosphatase, and the PSII core is dephosphorylated by the Pbcp/PBCP phosphatase [45]. It is unclear of the role of the Pbcp/PBCP phosphatase in the state 2 to state 1 transition, and if the phosphatases are regulated directly by the chloroplast redox state or if they are constitutively active [45].

The state transition has been imaged in *C. reinhardtii* cells using fluorescence lifetime imaging microscopy [221]. A 250 picosecond lifetime component appeared during the state 1 to state 2 transition; the use of the *Stt7* mutant and a mutant missing PSI and PSII, permitted assignment of the 250 picosecond component to phosphorylated LHCII that dissociates from PSII in state 2 [221]. Furthermore, some of the phosphorylated LHCIIs were energy dissipative aggregates [221], suggesting the existence of an uncoupled LHCII pool during the state transition [45]. Perhaps in transit from PSII to PSI, LHCII occurs as an energy dissipative aggregate, safely turning the uncoupled LHCII "off". Many of these transient LHCII may, in fact, remain quenched and never transfer excitation energy to PSI (see discussion below).

State transitions in green algae have traditionally been thought to be of much greater amplitude than those seen in higher plants, due to the larger changes in Chl fluorescence yield that have been observed in green algae. In *C. reinhardtii* up to 80% of LHCIIs were proposed to dissociate from PSII upon transition to state 2 [222]. PSII was found to have a 2–3 times smaller antenna size in state 2, attributed to the disassembly of PSII–LHCII supercomplexes and megacomplexes into PSII core only complexes [223]. However, recent picosecond fluorescence kinetic studies have found that only a small portion of dissociated LHCII in *C. reinhardtii* actually binds to PSI in state 2, with the remainder being in a (aggregated) quenched state not associated with either of the photosystems [218,221,224]. Based on absorbance measurements of intact cells, [218] reported a 70% loss in PSII antenna size, but only a 20% gain in PSI antenna size during the transition from state 1 to state 2, suggesting that a large subpopulation of the phospho-LHCIIs do not bind to PSI, but instead form energetically quenched complexes, either associated with PSII supercomplexes or in a free form.

In green algae, such as *C. reinhardtii*, the state 1 to state 2 transition was originally thought to act as a switch from photosynthetic linear electron transport to the cyclic electron transport pathway through PSI and the Cyt_b6_f complex [225,226]. But more recently, the state transition has been shown to be independent of cyclic electron transport efficiency, with the switch to cyclic electron transport proposed to involve a separate redox response [227]. Evidence from in vivo state transition studies [218,221,224] is pointing to new ideas about the physiological importance of the state transition in *C. reinhardtii* — that state 2 can indeed provide a photoprotective state for PSII in which LHCII is quenched. So it should now be understood that LHCII quenching has two feedback regulatory pathways for sensing photosynthetic redox poise: (i) phosphorylation regulated quenching triggered by PQH_2 binding to $Cytb_6f$ Qo and (ii) protonation regulated qE quenching (see previous sections on qE) triggered by a rise in ΔpH stemming from insufficient ATP–NADPH cycling during the carbon fixation reactions. Furthermore, since significant amounts of qE quenching are induced only after high light acclimation and are not considered constitutive in green algae (see Section 4.2), the relatively fast induced, photoprotective quenching of LHCII may be fulfilled by the state transition in green algae. The use of PsbS protonation to induce LHCII aggregation during qE seemingly provides higher plants with a faster response to the photosynthetic redox poise than the state transition long-range diffusion of LHCII within a heavily crowded thylakoid.

7.3. State transitions in cyanobacteria

State transitions in cyanobacteria occur on a faster time scale (seconds to few minutes) than those found in higher plants and green algae, probably due to the fact that the mobile antenna (PBS) is not an integral membrane protein, but instead can rapidly migrate along the comparatively much less crowded cytosolic surface of the thylakoid. As in higher plants and green algae, state transitions respond to changes in redox state of the PQ pool [228], although how the signal is transduced from the PQ pool is not yet understood [161], and some components of the state transition may not be redox controlled, but could be directly illumination dependent [229]. Dark conditions induce state 2 in cyanobacteria due to dark respiration reducing the PQ pool; weak far-red or blue light are effective at driving cells to state 1 because PSI has ~3× the chlorophyll *a* of PSII [161]. Contrary to the earlier view that state transitions were only physiologically important during acclimation to low light stress in cyanobacteria (as measured in *Synechocystis* 6803) [187,230] has shown that in *Spirulina platensis* state transitions are active at higher irradiance intensities giving the state transition a photoprotective role in energy dissipation.

The distribution of photosynthetic proteins within the cyanobacterial thylakoid is rather homogeneous (there are no grana), but also densely packed (see [160]. Fluorescence recovery after photobleaching (FRAP) measurements have revealed that whereas the PSII core is immobile under normal conditions [231], the PBS has a high rate of diffusion [231–233]. The close proximity of PSII and PSI cores [234] permits excitation energy transfer (i.e. spillover) from the Chl of the PSII core to the Chl of PSI when the PSII reaction center is closed [235]. The PBS core binds to PSII via interaction with the PBS core membrane linker polypeptide L_{cm}, which facilitates highly efficient excitation energy transfer from the APC terminal emitters to the PSII core Chl [15,236]. PBS can also transfer excitation energy to PSI both from the PBS core and also directly from the PBS rod ends [233,236–239]; however, energy transfer to PSI may be slower than to PSII [240]. Recently, a PBS–PSI supercomplex was isolated from *Anabaena* sp. PCC 7120 composed of a PSI trimer plus a unique type of a PBS that is rod-shaped, but lacking core phycobiliproteins [241]. This "core-less" PBS exhibited efficient energy transfer to PSI [241], but does not seem to be involved in state transitions [242]. Thus, there are two possible modes for regulating the distribution of excitation energy between PSI and PSII during state transitions in cyanobacteria: PBS-to-photosystem energy transfer (via transient PBS–photosystem associations, termed "mobile PBS") and PSII to PSI energy transfer (via energy spillover).

The mobile PBS versus energy spillover mechanism has been long discussed (see [161]). According to the mobile PBS model, the transition to state 2 occurs when PBS dissociates from PSII, migrates along the cytoplasmic face of the thylakoid, and then binds to PSI. In support of the role of PBS mobility in state transitions, the presence of high concentrations of phosphate, sucrose, glutaraldehyde, or betaine has been shown to lock-in the preexisting light state and inhibit PBS movement [229,231–233,243]. Mobile PBS finely explains the redistribution of phycobilin absorbed excitation energy during state transitions, yet

it is unable to explain the changes in Chl absorbed excitation energy, thereby the necessity for inclusion of the spillover mechanism [235,244–247].

Both mobile PBS and spillover mechanisms are now believed to contribute to state transitions [160,161,187,229,235,248], with a greater magnitude of energy redistribution likely from mobile PBS under natural light conditions, since spillover might only occur during dark adaption [229]. The model proposed by [235] suggests that state transitions are dually controlled by variation in energy transfer rates from PBS-to-PSI and in energy spillover from PSII-to-PSI. This mechanism for excitation energy redistribution would only need subtle changes in PBS and photosystem arrangement and would not require long-scale PBS or photosystem movement. Yet, large modifications in photosystem macro-organization within the thylakoid have been observed between state 1 and state 2 conditions. In state 1, PSII particles are found organized into rows as opposed to the more random configuration of state 2 [234,249,250]. When thylakoids are transformed into a (semi)solid crystalline state using low temperature and lipid mutations, the state transition is inhibited [251]. The spillover of excitation energy from PSII to PSI would decrease when PSII is organized into rows in state 1 due to loss of PSII–PSI physical connections [234]. The oligomeric state of PSI (trimeric vs. monomeric) has been implicated in regulating energy spillover to PSI [229,252,253] and in PBS mobility [254], whereby PSI trimers disassociate into monomers during dark adaptation to state 1 [229]. Li et al. [229] have suggested that energy spillover from PSII to PSI only occurs under dark induced state 2 conditions, and that PSI trimer breakup is light on/off controlled and mediated by a dark respiration driven rise in cytosolic pH. The state transition models for *S. platensis* presented by [233,239,248] have stressed the importance of the changeover from PBS core-to-PSII energy transfer in state 1, to PBS rod-to-PSI energy transfer in state 2. Fig. 3B illustrates the possible antenna configurations that may form during state 1 and state 2 in cyanobacteria.

Thermal buildup within the antenna system may cause a loss in PSII antenna size via the detachment/disassembly of PBS in cyanobacteria. Stoitchkova et al. [255] showed that high light and high temperature treatments in *Synechocystis* PCC 6803 resulted in a similar detachment of PBS from the thylakoid and disassembly of PBS. A follow-up study [256] revealed that the effect was inducible with high light absorbed by either phycobilins or Chl *a*. Pigment over-excitation, including back energy transfer from PSII to PBS APC, was suggested to increase the localized buildup of heat from the non-radiative dissipation of excitation energy and "melt" thermo-labile elements of the PBS (likely L_{cm} which is the linker polypeptide critical for PBS detachment and core disassembly, and also rod linker polypeptides for further PBS disassembly) [255,256]. The effect on PBS was shown to be photon dose dependent; however, PBS detachment/disassembly in the minutes timescale was only obtained with un-natural high irradiances — an hour time scale (3 h) was required to see a comparable effect with more moderate (600 μE) high light intensities [255,256]. In such, thermally induced PBS disassembly would not qualify as a fast, reversible form of NPQ, but more of a long-term acclimation in cyanobacteria to excitation intensities that cannot be regulated effectively by the OCP and qT NPQ mechanisms.

The $Cytb_6f$ complex appears to be involved in triggering the state transition in cyanobacteria, based on the effect of $Cytb_6f$ inhibitors on state transitions [257,258], but the interaction with the Rieske protein is likely different than that during the activation/deactivation of the Stt7/STN7 kinase in green algae/higher plants [161]. Current consensus is that cyanobacterial state transitions do not involve a kinase, though it was proposed in the past [259]. State 1 to state 2 equilibrium of mobile PBS would be conceptually maintained by the binding affinities of PBS to PSII and PSI [187], with the redox signal somehow modulating those binding affinities. *Synechocystis* PCC 6803 cells with deletion of the *rpaC* gene, coding for a small transmembrane protein, were unable to redistribute PBS excitation energy (cells were locked in state 1), yet the cells could still perform spillover of Chl absorbed excitation energy [187,260]. Is RpaC the PQ redox signal carrier between $Cytb_6f$ and PBS? Does "activated" RpaC weaken the PBS–PSII association promoting the release of PBS? Still to be fully addressed is the extent to which the PBS–OCP mechanism interacts with the PBS-qT mechanism. Does OCP interaction with L_{cm} during intense blue irradiation enhance/ perturb the transition to state 2, are PBS–OCP mobile, and can OCP-quenched PBS still transfer excitation energy to PSI as via PBS rod-to-PSI energy transfer? As one can see, many aspects of the cyanobacterial state transition remain to be elucidated, especially the signaling pathway from the PQ pool to the PBS–photosystem interface.

8. Final thoughts and perspectives

The antenna-based approach for the fast regulation of PSII excitation has clearly been important for the survival fitness of photosynthesizers since the advent of an oxygen evolving PSII. Some form of light harvesting NPQ has been observed in "all" PSII containing photosynthetic lineages thus so far investigated [6,48]. NPQ pathways have become integrated into the PSII antenna system (e.g. LHC aggregation), so that quenching can rapidly "self-activate" in response to a rise in PSII excitation pressure. Of additional interest is tracking the evolution of NPQ among the divergent photosynthetic groups, particularly within the red algal lineage, and the algal groups which derived their chloroplast from the secondary endosymbiosis of a red algal ancestor. Red algae maintain PBS but have lost OCP. The NPQ mechanisms of red algae remain somewhat of an enigma — depending on the species and experimental conditions, both cyanobacterial-like state transitions and qE have been observed. There can be a mobile PBS type of state transition [261], PSII excitation energy spillover to PSI [262–264], and ΔpH-dependent quenching [265]. Diatoms perform a robust qE centered in membrane integral LHCs, but based on their red algal ancestry, how did the diatom qE mechanism evolve? Also of curiosity is the evolution of the xanthophyll cycle from, probably, a biosynthetic pathway for producing light-harvesting carotenoids, towards a mechanism for regulating the sensitivity of LHC to protonation (see [266]).

The role of NPQ capacity in niche partitioning seems also to be important, as epitomized in the diatoms, where energy dissipation from NPQ may be very weak, as in the stable light environment of diatom species from the open ocean, or several fold higher in magnitude (as measured via the Stern–Volmer *NPQ* quenching parameter) in diatom species from unstable environments such as shorelines and estuaries [113]. The importance of NPQ in regulating photosynthesis within the natural environment will surely become more heavily investigated, as portable and more sophisticated Chl fluorometers are used to probe the variable fluorescence in all green things, from the forest canopy to the ocean waters. The physiological phenomenon witnessed as a non-photochemical quenching of the PSII fluorescence signal is in reality a mixture of several different NPQ components that are activated and deactivated over differing time scales. Enhanced kinetic and spectral analysis of the fluorescence signal, combined with the fitting of fluorescence traces to multicomponent quenching models (as in *Chlamydomonas* by [267]), will perhaps permit the ready investigation of photosynthetic energy partitioning within field conditions. How much of the solar energy that strikes the earth's surface is, in actuality, converted to heat by NPQ mechanisms, and how does this vary on cellular, plant, ecosystem, global and seasonal scales (preliminarily addressed in [70])? The remote sensing of Chl fluorescence from space (e.g. [268]) could permit the measurement of NPQ and its influence on energy and carbon fluxes on a truly global scale. And, due to the profound influence of quenching mechanisms on the efficiency of photosynthesis, the question arises as to how manipulation or engineering of NPQ mechanisms could be used to advantage in agriculture [269].

Transparency Document

The Transparency document associated with this article can be found, in the online version.

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