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REVIEW

Photosynthetic Control and Its Role in Protection of Photosystem I against Photoinhibition

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Abstract—This review addresses photosynthetic control as a protective mechanism that prevents photoinhibition of photosystem I under conditions of imbalance between CO_2 assimilation during the Calvin–Benson–Bassham cycle and light reactions in the thylakoid photosynthetic apparatus. We discuss the pathways of photosystem I photoinhibition and describe protective mechanisms that prevent photodamage of photosystem I. We propose a hypothesis regarding the influence of photosynthetic control on formation of reactive oxygen species in photosystem I. pH-sensitivity of plastoquinol oxidation at the quinol-oxidizing (Qo) site of the cytochrome b_6f complex is analyzed, and function of two proton-conducting channels that release protons into the thylakoid lumen from the cytochrome b_6f complex is described. We examine impact of photosynthetic control on the functioning of the cytochrome b_6f complex itself, and propose a hypothesis regarding the preferential activation of photosynthetic control in the thylakoid grana, which ensures operation of the cyclic electron transport around photosystem I as a main protective mechanism.

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Keywords: photosynthesis, photosynthetic electron transport chain, photosynthetic control, cytochrome b_6f complex, photosystem I, photoinhibition, PGR5, cyclic electron transport, reactive oxygen species

INTRODUCTION

Variation in the intensity of environmental factors could disrupt the balance in plants between light harvesting, charge separation within the thylakoid photosynthetic machinery, and CO_2 assimilation in the Calvin–Benson–Bassham cycle (CBB cycle), leading to decline in photosynthetic activity – the so-called photoinhibition (PI). The primary targets of PI are photosynthetic reaction centers, photosystem II (PSII), and photosystem I (PSI). Photoinhibition of PSII (PI(II)) has been thoroughly characterized, while photoinhibition of PSI (PI(I)) remained undetected in whole plants until 1994, and PI(I) was observed only in *in vitro* experiments using isolated structures. PI(I) in plants was first observed in the cold-sensitive cucumber at low temperatures [1] and was later reported for several other species (reviewed in [2]). A breakthrough came from the experiments using artificial fluctuating light (FL), which imitated changing natural light environment [3]. Later, other protocols for triggering PI(I) were developed, including repetitive short pulses of saturating light (rSP) [4].

It is now evident that PI(I) poses a greater threat to plant viability than PI(II), because repair of the damaged PSI complexes requires a day or more, depending on the damage severity [5], whereas PSII is repaired within hours. A consensus has emerged that PSI in plants is better protected than PSII [6]. The defense mechanisms differ among the groups of photosynthetic organisms [7]. In angiosperms, a key protective pathway is cyclic electron transport around PSI (CET(I)), which in C_3 plants operates mainly via the Proton Gradient Regulation 5 (PGR5)-dependent route. Accordingly, the *Arabidopsis thaliana* mutant lacking PGR5 protein cannot survive under FL conditions [3] due to severe PI(I) [8, 9]. Several authors [2, 7] have proposed existence of a universal mechanism

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protecting PSI in various groups of photosynthetic organisms, namely photosynthetic control (PhotCon).

PhotCon involves slowing down the photosynthetic electron transport at the step of plastoquinol (PQH_2) oxidation within the cytochrome b_6f complex (Cyt-b_6f), triggered by acidification of the thylakoid lumen. Imp`aired activation of PhotCon is often considered as the primary cause of the elevated PI(I) observed in the *pgr5* mutants [8], although this remains a matter of debate [10]. The PI(I) phenomenon has been reviewed in several recent publications, as well as effects of the pgr5 mutation, and PhotCon itself [2, 6, 11, 12]. Nevertheless, the precise mechanisms by which lumen pH modulates the rate of PQH_2 oxidation in Cyt-b_6f and the way PhotCon prevents PI(I) are still poorly understood. Moreover, recent reviews pay little attention to accumulating experimental evidence that PhotCon indeed protects PSI, and they seldom critically assess the strength of that evidence. It also remains unclear whether PhotCon fulfils functions beyond the PSI protection; for instance, review [12] considers defense against PI(I) as the sole function of PhotCon. These issues are addressed in the present review.

STRUCTURE AND FUNCTIONING OF THE PHOTOSYNTHETIC ELECTRON TRANSPORT CHAIN OF HIGHER PLANTS

Light harvesting by photosynthetic apparatus initiates electron flow through the photosynthetic electron transport chain (PETC). In PSII, electrons from water are used for reduction of lipid-soluble prenylquinone, plastoquinone (PQ), to PQH_2, which diffuses within the lipid bilayer to Cyt-b_6f, where it is oxidized, and electrons are used for reduction of the lumenal copper protein plastocyanin (Pc). Pc then donates electrons to PSI, which reduces the stromal carrier ferredoxin (Fd). Fd feeds electrons into various metabolic pathways of the chloroplast [13], primarily reduction of $NADP^+$ to NADPH catalyzed by ferredoxin:$NADP^+$ oxidoreductase (FNR). Electron flow through the PETC is coupled with generation of the proton-motive force (pmf) across the thylakoid membrane, which consists mainly of the trans-thylakoid pH gradient (ΔpH) [14]. The pmf drives rotation of the thylakoid ATP-synthase, producing ATP from ADP and Pi. The CBB cycle uses ATP and NADPH in a ratio of 1.5. Given the number of H^+-transporting c-subunits of the chloroplast ATP-synthase, it was calculated that linear electron flow yields an ATP/NADPH ratio of only ~1.29 [15]. Therefore, additional ATP synthesis is required for optimal functioning of CBB cycle, which is produced in chloroplasts via alternative electron transport pathways that contribute to ΔpH formation without producing NADPH.

In angiosperms, CET(I) operates via two routes: an antimycin-A (AA)-sensitive, PGR5-dependent pathway that is predominant in C_3 plants, and an AA-insensitive pathway mediated by the NADH-dehydrogenase-like (NDH) complex that is predominant in C_4 species [16, 17]. In both routes, Fd reduces the PQ pool, which is then re-oxidized by Cyt-b_6f, returning electrons to PSI. NDH is homologous to the respiratory Complex I but accepts electrons from Fd rather than NAD(P)H. The Cyt-b_6f itself is likely an enzyme that oxidizes Fd and reduces PQ in the AA-sensitive CET(I) pathway [18] (see reviews [19, 20] for details).

Molecular O_2 serves as an alternative electron sink for PETC. During photorespiration, for example, RUBISCO catalyzes an oxygenase reaction with O_2 instead of carboxylase reaction with CO_2; since regeneration of the formed 3-phosphoglycerate requires ATP and reducing equivalents generated by linear electron flow, photorespiration is considered as an O_2-dependent alternative electron pathway [7]. Additional routes ultimately reduce O_2 to H_2O within the chloroplast; collectively, these are termed water–water cycles. One such cycle begins with direct reduction of O_2 by PETC components, yielding superoxide anion radical ($O_2^{\bullet-}$) as the primary product, and hydrogen peroxide (H_2O_2) as the stable product. H_2O_2 is then detoxified to H_2O with involvement of ascorbate and ascorbate peroxidase, and ascorbate is regenerated by electrons supplied by the PETC [21]. A shorter water-water cycle is driven by the plastid terminal oxidase, which reduces O_2 to water while oxidizing PQH_2. A four-electron reduction of O_2 to H_2O by NADPH molecules catalyzed by flavodiiron proteins occurs in all photosynthetic organisms except angiosperms [22].

Substantial evidence indicates that PSII, Cyt-b_6f, and PSI are distributed heterogeneously within the thylakoid membrane. Cryo-electron microscopy of intact spinach chloroplasts, published in 2025 [23], shows that the grana stacks contain only PSII and Cyt-b_6f, whereas stromal lamellae contain PSI, Cyt-b_6f, and ATP-synthase. The grana-localized PSII and Cyt-b_6f perform linear electron transport to PSI complexes in stromal thylakoids, with Pc shuttling electrons from grana to stromal regions [24]. Stromal thylakoid Cyt-b_6f and PSI perform CET(I).

PHOTOINHIBITION OF PHOTOSYSTEM I

PSI is a multisubunit pigment–protein complex embedded in the thylakoid membrane. Its electron-transfer cofactors are located in three protein subunits – PsaA, PsaB, and PsaC. Cofactors, from the primary donor P_{700} (a chlorophyll-*a* special pair) to the Fe_4-S_4 cluster F_X, are arranged in two pseudo-symmetrical branches (A- and B-) within the PsaA/PsaB

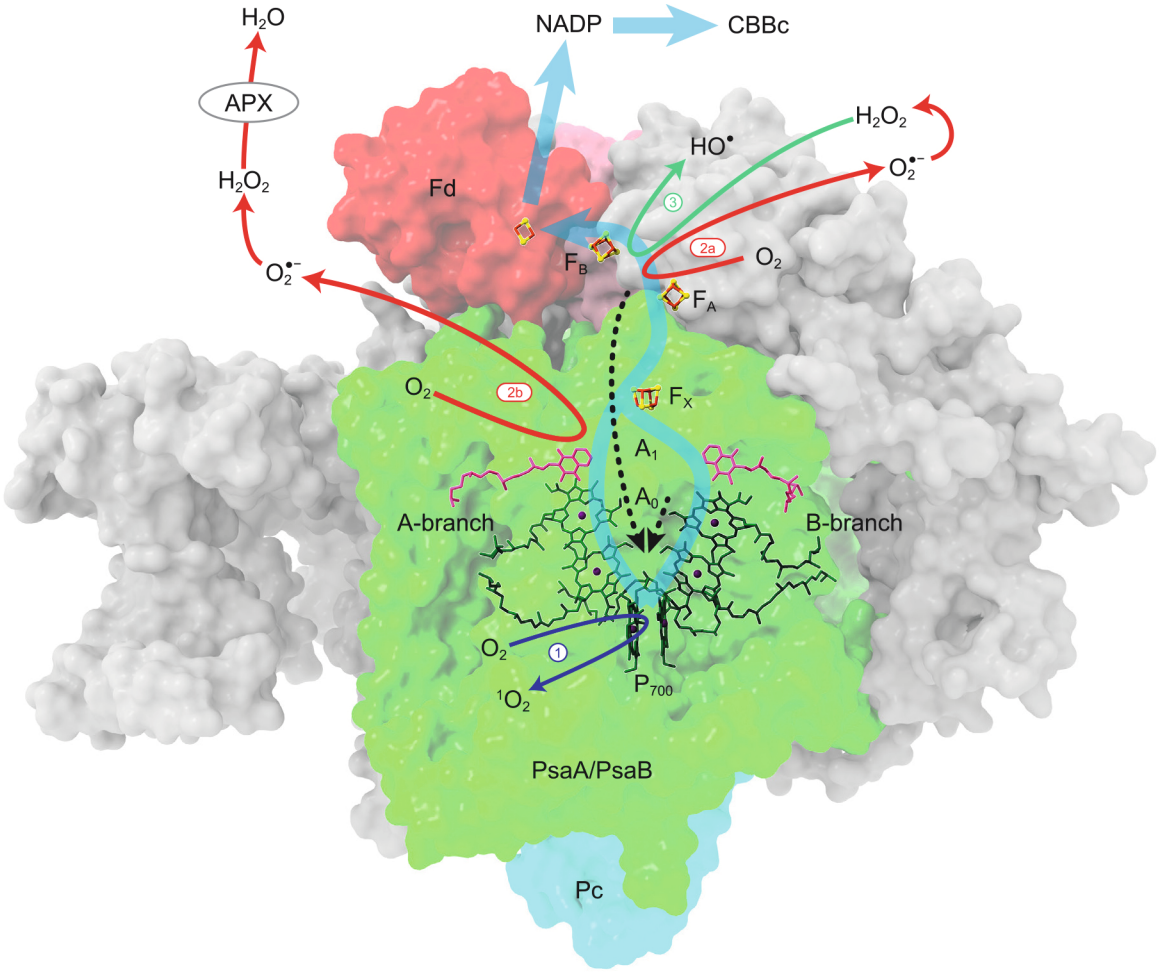

Fig. 1. Organization of the PSI electron-transfer chain and ROS-generating pathways involving PSI cofactors: 1) formation of $^1O_2$ via the reaction of O_2 with P_{700} triplet ($^3P_{700}$) produced during charge recombination with intermediate PSI cofactors. 2) Formation of $O_2^{\bullet-}$ via reaction of O_2 with (a) the F_A/F_B clusters and (b) the phylloquinone in the A_1-sites. 3) Formation of $HO^\bullet$ via reaction of H_2O_2 with F_A/F_B clusters. Thick blue arrows indicate forward electron transfer within PSI and onward to Fd, $NADP^+$, and the CBB cycle; dashed arrows denote charge recombination. All other arrows depict ROS-generation routes involving PSI cofactors. The scheme is based on the structure of PSI in complex with Pc and Fd (PDB: 6YEZ) [31].

heterodimer; electrons are transferred from F_X to two terminal Fe_4-S_4 clusters, F_A and F_B, on the PsaC subunit (Fig. 1). In addition to P_{700}, four more chlorophyll-*a* molecules participate in electron transfer: the two most distant from P_{700} serve as the A_0 cofactors on the A and B branches. Between each A_0 and cluster F_X a phylloquinone molecule is located in the A- and B-branches (the A_1 cofactor). The oxidized P_{700} (P_{700}^+) is reduced by Pc, while the F_A/F_B clusters donate electrons to Fd.

PSI can also produce reactive oxygen species (ROS). $O_2^{\bullet-}$ is produced via reduction of molecular O_2 by the terminal clusters F_A/F_B (whose contribution saturates at moderate light intensities) and the phylloquinones in the A_1-sites, mainly in the A branch (whose contribution increases with increasing light intensity) [25] (Fig. 1). Generation of $O_2^{\bullet-}$ by PSI cofactors occurs in parallel with Fd reduction [25]. Under $NADP^+$ limitation, the reduced Fd itself produces $O_2^{\bullet-}$, but sufficient $NADP^+$ minimizes electron leakage from Fd to O_2 [26]. In the stroma, $O_2^{\bullet-}$ undergoes dismutation to H_2O_2, which is scavenged by the chloroplast antioxidant system. When H_2O_2 production outpaces its detoxification, H_2O_2 accumulates. The light-reduced F_A/F_B clusters can then catalyze conversion of the excess H_2O_2 into hydroxyl radicals ($HO^\bullet$) [27]. Several lines of indirect evidence also suggest that PSI can generate singlet oxygen ($^1O_2$) [28, 29]. Production of $^1O_2$ is feasible via the P_{700} triplet ($^3P_{700}$) formed during charge recombination between P_{700}^+ and A_0^- but not between P_{700}^+ and the terminal F_A/F_B clusters [30].

In the pioneering study that first demonstrated PI(I) in a cucumber plant under chilling stress, removal of O_2 markedly preserved PSI activity, implicating ROS formation by PSI as the underlying cause of PI(I) emergence [1]. Currently, the data have been

accumulated indicating that the ROS-dependent PI(I) proceeds via two distinct mechanisms, depending on the triggering stress, which leads to the primary damage on either acceptor or donor sides of the PSI complex.

Under chilling stress, the initial damage occurs at the Fe_4-S_4 clusters of PSI [32, 33], whereas destruction of P_{700} itself requires more severe treatments [32]. *In vitro* experiments with the illuminated spinach thylakoids revealed that $HO^{\bullet}$ scavengers provide protection against PI(I) [34] and that exogenous H_2O_2 accelerates PI(I) [35]. Importantly, photoinhibition was strongly suppressed by adding methyl viologen (MV), an efficient acceptor of electrons from PSI [34, 35]. Because MV itself enhances H_2O_2 production during illumination, these findings indicate that the critical factor for PSI photodamage is not the amount of H_2O_2 accumulated *per se*, but rather efficiency of the electron outflow from PSI. Consistent with this view, MV lowered $HO^{\bullet}$ generation in thylakoids [27]. It was concluded based on the data in [34, 35] that in the case of inefficient electron outflow from PSI the reduced cofactors reduce O_2 producing H_2O_2, and next, the reduced terminal clusters F_A/F_B catalyze $HO^{\bullet}$ formation from that H_2O_2, acting as a Fenton-type catalyst [36].

Loss of EPR signal from the F_A/F_B clusters has been reported both in the Arabidopsis *pgr5* mutant and in the wild-type (WT) plants exposed to high light [37, 38], suggesting that the similar PI(I) mechanism related to the impaired electron outflow from PSI is realized in the plants under FL and high light.

By contrast, the cucumber leaves subjected to rSP (a long series of 300 ms saturating flashes separated by 10 s dark intervals) show no loss of the EPR signal of the F_A/F_B clusters [33]. Thus, the rSP treatment does not promote $HO^{\bullet}$ production on the acceptor side of PSI, even though each flash fully reduces the PSI cofactors and Fd [39], a condition that would favor over-reduction. After the rSP treatment, however, the PSI complexes show a discrepancy between the kinetics of charge separation and the EPR signal of F_X [33], interpreted as structural damage between P_{700} and A_1 in the A-branch, caused by the ROS generated in close vicinity of P_{700} and A_1-sites. Detailed study of the rSP-induced PI(I) in the spinach chloroplasts [28] assumed that both $O_2^{\bullet-}$ produced at the A_1-sites and $^1O_2$ contribute to the damage. The proof of $^1O_2$ involvement is based on the protective effect of an amphiphilic vitamin E analogue, a specific $^1O_2$ quencher. The similar effect of $^1O_2$ scavengers had been observed previously in the PSI-enriched thylakoid membranes devoid of PSII [40]; these preparations cannot form H_2O_2 because electron donation to PSI is absent, yet $^1O_2$ can be formed through charge recombination, which is the only pathway of P_{700}^{+} re-reduction under those conditions. Involvement of $O_2^{\bullet-}$ from the A_1-sites, however, is less firmly supported by the existing evidence. MV provided significant protection during the rSP treatment [28], and MV indeed suppressed $O_2^{\bullet-}$ generation at the A_1-sites [25]. However, MV also diminishes charge recombination in PSI [41], so its protective action may stem from the reduced $^1O_2$ formation rather than from suppression of $O_2^{\bullet-}$ formation at the A_1-sites. Nonetheless, lack of direct evidence does not rule out a contributory role for the $O_2^{\bullet-}$ generated at the A_1-sites in PI(I) caused by rSP treatment.

A series of studies employing the rSP protocol led to formulation of the "P_{700} oxidation" concept as the principal mechanism protecting PSI against photoinhibition [7]. This term describes presence of the detectable P_{700}^{+} signal under actinic light, which is sustained both by electron outflow from PSI to Fd, which is manifested by low values of the quantum yield of non-photochemical losses on the acceptor side of PSI, Y(NA), and by the regulatory decrease in electron inflow to PSI, which is manifested by the high values of quantum yield of non-photochemical losses on the donor side of PSI, Y(ND). Accordingly, the PSI-protective pathways fall into three categories: 1) mechanisms regulating electron outflow from PSI, 2) mechanisms regulating electron inflow to PSI, 3) CET(I), which should be considered separately (see below).

The first group includes the CBB cycle and alternative oxygen-dependent pathways, which promote oxidation of Fd and NADPH pools and, subsequently, of P_{700} [42, 43]. The CBB cycle is the primary sink of electrons from PSI; therefore, ensuring its optimal operation is the key PSI-protection strategy. For instance, increasing CO_2 concentration from 400 ppm to 800 ppm (the CBB cycle-optimum for C_3 plants) markedly reduced PI(I) in the Arabidopsis leaves under FL [44]. When the CBB cycle activity is restricted, alternative pathways provide auxiliary Fd oxidation, mitigating PI(I). Notably, these alternative pathways also contribute to ΔpH formation, supplying extra ATP for the CBB cycle and other metabolic processes in chloroplasts, as well as activating defense mechanisms regulating electron inflow to PSI.

The second group includes (i) PhotCon; (ii) downregulation of PSII activity [45]; (iii) diversion of electrons to an alternative acceptor upstream of PSI, most notably via oxidation of the PQ pool by O_2 mediated by the plastid terminal oxidase [46] and, possibly, spontaneous PQ reactions with O_2 and ROS [47, 48]; (iv) dynamic modulation of grana diameter, which affects the rate of Pc diffusion from the grana-embedded Cyt-b_6f to PSI complexes in the stromal lamellae [24].

A substantial body of evidence indicates importance of CET(I) in protecting PSI. The Arabidopsis double mutants lacking both the NDH complex and the PGR5 protein are virtually unviable [16].

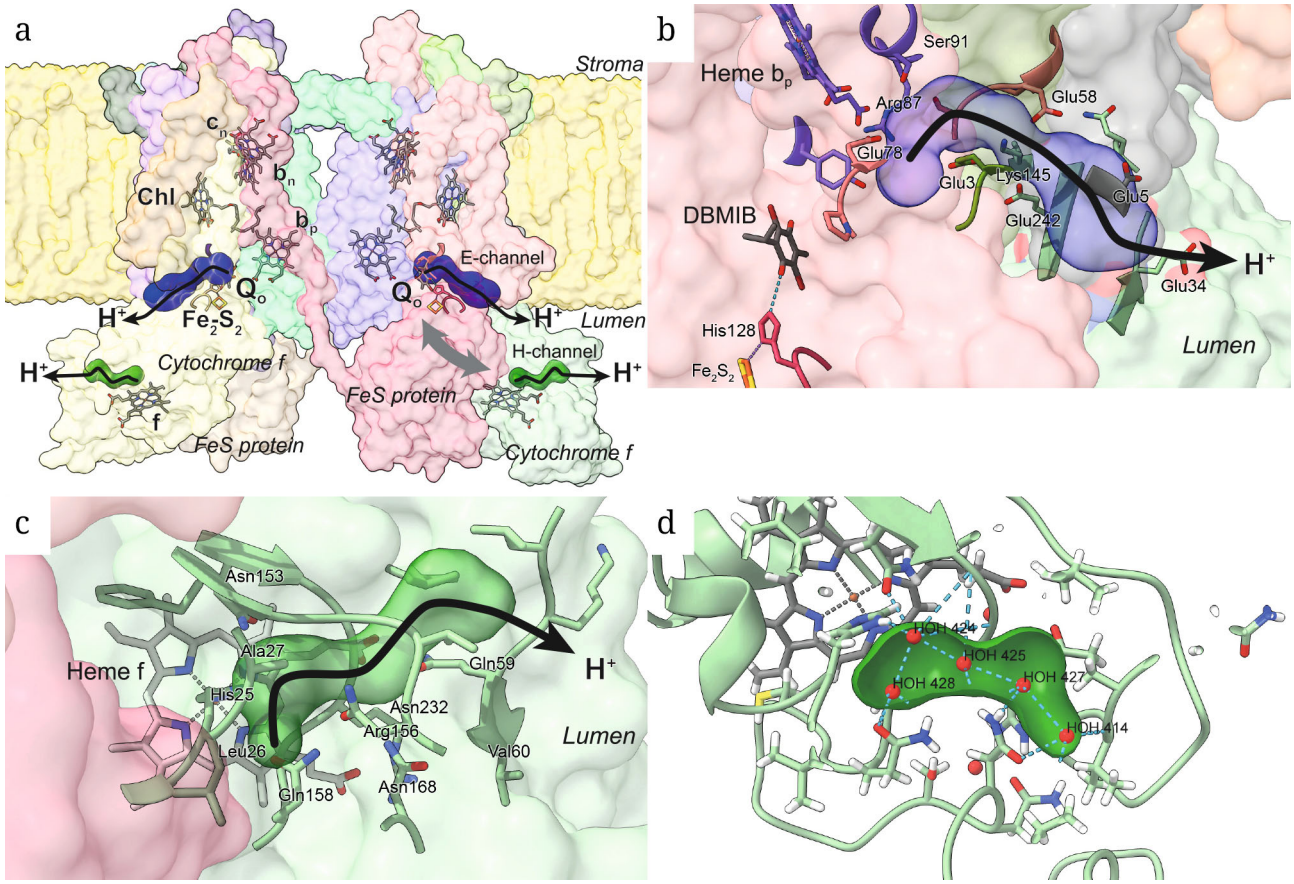

Fig. 2. Visualization of proton-releasing channels of the Qo site of Cyt-b_6f. Overall arrangement of channels in spinach Cyt-b_6f (PDB 9ES9) [56] (a). E-channel in the spinach Cyt-b_6f (PDB 9ES9) rendered according [55] (b). H-channel in the spinach Cyt-b_6f (PDB 9ES9) rendered according [57-59] (c). H-channel with bound H_2O molecules in the Cyt-b_6f from *Nostoc sp.* PCC 7120 (PDB 4H44) [60] (d). Channels were visualized with MOLE 2.5 (https://moleonline.cz) and ChimeraX 1.9 (https://www.cgl.ucsf.edu/chimerax/).

The mutants that lack only the NDH complex show enhanced PI(I) under FL [49]. Infiltrating the Arabidopsis leaves with antimycin A (AA) likewise causes a marked loss of PSI activity under FL [50]. In the mutants with impaired binding of thylakoid FNR, which likely acts as a regulator switch between the linear electron transport and AA-sensitive CET(I) [51], the increased PSI photoinhibition was observed upon transfer of plants to high light [52]. Numerous studies further demonstrate that the *pgr5* mutations impair P_{700} oxidation, making PSI more vulnerable to photoinhibition under both FL and high light [8, 37, 50].

Because CET(I) recycles electrons, its steady-state operation inherently produces equal electron outflow from and inflow to PSI; therefore, by itself it cannot keep P_{700} oxidized. Therefore, the protective role of CET(I) is attributed to its proton-pumping activity, which (i) generates additional ATP without concomitant NADPH production, thereby optimizing CBB cycle turnover and indirectly relieving the acceptor-side limitation of PSI, and/or (ii) acidifies the thylakoid lumen, thereby triggering PhotCon and indirectly activating defense mechanisms regulating electron inflow to PSI.

Thus, unraveling the complete picture of molecular mechanisms protecting PSI is complicated by the challenge of separating effects on the acceptor and donor sides of PSI. It is possible that both factors are at play simultaneously. Taking into consideration existence of mechanisms of PI(I) development – one causing primary damage on the acceptor side, and the other on the donor side – it seems plausible that regulation of electron outflow from PSI is crucial for preventing the acceptor-side PI(I), whereas regulation of electron inflow is crucial for preventing the donor-side PI(I). Nevertheless, several experimental lines of evidence (see below) indicate that PhotCon could also protect PSI under conditions that induce the acceptor-side PI(I).

PHOTOSYNTHETIC CONTROL

Oxidation of PQH_2 at the Qo site of Cyt-b_6f and proton release into the lumen. The cytochrome-b_6f complex (Cyt-b_6f) is a functional dimer (Fig. 2a). Each monomer comprises cytochrome (cyt) *f*, cyt b_6, Rieske

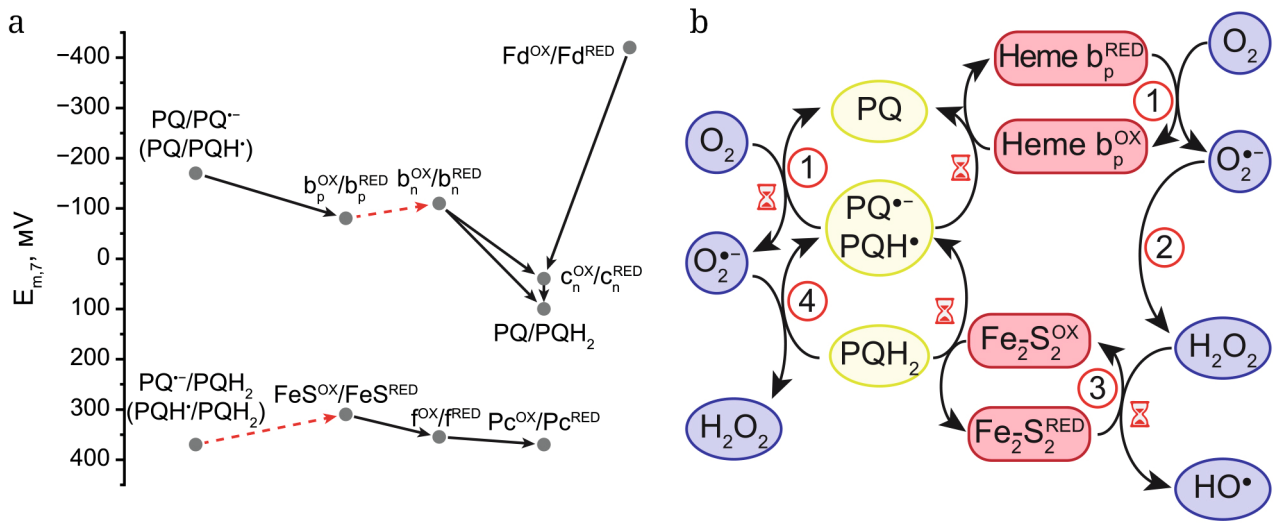

Fig. 3. Electron transfer in Cyt-b_6f. Redox-potential diagram ($E_{m,7}$) of the Cyt-b_6f electron transfer cofactors (a). Red dashed arrow marks endergonic steps of the direct electron transfer. Quinone potentials (−170 mV for PQ/PQ$^{\bullet-}$ and +370 mV for PQ$^{\bullet-}$/PQH$_2$) are for aqueous solution because site-specific values are unknown. $E_{m,7}$ values for the low-potential branch are from [64]; for the high-potential branch – from [65]. Scheme of ROS formation in the Qo site (b). For simplicity, some reactants and products are omitted. 1) Oxidation of PQ$^{\bullet-}$ (PQH$^{\bullet}$) and heme b_p by O_2. 2) Dismutation of $O_2^{\bullet-}$ to H_2O_2. 3) Fe_2-S_2-catalyzed decomposition of H_2O_2 to HO$^{\bullet}$. 4) Reduction of $O_2^{\bullet-}$ to H_2O_2 by PQH$_2$ bound in the Qo site. The hourglass symbol denotes a hypothesized slowdown of the reaction when PhotCon is activated.

iron–sulfur protein (ISP), subunit IV (sub IV), and small subunits PetG, PetL, PetM, and PetN. Its prosthetic groups include: Fe_2-S_2 cluster in the ISP, two *b*-type hemes in cyt b_6 (hemes b_p and b_n, oriented towards the positively charged lumenal p-side and negatively charged stromal n-side, respectively), c_n-type heme in cyt b_6, *c*-type heme in cyt *f*, one chlorophyll *a*, and β-carotene in sub IV (for detailed architecture see [19, 20, 53]). PQH$_2$ and PQ diffuse into their respective binding sites through the inter-monomer cavity [54]: the quinol-oxidizing Qo site on the luminal side and the quinone-reducing Qr site on the stromal side. A phytyl "tail" of the chlorophyll *a* molecule occupies the Qo site in either open conformation, permitting PQH$_2$ access, or closed conformation, which restricts PQH$_2$ access [55].

Oxidation of PQH$_2$ at the Qo site proceeds via electron bifurcation – oxidation of the two-electron donor by the high-potential and low-potential single-electron acceptors (Fig. 3a). The first electron from PQH$_2$ is accepted by the Fe_2-S_2 cluster of ISP, after which the hydrophilic ISP domain rotates towards cyt *f*, changing its position from proximal to distal relative to the heme b_p, and the electron is then transferred to cytochrome f and further to Pc (high-potential branch of Cyt-b_6f). The second electron from PQH$_2$ is transferred through the hemes of cyt b_6 to the PQ molecule in the Qr site, thereby completing the Q-cycle (low-potential branch of Cyt-b_6f).

Oxidation of PQH$_2$ is coupled to proton transfer [61]. Two residues are critical for binding, stabilizing, and deprotonating PQH$_2$ and its semiquinone form PQH$^{\bullet}$: H128 of the ISP [62], and E78 of sub IV [60, 63] (spinach nomenclature, PDB 6RQF). Initially, PQH$_2$ forms a hydrogen bond between its carbonyl oxygen and N_ε atom of the H128; the first proton is transferred to H128 (reaction 1a), and only then the Fe_2-S_2 cluster oxidizes PQH$^-$ to the neutral semiquinone PQH$^{\bullet}$ (reaction 1b).

$$PQH_2 + H128\sim ISP^{ox} \rightarrow PQH^- + \\ + H\text{-}H128\sim ISP^{ox} \quad (1a)$$

$$PQH^- + H\text{-}H128\sim ISP^{ox} \rightarrow PQH^{\bullet} + \\ + H\text{-}H128\sim ISP^{red} \quad (1b)$$

In the second step of the reaction, according to quantum-chemical modelling, PQH$^{\bullet}$ migrates within the Qo site toward the heme b_p [66]. PQH$^{\bullet}$ forms a hydrogen bond with the deprotonated carboxylate of E78 (reaction 2a) and is next oxidized by the heme b_p to PQ (reaction 2b).

$$PQH^{\bullet} + E78 + \text{heme } b_p{}^{ox} \rightarrow PQ^{\bullet-} + \\ + H\text{-}E78 + \text{heme } b_p{}^{ox} \quad (2a)$$

$$PQ^{\bullet-} + H\text{-}E78 + \text{heme } b_p{}^{ox} \rightarrow PQ + \\ + H\text{-}E78 + \text{heme } b_p{}^{red} \quad (2b)$$

After oxidation, the PQ molecule leaves the Qo site through the one-way diffusion channel [67] allowing the next PQH$_2$ to bind. For the detailed thermodynamic and kinetic aspects of PQH$_2$ oxidation, electron and proton transfer coupling, see reviews [19, 20, 53].

Because binding of PQH_2 and $PQH^{\bullet}$ requires H128 (ISP) and E78 (sub IV) to be deprotonated, the overall oxidation rate is governed by the efficiency of proton dissociation from these residues, as has been shown in studies using site-directed mutagenesis [58, 59, 63, 68]. Proton affinity of the H128 residue is redox-dependent: when the Fe_2-S_2 cluster is reduced, pK_a (H128) is ~8.3-8.9, but when the cluster is oxidized, it drops to ~6-6.5 [69]. Thus, once the cluster donates its electron, affinity of the H128 residue for H^+ decreases, and proton is released into the lumen. The proton from E78 is likewise released to the lumen, but current evidence suggests its transfer is not controlled by the redox state of Cyt-b_6f cofactors. The proton-releasing channels have been modelled in the Cyt-b_6f structures [55, 57, 60]. We denote them as H-channel and E-channel, responsible for releasing protons from H128 and E78, respectively (Fig. 2, b-d).

The H-channel was first identified in the cyt *f* from *Brassica rapa* (PDB 1HCZ) [57] as an intraprotein chain of five water molecules (Fig. 2, c, d) coordinated by highly conserved N and Q residues. The chain extends in two directions from H25 (the heme *c* ligand in cyt *f*): one water molecule is oriented toward the ISP, while four water molecules stretch ~11 Å toward K66, a residue involved in Pc binding on the luminal surface. It remains uncertain how the proton leaves H128: whether it is transferred directly to the first water molecule or to the H25 (~3.5 Å away). Several *Chlamydomonas reinhardtii* mutants carrying substitutions of the residues that coordinate water molecules in H-channel have been analyzed [58]. The mutation N168F removed last two water molecules in the chain, as a result the mutant lost the ability to grow photoautotrophically. All H-channel mutants exhibited slower cyt *f* reduction and slower generation of the slow phase of carotenoid electrochromic shift, a marker for vectoral electron transfer along the low-potential branch of Cyt-b_6f [58, 59]. Collectively, these data confirm that the disruption of H-channel compromises coordinated PQH_2 oxidation.

A hydrophilic region about 15.5 Å long and 4-6 Å in diameter, beginning at E78 and extending toward the lumen, was identified in the structure of Cyt-b_6f from *Mastigocladus laminosus* (PDB 4H13) [60]; this hydrophilic region is the primary candidate for E-channel. In that structure, the E78 side chain faces away from the Qo site toward the heme b_p; its carboxyl group forms hydrogen bond with a water molecule (at a distance of 3 Å) and with the side chain of R87 in the cyt b_6 (at a distance of 2.5 Å). The channel is lined with polar residues – R87 and S91 (both in cyt b_6), E3 (PetG), and D58 (subunit IV). Later, a similar channel was modelled in the spinach Cyt-b_6f structure (PDB 6RQF; Fig. 2b) [55]. Here, E78, likewise, points toward the heme b_p, and the channel is lined with R87 and S91 (cyt b_6), E3 (PetG), E58 (sub IV), E5 (PetM), and K145, E242, E34 (cyt *f*).

It must be emphasized that the exact proton-releasing trajectories from the Qo site to the lumen remains uncertain; the existing suggestions are based on hydrophilic intraprotein regions revealed by the structural studies and site-directed mutagenesis data. In the cytochrome-bc_1 complex (Cyt-bc_1), modelling predicts at least five distinct pathways for proton release from the Qo site, involving conserved cyt *b* residues and bound water molecules [70]. Whether the similar multitude of proton release pathways exist in Cyt-b_6f remains unknown. No counterpart of the H-channel has been found in cyt c_1 [71]. Thus, proton release from the Qo site of the Cyt-b_6f could be under tighter control than in the Cyt-bc_1, which could be an evolutionary adaptation to the lower luminal pH typical of illuminated chloroplasts.

pH-dependence of PQH_2 oxidation (photosynthetic control). Influence of pH on PQH_2 oxidation in the Qo site has been revealed from pH-dependence of the P_{700}^+ [72-74], cyt *f*, and cyt b_6 reduction [75]. Slowdown of PQH_2 oxidation with the luminal pH decrease is attributed to accumulation in the lumen of one reaction product, H^+. However, catalytic center of the Qo site is spatially isolated from the lumen, preventing direct contact between PQH_2 and H^+ accumulated in the lumen. This poses a question, how does the Qo site mechanistically "senses" acidification of the lumen? The prevailing view is that PhotCon arises from the backpressure exerted by the increasing content of H^+ in the lumen on the proton-accepting groups in the Qo site [76]. A simple model in which PQH_2 oxidation is slowed down due to the luminal accumulation of H^+, however, has been criticized [77]: since the E_0 difference between PQH_2 and Pc is approximately 300 mV (Fig. 3a), ΔpH values larger than those normally attained under typical physiological conditions would be required to slow down electron transfer from PQH_2 to Pc (see also [20]), although such extreme lumen acidification might occur during stress [78, 79]. Finazzi et al. proposed an alternative interpretation of PhotCon [77] based on existence of a pH-dependent regulation of Cyt-b_6f's transition between two kinetic states: location of the hydrophilic ISP domain near the Qo site or near cyt *f*. According to this hypothesis, acidification of the lumen would hinder deprotonation of H128, prolonging residence of the ISP domain next to cyt *f* and thus slowing PQH_2 oxidation in the Qo site. Whether the ISP can revert to the Qo proximal conformation while H128 remains protonated is unknown; given the likelihood of stochastic nature of ISP conformational shifts [80], this possibility cannot be excluded.

Threshold of the luminal pH for PhotCon activation (~6) corresponds to the pK_a of H128 when

the Fe_2-S_2 cluster is oxidized (~6-6.5). This allowed suggesting a hypothesis according to which PhotCon is triggered precisely by the prolonged lifetime of the protonated H128 state [53, 77]. By contrast, the predicted pK_a of E78 is much lower, around 3.9 [60]. At first glance, such acidic pK_a suggests that at luminal pH 5-6 E78 would not retain a proton long enough to slow down $PQH^{\bullet}$ oxidation.

Useful insights into the protonation state of H128 and E78 come from the studies with competitive inhibitors of PQH_2 oxidation at the Qo site. One of such inhibitors, 2,5-dibromo-3-methyl-6-isopropylbenzoquinone (DBMIB), initially binds to H128 in the reduced form ($DBMIBH_2$), which is followed by oxidation to semiquinone by the Rieske cluster resulting in a strong binding of the Fe_2-S_2 cluster in Qo site [56, 81, 82]. Therefore, the protonated H128 should interfere with binding of the reduced DBMIB. Indeed, inhibitory activity of DBMIB in the isolated pea thylakoids decreased under conditions facilitating lumen acidification [83]. Moreover, inhibitory activity of DBMIB was 3- to 10-fold lower in the Arabidopsis *pgr1* mutant [83], in which the P194L substitution in ISP raised pK_a (H128) by ~1 pH unit [84]. As a consequence, at pH 7.6 (the condition used in [83]) the fraction of Cyt-b_6f with protonated H128 was about 7.5-fold higher in the *pgr1* mutant than in the WT, which is in agreement with the fold difference in DBMIB binding between the genotypes.

Changes in luminal pH had an opposite effect on another inhibitor of PQH_2 oxidation at the Qo site, 2,4-dinitrophenyl ether of 2-iodo-4-nitrothymol (DNP-INT): acidic lumen pH promoted stronger DNP-INT inhibitory activity in the pea and spinach thylakoids [83, 85]. Binding of DNP-INT in the *pgr1* mutant was 20-50% stronger than in the WT [83]. The DNP-INT molecule lacks proton-donating groups and can only act as a hydrogen-bond acceptor via its nitro groups. Therefore, improved binding under conditions that increase the fraction of Cyt-b_6f with protonated H128 implies that ISP with the protonated H128 can still reside at the Qo site. Thus, a longer residence time of the protonated H128 in the proximal position would restrict PQH_2 oxidation in the absence of inhibitors. Since no fold difference in the DNP-INT binding was observed between the *pgr1* mutant and the WT, as was the case with DBMIB, these results indicate that the protonated H128 is neither the sole nor the key group involved in DNP-INT binding.

Inhibitory activity of DNP-INT in the spinach thylakoids is also enhanced when gramicidin D and valinomycin were added together [85]. This effect has been attributed to the ability of valinomycin in the complex with potassium ions to shield the ionized E/D side chains of proteins [86]. Such shielding could mimic the protonated state of E-channel and interfere with the H^+ release from E78, thus prolonging its protonated state and enabling formation of a hydrogen bond with DNP-INT. Hence, the higher Qo site affinity for DNP-INT at low luminal pH suggests that H^+ may persist on E78 and/or on other E-channel components, thereby slowing $PQH^{\bullet}$ oxidation.

Substitution of E78 with K or L in *C. reinhardtii* significantly slowed PQH_2 oxidation, especially at luminal pH 5-6 [63, 68]. This finding led to the idea that at neutral pH some alternative acceptor could take over the deprotonation of $PQH^{\bullet}$, whereas under acidic conditions E78 itself is indispensable for the efficient H^+ removal from the Qo site [68]. Together with the DNP-INT data [85], these results indicate that the E-channel contributes to PhotCon alongside the H-channel. Given that the predicted pKa of E78 is around 3.9 [60], at a lumen pH of 5-6, E78 should be in an ionized state. Therefore, it can be assumed that retention of H^+ on it might be related to the stronger local acidification of the lumen near the Cyt-b_6f, or it might be determined by the retention of H^+ on the intermediate proton-accepting groups within the E-channel, which could possess higher pKa values. For instance, the predicted pKa for the E3 (PetG) residue in the E-channel is 4.5 [60].

Altogether, the proton-releasing channels of Cyt-b_6f appear to serve as sensors of luminal pH for Qo site. They are hydrated cavities lined with bound water molecules (H-channel) and/or ionizable groups (E-channel) through which protons are transferred from one acceptor to the next. Acidification of lumen slows H^+ transfer along these channels, extending lifetimes of the initial proton acceptors – E78 and H128 – in their protonated forms. Different pK_a values of E78 and H128, coupled with the fact that H128 deprotonation is redox-controlled whereas E78 deprotonation is not, suggest two tiers of PhotCon activation that operate at different luminal pH levels. Unlike the H-channel, the E-channel makes direct contact with the Qo site. Hence, the prolonged protonated state of E78 is likely to modulate both redox chemistry of plastoquinone species and ROS formation within the Qo site (see below).

Link between photosynthetic control and protection of PSI against photoinhibition. Illuminating leaves with high light results in P_{700} oxidation, whereas other components of the chain become predominantly reduced [87]. This implies existence of a regulatory process that slows electron flow to PSI, and PhotCon is regarded as such process [88]. Yet this does not automatically mean that PhotCon prevents PI(I). The view of PhotCon as a PSI-protective mechanism under conditions of photoinhibition rests on a set of observations that are analyzed in this section.

Studying the impact of PhotCon is challenging due to the shortage of reliable protocols to detect

its activation. Researchers often use the parameter Y(ND) (fraction of PSI centers that can be oxidized in the light [10]) and interpret it as shortfall of electrons at the donor side of PSI caused by PhotCon. However, such limitation could also result from the down-regulation of PSII activity, or from the increased diversion of electrons to an alternative acceptor upstream of PSI. Suitability of the Y(ND) parameter as a PhotCon marker has been questioned recently [12]. Moreover, plants that display severe PI(I) typically show low Y(ND) values accompanied by high Y(NA) values, which is especially important given that inefficient electron outflow from PSI causes the acceptor-side photodamage (see above). An alternative method to assess PhotCon is to measure the rate of P_{700}^+ re-reduction upon switching off the light. Slowdown of P_{700}^+ re-reduction with increasing light intensity was observed in the *Silene dioica* leaves [89], but not in the pea leaves [90]; as suggested in [87], the latter study may have omitted the very low light intensities at which PhotCon is activated. Restriction of electron outflow from PSI has also been reported to activate PhotCon [89, 91]. Correlation between the P_{700}^+ re-reduction rate and the fraction of open PSII centers measured in a wide range of light intensities has been suggested as an estimate for PhotCon [87].

Studying protective role of PhotCon is also complicated by the difficulty of separating the effects on the donor and acceptor sides of PSI. During chilling stress, for instance, dissociation of the coupling factor of chloroplast ATP-synthase has been proposed to raise conductivity of the thylakoid membrane to protons, leading to collapse of ΔpH and absence of PhotCon activation [2]. Yet that same dissociation also decreases ATP synthesis, thus slowing the CBB cycle and restricting electron outflow from PSI, which is the primary trigger of the acceptor-side PI(I). Whether PhotCon itself protects PSI under chilling stress therefore remains uncertain.

Infiltration of Arabidopsis leaves with diuron, an inhibitor of electron transport from PSII to the PQ pool, mitigated the loss of PSI activity in high light [8]. The authors interpreted this as evidence that PhotCon prevents PI(I). In reality, absence of electron flow to PSI also removes the electrons that would otherwise reduce O_2 and generate $HO^\bullet$ by the F_A/F_B clusters, the root cause of acceptor-side PI(I). Thus, diuron more likely suppressed ROS formation in PSI rather than imitated PhotCon activation. Infiltration of Arabidopsis leaves with nigericin, a proton-potassium antiporter that dissipates ΔpH but not the electrical component of pmf, led to the significant loss of PSI activity in high light [88]. According to the authors, ATP synthesis by ATP-synthase complexes was not disrupted under these conditions. However, because neither ATP-synthase activity nor CO_2 assimilation were measured, we cannot completely rule out an explanation associated with insufficient ATP supply for optimal functioning of the CBB cycle.

For several Arabidopsis mutants, a correlation has been observed between the high level of PI(I) and elevated values of conductivity of the thylakoid membranes for H^+ (the gH^+ parameter), which are accompanied by the decrease in pmf. In the *cfq* (coupling factor quick recovery) mutant, the point substitution E244K in the γ_1-subunit of ATP-synthase disrupts the thiol-dependent regulation of the enzyme, thereby increasing H^+ leakage through the c-ring of ATP synthase [92]. In the *hope2* (hunger for oxygen in photosynthetic electron transport reaction 2) mutant, the G134D substitution in the same γ_1-subunit is thought to impair metabolic regulation of ATP-synthase, likewise enhancing the rate of H^+ efflux through the c-ring [93]. In the Arabidopsis mutant DPGRox, the variant expressing the potassium-proton antiporter KEA3 with G422R point substitution, the increased H^+ efflux from the lumen to the stroma via this antiporter was observed [94]. Comparison of these mutants suggests that the cause of PI(I) is the increased dissipation of ΔpH, leading the authors to conclude that the failure of PhotCon activation underlies PI(I). However, the elevated gH^+ values in the DPGRox mutant may indicate insufficient ATP synthesis for optimal CBB cycle activity. For the *cfq* and *hope2* mutants, it has been proposed by the authors that the enhanced H^+ leakage through the c-ring of ATP-synthase is accompanied by the extra ATP production [92, 93]. Yet the *in vitro* experiments with the *cfq* mutant showed that both the synthase and hydrolase activities of ATP-synthase were lower than in the WT [95]. Indeed, all three mutants exhibited not only low Y(ND) values, which could be explained by the increased ΔpH dissipation, but also high Y(NA) values [92-94, 96] indicating restricted electron outflow from PSI.

In our opinion, the most convincing evidence comes from the Arabidopsis *pgr1* mutant, in which the P194L substitution in ISP shifts activation of PhotCon to more alkaline luminal pH values. In this mutant, the PSI was inhibited to a lesser extent than in the WT upon exposure to FL, indicating protective effect of PhotCon in the context of PI(I) [10]. Introducing the ISP-P194L point substitution into the *pgr5* mutant likewise made PSI more resistant to FL in the mature plants in comparison with the single *pgr5* mutant. The Y(ND) values in the double mutant, though slightly higher than in the single *pgr5* mutant, still remained significantly lower than in the WT or the single *pgr1* mutant. Thus, the enhanced PSI stability in the double mutant cannot be attributed solely to the forced activation of PhotCon at more alkaline luminal pH values.

Taken together, these results support the idea that PhotCon might protect PSI under conditions of

photoinhibition, even those leading to the acceptor-side damage. Yet each individual observation still allows alternative explanations, such as, for example, that the impaired ATP synthesis would slow CBB cycle turnover at the onset of stress and thereby favor the acceptor-side limitation of PSI.

Is the defective photosynthetic control the cause of PSI photoinhibition in the pgr5 mutants? The increased PI(I) in the *pgr5* mutant is also often linked to the impaired PhotCon activation. The *pgr5* mutant was first isolated during the screening of Arabidopsis lines for the reduced induction of non-photochemical quenching due to the lower pmf values [9]. The *in vitro* experiments showed that the AA-sensitive CET(I) is absent in this mutant [9]. However, the *in vivo* studies showed that under non-stress conditions, the *pgr5* mutant can perform CET(I) as efficiently as WT, and the PGR5 protein is essential for enhancing CET(I) under abrupt changes of conditions [97]. The *pgr5* mutant is unable to grow under FL, and the reason for this is its pronounced PI(I) [8]. This example underscores both the necessity of preventing PI(I) for plant survival and the key role of PGR5 in PSI protection in C_3 plants. As mentioned above, introducing the *pgr1* mutation (ISP-P194L) into the *pgr5* mitigated PI(I) in mature plants [10]; nevertheless, this does not necessarily mean that the defective PhotCon is the primary cause of the strong PI(I) seen in the single *pgr5* mutant. Expression of the flavodiiron protein genes of *Physcomitrella patens* moss, which provide an efficient electron outflow from PSI, in the Arabidopsis *pgr5* mutant diminished PI(I) under FL [10, 43], confirming that PI(I) in the single *pgr5* mutant is associated with restricted electron outflow from PSI.

Screening for the secondary Arabidopsis mutations that enable *pgr5* seedlings to survive FL was carried out in the study [98], where several alterations affecting PSII function and stability, Cyt-b_6f assembly, Pc biosynthesis, chloroplast fructose-1,6-bisphosphatase, and other factors were identified, all of which partially compensated negative effects of the absence of functional PGR5 protein. However, targeted introduction of the ISP-P194L substitution (the *pgr1* mutation) into the *pgr5* did not provide seedling survival under FL. Hence, artificially forcing PhotCon by raising the pK_a of H128 cannot counteract physiological impairment resulting from the absence of functional PGR5 protein during the long-term exposure of the plant to FL over the vegetative period.

Recently, it was shown that, unlike the WT, the *pgr5* mutant displays oscillatory changes in the chlorophyll fluorescence, P_{700} absorbance, and the signal of electrochromic shift of carotenoid absorbance in response to abrupt changes in light intensity or CO_2 concentration [99]. Such oscillations were not observed in the *hope2* mutant, which showed impaired induction of the parameter Y(ND) interpreted as activation of PhotCon. The authors concluded that the oscillations in the *pgr5* mutant are not associated with the impaired PhotCon activation; instead, these oscillations arise from the ATP deficiency that develops under abrupt environmental changes. This interpretation is consistent with the earlier work proposing that the PGR5-dependent CET(I) is required primarily to balance ATP production relative to NADPH [9, 100]. It is worth noting that the *hope2* mutant also exhibited increased activity of the PGR5-dependent CET(I) [96], which could counteract negative effect of the elevated gH^+ and thereby dampen potential oscillations.

In summary, the recent evidence indicates that the increased PI(I) in the *pgr5* mutant is not caused by the defective PhotCon activation but is, most likely, related to insufficient ATP production and inability to balance between ATP and NADPH.

Hypothetical mechanism by which photosynthetic control might suppress ROS formation in PSI. The proposed role of PhotCon in PSI protection is commonly linked to the decrease in ROS formation within PSI, although there is no direct experimental evidence for this claim. It remains unclear how PhotCon can decrease ROS generation in PSI, and which specific ROS are affected. PSI produces $O_2^{\bullet-}$, $HO^{\bullet}$, and $^1O_2$ (see above); all three have been suggested as immediate damaging agents in PSI under different scenarios of photoinhibition. A series of studies by K. Sonoike showed that the key factor in the acceptor-side PI(I) is not the amount of H_2O_2 produced near the terminal F_A/F_B clusters but rather efficiency of the electron outflow from these clusters, which determines the probability of $HO^{\bullet}$ generation (see above). We, therefore, hypothesize that PhotCon activation could modify $HO^{\bullet}$ generation while not affecting the rate of H_2O_2 formation close to F_A/F_B. There is still no clear understanding of how the luminal pH influences O_2 reduction in PSI. However, the work with the isolated PSI complexes from the cyanobacterium *Synechocystis* sp. PCC 6803 and the alga *C. reinhardtii* showed that increasing concentration over a wide range of either the artificial electron donor *N,N,N′,N′*-tetramethyl-*p*-phenylenediamine or the natural donor Pc did not increase the $O_2^{\bullet-}$ production (and thus production of H_2O_2) by PSI, even though it improved efficiency of electron donation to P_{700}^+ [25, 101]. High donor concentrations in such systems mimic rapid electron flow to PSI due to the absence of PhotCon activation. Moreover, the rate of O_2 reduction in the isolated *C. reinhardtii* PSI complexes changed only negligibly when the electron outflow from PSI to Fd and $NADP^+$ was enabled [25, 26, 102]. These observations confirm that the decrease in the P_{700}^+ reduction rate due to PhotCon activation does not necessarily lead to the decrease in generation of $O_2^{\bullet-}$ and, consequently, of H_2O_2 in PSI.

In the absence of MV or other efficient acceptors, the principal route for oxidizing the F_A/F_B clusters is charge recombination with P_{700}^+ [41]. Activation of PhotCon slows P_{700}^+ re-reduction, allowing electrons from F_A/F_B to return to P_{700}^+ and thus preventing the reaction of F_A/F_B with H_2O_2. In the case of $^1O_2$, which is assumed to be responsible for PI(I) during rSP treatments, enabling safe charge recombination from F_A/F_B due to a slower electron flow from Cyt-b_6f to PSI might likewise diminish charge recombination with the intermediate PSI cofactors and thereby suppress $^1O_2$ generation.

Photosynthetic control modulates the functioning of cytochrome b_6f complex. Besides directly limiting the rate of electron donation to PSI, PhotCon necessarily influences processes associated with Cyt-b_6f activity and themselves affecting energy balance in the PETC: operation of CET(I), activation/deactivation of the STN7 kinase, and ROS formation within Cyt-b_6f.

ROS formation. Oxidative modifications of amino acid residues in close vicinity of the Cyt-b_6f prosthetic groups have been detected in the Cyt-b_6f complexes purified from the field-grown spinach, indicating ROS production associated with these groups [103]. As in PSI, Cyt-b_6f can generate $^1O_2$ (via its single chlorophyll *a* molecule) [104], $HO^\bullet$ (via the ISP Fe_2-S_2 cluster), and $O_2^{\bullet-}$ (upon oxidation by O_2 of electron transfer cofactors possessing sufficiently low redox potential) [47]. The semiquinone formed in the Qo site is generally considered as the main source of $O_2^{\bullet-}$, although the role of heme b_p is not ruled out [103, 105]. It should be noted that $PQH^\bullet$ can reside transiently in the spin-coupled state with the reduced Fe_2-S_2 cluster, a state that does not react with O_2 [106]. $O_2^{\bullet-}$ undergoes dismutation with another $O_2^{\bullet-}$ to yield H_2O_2, but it is unclear how efficient is this reaction directly within the Qo site, because proton availability is a limiting factor for this reaction. In any case, $O_2^{\bullet-}$ could produce H_2O_2 upon exiting into the lumen, which could occur via diffusion through the E-channel. It is also plausible that the PQH_2 molecule within the Qo site or *en route* to it inside the complex, could reduce $O_2^{\bullet-}$ to H_2O_2, as has been demonstrated for PQH_2 molecules in the membrane [107]. In this regard, it is worth mentioning that quantitative assessments of the $O_2^{\bullet-}$ generation rate by Cyt-b_6f [105] were derived from the experiments that monitored fluorescence of resorufin, produced in the reaction of Amplex Red dye with H_2O_2, but not $O_2^{\bullet-}$.

Discussing effects of PhotCon on generation of $^1O_2$ in Cyt-b_6f is complicated by the absence of information on how conformational changes in the chlorophyll *a* molecule position might be linked to lumen pH, and how these changes could affect probability of the molecule transitioning to triplet state, which is required for generation of $^1O_2$. By contrast, there is a stronger evidence to suggest that PhotCon suppresses electron leakage to O_2. When PhotCon is activated, the PQ pool becomes more reduced, limiting availability of PQ molecules for the Qr site and for Q-cycle turnover. This would increase the probability of reverse electron transfer from heme b_n to heme b_p, which increases the probability of heme b_p oxidation by O_2; however, the reaction of heme b_p with O_2 should be suppressed by the subsequent electron transfer from heme b_p to quinone species in the Qo site. Reduction of PQ or $PQ^{\bullet-}$ by heme b_p is thermodynamically unfavorable, whereas reduction of $PQH^\bullet$ by heme b_p is thermodynamically more favorable. As stated above, lifetime of $PQH^\bullet$ is governed by the protonated state of E78, manifestation of PhotCon activation. Prolonging the lifetime of $PQH^\bullet$ itself in the Qo site decreases the probability of $O_2^{\bullet-}$ formation, because $PQH^\bullet$ reduces O_2 less efficiently than $PQ^{\bullet-}$ due to its more positive redox potential [108]. Another manifestation of PhotCon activation is longer lifetime of the protonated H128. On the one hand, this could retain the Fe_2-S_2 cluster in its distal position, away from the low-potential cofactors capable of producing $O_2^{\bullet-}$ and H_2O_2 with the Qo site, thereby minimizing the risk of forming the most dangerous ROS, $HO^\bullet$. On the other hand, it would hinder catalytic oxidation of PQH_2 in the Qo site, increasing likelihood of PQH_2 reacting with $O_2^{\bullet-}$ and converting it into the less reactive H_2O_2. Thus, PhotCon activation should decrease formation of both $O_2^{\bullet-}$ and $HO^\bullet$, lowering the chance of oxidative protein modifications that could impair the entire complex. However, the amount of H_2O_2 produced would depend on the combined reactions, so PhotCon could in principle increase the H_2O_2 levels in Cyt-b_6f by both preventing H_2O_2 decomposition into $HO^\bullet$ within the Qo site and stimulating the reaction of $O_2^{\bullet-}$ with PQH_2 instead of $O_2^{\bullet-}$ dismutation.

Activation of the STN7/STT7 kinase. The STN7/STT7 kinase is structurally and functionally associated with Cyt-b_6f [19, 20] and phosphorylates several proteins [109, 110], primarily the light-harvesting complex II (LHCII) proteins. Redistribution of phosphorylated LHCII from PSII to PSI could also reduce PI(I) under FL [111]. STN7 has also been shown to phosphorylate FNR [110], which is likely involved in switching between the linear electron transport and CET(I) [51], and Thylakoid Soluble Protein 9 [109], a protein that binds to the stromal side of Cyt-b_6f and, likely, contributes to CET(I) regulation [67]. Within the STN7 context, two distinct but related issues remain unresolved: (i) the mechanism of kinase activation, which is tied to PQH_2 oxidation in Cyt-b_6f, and (ii) the mechanism of kinase inactivation in high light. In particular, one activation model posits that the lumen-facing N-terminus of STN7 contacts

the hydrophilic domain of ISP; during the conformational changes accompanying electron transfer from PQH_2 to cyt *f*; this contact induces the kinase switch to its active dimeric state via formation of an intermolecular disulfide bridge between the N-termini of two STN7 proteins [112]. Suppression of the STN7 activity in high light is believed to involve reduction of this intermolecular disulfide by the trans-membrane protein system that transfers electrons from stromal thioredoxins to the luminal thiol-regulated targets [113] followed by formation of an intramolecular disulfide bridge that renders STN7 inactive. H_2O_2 produced by the PETC components in high light may participate in this inactivation; indeed, adding catalase, which decomposes H_2O_2, to the Arabidopsis thylakoids led to the increased accumulation of phosphorylated LHCII proteins in high light [114].

In vitro experiments showed that lumen acidification lowered accumulation of the phosphorylated LHCII proteins in the maize chloroplasts [115] indicating correlation between the conditions that trigger PhotCon and STN7 kinase activity. One possible explanation is that lowering lumen pH raises the midpoint redox potentials (E_m) of the thiol groups in STN7 and in other proteins involved in regulating its activity [113] with simultaneous increase the E_m of H_2O_2, making it a stronger oxidant. Another explanation is direct effect of PhotCon on the STN7 activity [20]. For instance, when the luminal pH drops below the pK_a of H128, the hydrophilic domain in ISP may reside longer near the cyt *f*, which could restrict the domain interaction with the N-terminus of STN7 and thereby suppress kinase activation. As noted above, PhotCon could hypothetically increase the H_2O_2 production in Cyt-b_6f; the produced H_2O_2 might inactivate STN7 by oxidizing its thiols with formation of an intramolecular disulfide bond.

The Q-cycle and the AA-sensitive CET(I). Electron transfer along the low-potential branch of Cyt-b_6f reduces the PQ molecule to PQH_2 in the Qr site via the Q-cycle, which can operate in two modes: (i) when both electrons needed to reduce PQ in the Qr site originate from the Qo site via sequential oxidation of two PQH_2 molecules (an electron from the first PQH_2 molecule is transferred to heme c_n, an electron from the second PQH_2 molecule is transferred to the PQ molecule from heme b_n simultaneously with the transfer of an electron from heme c_n to PQ) and (ii) when one electron for PQ reduction comes from the Qo site (via heme b_n), while the second is supplied by the reduced Fd through heme c_n [19]. This latter mode is realized during the AA-sensitive CET(I), in which Cyt-b_6f, either alone or in complex with FNR, oxidizes Fd and reduces PQ, provided that PGR5 is present. In the *C. reinhardtii* lacking PGR5, switching between these two Q-cycle modes is impaired [18]. Several hypotheses have been proposed to explain how competition between the two modes is regulated when Fd can donate electrons to Cyt-b_6f [20, 64]. Recent measurements of the redox potentials of cyt b_6 hemes have shown that the E_m of heme b_n is about 30-50 mV more negative than that of heme b_p [64]. In the process, endergonic electron transfer from heme b_p to heme b_n becomes feasible because it is coupled to the subsequent exergonic electron transfer to the PQ molecule in the Qr site or to heme c_n, resulting in the net decrease in free energy. Consequently, thermodynamic efficiency of the electron flow through the low-potential branch under steady-state conditions would depend on the proportion of oxidized PQ molecules in the membrane.

As noted above, activation of PhotCon could restrict availability of PQ for operation of the Q-cycle (although it is not ruled out that a PQ molecule from the Qo site may reach the Qr site without ever leaving Cyt-b_6f [67]). Prolonging the lifetime of protonated H128 could keep the ISP in its distal position, slowing turnover of the complex. Extending lifetime of the protonated E78 should slow oxidation of $PQH^{\bullet}$ by heme b_p. However, pK_a of E78 is lower than pK_a of H128 (see above), and with a moderate drop in luminal pH, PhotCon would be mediated mainly through the longer lifetime of the protonated H128, whereas E78 would remain predominantly ionized. Under these conditions the overall turnover of the complex would be slower, but the efficiency of electron transfer along the low-potential branch would be unaffected, maintaining rapid reduction of PQ at the Qr site.

When electrons are donated from Fd, the PhotCon-induced slowdown in the Cyt-b_6f turnover would also slow CET(I), because the PQH_2 generated in the Qr site must still be oxidized on the luminal side of Cyt-b_6f, and PhotCon, due to protonated H128, would limit the steady-state CET(I). Contradictions arise in this case, since it is often assumed that under the abrupt environmental changes CET(I) accelerates to acidify the lumen and activate PhotCon [2]. These contradictions could be resolved by assuming that Cyt-b_6f in the grana and unstacked lamellae perform different functions: electron transfer from PSII to Pc and operation of CET(I) respectively [23, 24]. The key factor is heterogeneity of the luminal pH [116]: in the grana, where PSII releases H^+ into the lumen and efficient H^+ efflux pathways are scarce, the lumen pH drops more sharply than in the stromal thylakoid regions enriched with ATP-synthase complexes that dissipate ΔpH. Consequently, abrupt environmental changes would activate PhotCon in the granal Cyt-b_6f rather than in the stromal Cyt-b_6f, thereby slowing PQH_2 oxidation specifically during linear electron flow from PSII to PSI, but not during CET(I) (Fig. 4). Moreover, activation of PhotCon in the grana would favor CET(I) over the linear flow, thereby maintaining proper

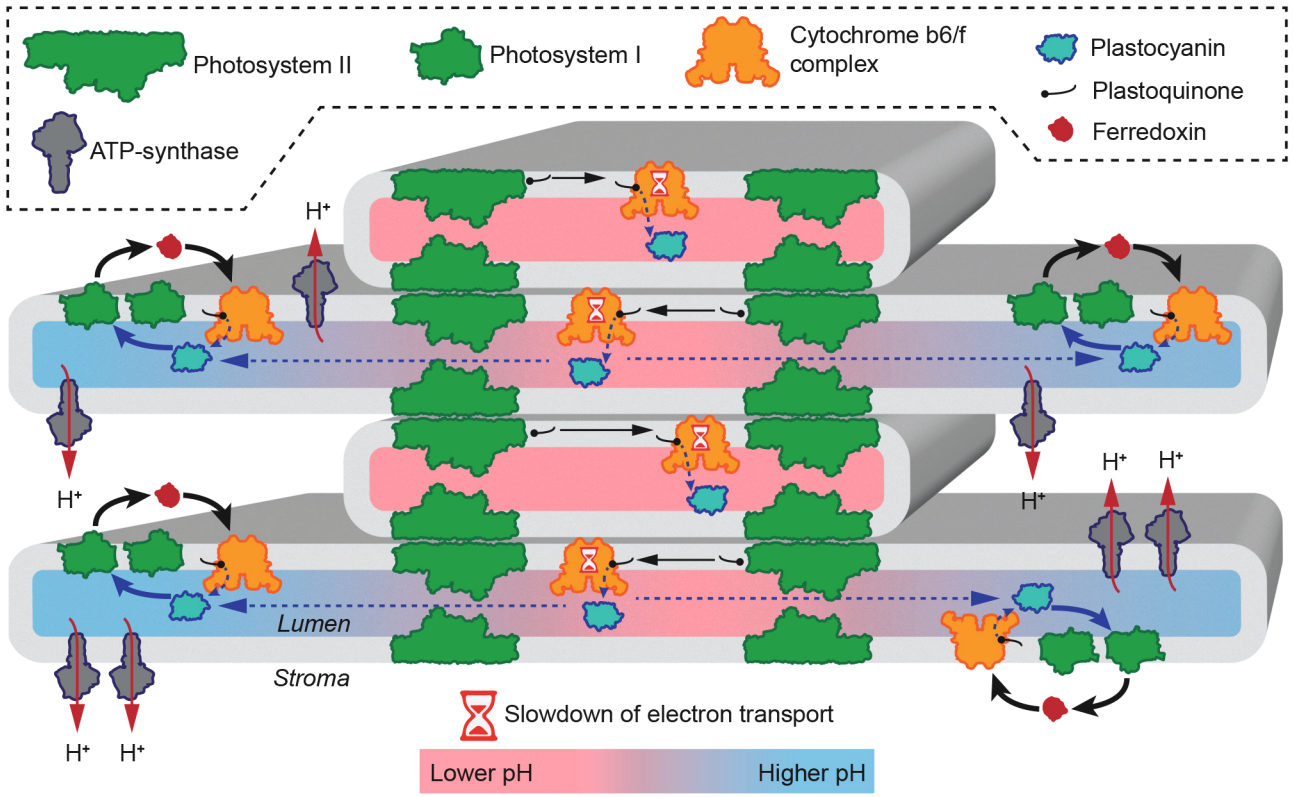

Fig. 4. Schematic representation of PhotCon activation under moderate lumen acidification, taking into account lateral heterogeneity of both the thylakoid membrane and lumen pH. The color gradient from red to blue depicts luminal pH gradient between grana and stromal lamellae (pH ≪ 7 in grana; pH < 7 in stromal regions). The red arrow indicates H^+ diffusion; the blue arrow shows Pc diffusion (dashed: linear electron flow; solid: CET(I)). The black arrow denotes electron transfer from PSI to the Cyt-b_6f via Fd during CET(I).

ATP/NADPH balance. It is this effect, rather than direct suppression of ROS formation in PSI, that could explain how PhotCon protects PSI under conditions that cause the acceptor-side PI(I), particularly under FL.

Under more severe stress, when ATP-synthase activity declines, the luminal pH of both the granal and stromal thylakoid regions is expected to drop, so PhotCon could also be triggered in the stromal Cyt-b_6f, thereby influencing CET(I). In this case, there is obviously no physiological need to supply additional H^+ to the lumen. Nevertheless, the electrons from Fd could still enter heme c_n, and because the PQ pool is virtually fully reduced under such conditions, efficiency of the electron transfer into the PQ pool is decreased. A hypothetical electron transfer from Fd to quinone species in the Qo site – a speculative (i.e., experimentally unverified) model of CET(I) that bypasses the bulk PQ pool – has been discussed in the review [19]. The possibility of this pathway depends on which quinone species occupies the Qo site. Reverse electron transfer from heme c_n to PQ in the Qo site comprises two endergonic steps (Fig. 3a) not coupled to a sufficiently exergonic reaction, making the process thermodynamically unfavorable. Electron transfer to the $PQ^{\bullet-}$ radical (forming the dianion PQ^{2-}) is also thermodynamically unfavorable in the absence of free protons. By contrast, transferring an electron to $PQH^{\bullet}$ (forming PQH^-) is favorable and energetically profitable, compensating for the endergonic step of electron transfer from heme c_n to heme b_n. As noted above, likelihood of $PQH^{\bullet}$ deprotonation to $PQ^{\bullet-}$ in the Qo site should be governed by the lifetime of protonated E78. Therefore, activation of PhotCon at the level of E78 would facilitate an alternative CET(I) pathway where electrons do not exit the Cyt-b_6f into the PQ pool. This alternative CET(I) would not pump extra H^+ to the lumen (which is unnecessary under the described conditions), yet it could still play a physiological role by ensuring steady-state PSI operation with zero net redox balance.

CONCLUSION

In this review we have thoroughly examined the causes of PSI photoinhibition and proposed that it proceeds by two distinct mechanisms leading to primary damage on either the acceptor or the donor side of PSI. We have raised the question of how experimentally substantiated is the notion that PhotCon functions

as a protective mechanism preventing PI(I). Our analysis of the literature on the *pgr5* mutant indicates that the key role of the PGR5 protein in preventing PI(I) lies in regulating CET(I) for supplying additional ATP, rather than for PhotCon activation. We suggest that activation of PhotCon under conditions of photoinhibition could suppress generation of the more reactive species ($HO^{\bullet}$ and $^1O_2$) in the PSI rather than $O_2^{\bullet-}$ or H_2O_2.

We have summarized current knowledge on the proton-releasing channels of the Qo site of the Cyt-b_6f and their role in PQH_2 oxidation, providing a more comprehensive understanding of the mechanisms underlying the Cyt-b_6f sensitivity to luminal pH and activation of PhotCon. We conclude that both amino acid residues accepting H^+ during PQH_2 oxidation, H128 of the ISP and E78 of the subunit IV, are involved in PhotCon activation. However, due to the differences in pK_a values, PhotCon activation at the level of these residues would occur at different lumen pH values. We have considered how PhotCon activation, including at the level of protonated E78, could hypothetically influence Cyt-b_6f functioning: its ROS production, STN7 kinase activation and deactivation, and operation of the AA-sensitive CET(I) pathway. Based on the notion of lateral heterogeneity of both thylakoid structure and luminal pH, we propose that PhotCon slows linear electron transport in the Cyt-b_6f located in the grana. Meanwhile, in the stromal lamellae, where the lumen pH drops to a lesser extent, the Cyt-b_6f activity under these same conditions would not slow down, thus facilitating CET(I). This could explain the apparent paradox in the scientific community: abrupt environmental changes simultaneously accelerate CET(I) and activate PhotCon. The enhanced CET(I) in the stromal thylakoids is crucial for additional ATP synthesis and maintenance of an optimal ATP/NADPH ratio for the CBB cycle and other chloroplast metabolic pathways. This, in turn, promotes efficient electron outflow from PSI, preventing PI(I). It is this competitive advantage provided to CET(I) in the stromal lamellae, achieved by suppressing linear transport at the grana level, that may underlie the protective action of PhotCon, preventing PI(I) on the acceptor side of PSI.

Ultimately, this review encourages readers to re-evaluate the mechanisms of PhotCon activation and its physiological role in regulating photosynthetic apparatus function.

Abbreviations. ΔpH, trans-thylakoid pH gradient; AA, antimycin A; CET(I), cyclic electron transport around PSI; Cyt, cytochrome; Cyt-bc_1 and Cyt-b_6f – cytochrome bc_1 and b_6f complexes, respectively; DBMIB, 2,5-dibromo-3-methyl-6-isopropylbenzoquinone; DNP-INT, 2,4-dinitrophenyl ether of 2-iodo-4-nitrothymol; E- and H-channels: proton-releasing channels that remove protons from E78 and H128, respectively; Fd, ferredoxin; FL, fluctuating light; FNR, ferredoxin-NADP-reductase; ISP, iron-sulfur protein; LHCII, light-harvesting complex II; MV, methyl viologen; Pc, plastocyanin; PETC, photosynthetic electron transport chain; PGR5, proton gradient regulation 5; PhotCon, photosynthetic control; PI(I) and PI(II), photoinhibition of photosystem I and photosystem II, respectively; pmf, proton motive force; PQ, PQH_2, $PQH^{\bullet}$, plastoquinone, plastohydroquinone, and plastosemiquinone, respectively; PSI and PSII, photosystem I and photosystem II, respectively; ROS, reactive oxygen species; rSP, repetitive short pulses; WT, wild type; Y(NA) and Y(ND), quantum yields of non-photochemical losses on the acceptor and donor sides of PSI, respectively.

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Conflict of interest. The authors of this work declare that they have no conflicts of interest.

REFERENCES

1. Terashima, I., Funayama, S., and Sonoike, K. (1994) The site of photoinhibition in leaves of *Cucumis sativus* L. at low temperatures is Photosystem I, not Photosystem II, *Planta*, **193**, 300-306, https://doi.org/10.1007/BF00192544.
2. Kono, M., Oguchi, R., and Terashima, I. (2022) Photoinhibition of PSI and PSII in nature and in the laboratory: ecological approaches, in *Progress in Botany* (Lüttge, U., Cánovas, F. M., Risueño, M.-C., Leuschner, C., and Pretzsch, H., eds) Vol. 84, Springer Nature Switzerland, pp. 241-292, https://doi.org/10.1007/124_2022_67.
3. Tikkanen, M., Grieco, M., Kangasjärvi, S., and Aro, E.-M. (2010) Thylakoid protein phosphorylation in higher plant chloroplasts optimizes electron transfer under fluctuating light, *Plant Physiol.*, **152**, 723-735, https://doi.org/10.1104/pp.109.150250.
4. Sejima, T., Takagi, D., Fukayama, H., Makino, A., and Miyake, C. (2014) Repetitive short-pulse light mainly inactivates Photosystem I in sunflower leaves, *Plant Cell Physiol.*, **55**, 1184-1193, https://doi.org/10.1093/pcp/pcu061.

5. Lempiäinen, T., Rintamäki, E., Aro, E.-M., and Tikkanen, M. (2022) Plants acclimate to Photosystem I photoinhibition by readjusting the photosynthetic machinery, *Plant Cell Environ.*, **45**, 2954-2971, https://doi.org/10.1111/pce.14400.
6. Lima-Melo, Y., Kılıç, M., Aro, E.-M., and Gollan, P. J. (2021) Photosystem I inhibition, protection and signalling: knowns and unknowns, *Front. Plant Sci.*, **12**, 791124, https://doi.org/10.3389/fpls.2021.791124.
7. Shimakawa, G., and Miyake, C. (2018) Oxidation of P700 ensures robust photosynthesis, *Front. Plant Sci.*, **9**, 1617, https://doi.org/10.3389/fpls.2018.01617.
8. Suorsa, M., Järvi, S., Grieco, M., Nurmi, M., Pietrzykowska, M., Rantala, M., Kangasjärvi, S., Paakkarinen, V., Tikkanen, M., Jansson, S., and Aro, E.-M. (2012) PROTON GRADIENT REGULATION5 is essential for proper acclimation of Arabidopsis Photosystem I to naturally and artificially fluctuating light conditions, *Plant Cell*, **24**, 2934-2948, https://doi.org/10.1105/tpc.112.097162.
9. Munekage, Y., Hojo, M., Meurer, J., Endo, T., Tasaka, M., and Shikanai, T. (2002) PGR5 is involved in cyclic electron flow around Photosystem I and is essential for photoprotection in Arabidopsis, *Cell*, **110**, 361-371, https://doi.org/10.1016/S0092-8674(02)00867-X.
10. Yamamoto, H., and Shikanai, T. (2019) PGR5-dependent cyclic electron flow protects Photosystem I under fluctuating light at donor and acceptor sides, *Plant Physiol.*, **179**, 588-600, https://doi.org/10.1104/pp.18.01343.
11. Shikanai, T. (2024) Molecular genetic dissection of the regulatory network of proton motive force in chloroplasts, *Plant Cell Physiol.*, **65**, 537-550, https://doi.org/10.1093/pcp/pcad157.
12. Degen, G. E., and Johnson, M. P. (2024) Photosynthetic control at the cytochrome b_6f complex, *Plant Cell*, **36**, 4065-4079, https://doi.org/10.1093/plcell/koae133.
13. Hanke, G., and Mulo, P. (2013) Plant type ferredoxins and ferredoxin-dependent metabolism, *Plant Cell Environ.*, **36**, 1071-1084, https://doi.org/10.1111/pce.12046.
14. Davis, G. A., Rutherford, A. W., and Kramer, D. M. (2017) Hacking the thylakoid proton motive force for improved photosynthesis: modulating ion flux rates that control proton motive force partitioning into Δψ and ΔpH, *Philos. Trans. R. Soc. B Biol. Sci.*, **372**, 20160381, https://doi.org/10.1098/rstb.2016.0381.
15. Allen, J. F. (2002) Photosynthesis of ATP – electrons, proton pumps, rotors, and poise, *Cell*, **110**, 273-276, https://doi.org/10.1016/S0092-8674(02)00870-X.
16. Munekage, Y., Hashimoto, M., Miyake, C., Tomizawa, K.-I., Endo, T., Tasaka, M., and Shikanai, T. (2004) Cyclic electron flow around photosystem I is essential for photosynthesis, *Nature*, **429**, 579-582, https://doi.org/10.1038/nature02598.
17. Takabayashi, A., Kishine, M., Asada, K., Endo, T., and Sato, F. (2005) Differential use of two cyclic electron flows around photosystem I for driving CO_2-concentration mechanism in C4 photosynthesis, *Proc. Natl. Acad. Sci. USA*, **102**, 16898-16903, https://doi.org/10.1073/pnas.0507095102.
18. Buchert, F., Mosebach, L., Gäbelein, P., and Hippler, M. (2020) PGR5 is required for efficient Q cycle in the cytochrome b_6f complex during cyclic electron flow, *Biochem. J.*, **477**, 1631-1650, https://doi.org/10.1042/BCJ20190914.
19. Sarewicz, M., Pintscher, S., Pietras, R., Borek, A., Bujnowicz, Ł., Hanke, G., Cramer, W. A., Finazzi, J., and Osyczka, A. (2021) Catalytic reactions and energy conservation in the cytochrome bc_1 and b_6f complexes of energy-transducing membranes, *Chem. Rev.*, **121**, 2020-2108, https://doi.org/10.1021/acs.chemrev.0c00712.
20. Malone, L. A., Proctor, M. S., Hitchcock, A., Hunter, C. N., and Johnson, M. P. (2021) Cytochrome b_6f – orchestrator of photosynthetic electron transfer, *Biochim. Biophys. Acta Bioenerg.*, **1862**, 148380, https://doi.org/10.1016/j.bbabio.2021.148380.
21. Asada, K. (1999) The water-water cycle in chloroplasts: scavenging of active oxygens and dissipation of excess photons, *Annu. Rev. Plant. Physiol. Plant. Mol. Biol.*, **50**, 601-639, https://doi.org/10.1146/annurev.arplant.50.1.601.
22. Ilík, P., Pavlovič, A., Kouřil, R., Alboresi, A., Morosinotto, T., Allahverdiyeva, Y., Aro, E.-M., Yamamoto, H., and Shikanai, T. (2017) Alternative electron transport mediated by flavodiiron proteins is operational in organisms from cyanobacteria up to gymnosperms, *New Phytol.*, **214**, 967-972, https://doi.org/10.1111/nph.14536.
23. Wietrzynski, W., Lamm, L., Wood, W. H., Loukeri, M.-J., Malone, L., Peng, T., Johnson, M. P., and Engel, B. D. (2025) Molecular architecture of thylakoid membranes within intact spinach chloroplasts, *eLife*, **14**, RP105496, https://doi.org/10.7554/eLife.105496.1.
24. Höhner, R., Pribil, M., Herbstová, M., Lopez, L. S., Kunz, H.-H., Li, M., Wood, M., Svoboda, V., Puthiyaveetil, S., Leister, D., and Kirchhoff, H. (2020) Plastocyanin is the long-range electron carrier between Photosystem II and Photosystem I in plants, *Proc. Natl. Acad. Sci. USA*, **117**, 15354-15362, https://doi.org/10.1073/pnas.2005832117.
25. Kozuleva, M., Petrova, A., Milrad, Y., Semenov, A., Ivanov, B., Redding, K. E., and Yacoby, I. (2021) Phylloquinone is the principal Mehler reaction site within Photosystem I in high light, *Plant Physiol.*, **186**, 1848-1858, https://doi.org/10.1093/plphys/kiab221.
26. Kozuleva, M. A., and Ivanov, B. N. (2010) Evaluation of the participation of ferredoxin in oxygen reduction in the photosynthetic electron transport chain

of isolated pea thylakoids, *Photosynth. Res.*, **105**, 51-61, https://doi.org/10.1007/s11120-010-9565-5.

27. Šnyrychová, I., Pospíšil, P., and Nauš, J. (2006) Reaction pathways involved in the production of hydroxyl radicals in thylakoid membrane: EPR spin-trapping study, *Photochem. Photobiol. Sci.*, **5**, 472-476, https://doi.org/10.1039/B514394B.
28. Takagi, D., Takumi, S., Hashiguchi, M., Sejima, T., and Miyake, C. (2016) Superoxide and singlet oxygen produced within the thylakoid membranes both cause Photosystem I photoinhibition, *Plant Physiol.*, **171**, 1626-1634, https://doi.org/10.1104/pp.16.00246.
29. Kale, R., Sallans, L., Frankel, L. K., and Bricker, T. M. (2020) Natively oxidized amino acid residues in the spinach PS I-LHC I supercomplex, *Photosynth. Res.*, **143**, 263-273, https://doi.org/10.1007/s11120-019-00698-7.
30. Mathis, P., and Setif, P. (1981) Near infra-red absorption spectra of the chlorophyll a cations and triplet state *in vitro* and *in vivo*, *Isr. J. Chem.*, **21**, 316-320, https://doi.org/10.1002/ijch.198100057.
31. Caspy, I., Borovikova-Sheinker, A., Klaiman, D., Shkolnisky, Y., and Nelson, N. (2020) The structure of a triple complex of plant Photosystem I with ferredoxin and plastocyanin, *Nat. Plants*, **6**, 1300-1305, https://doi.org/10.1038/s41477-020-00779-9.
32. Sonoike, K., Terashima, I., Iwaki, M., and Itoh, S. (1995) Destruction of Photosystem I iron-sulfur centers in leaves of *Cucumis sativus* L. by weak illumination at chilling temperatures, *FEBS Lett.*, **362**, 235-238, https://doi.org/10.1016/0014-5793(95)00254-7.
33. Shimakawa, G., Müller, P., Miyake, C., Krieger-Liszkay, A., and Sétif, P. (2024) Photo-oxidative damage of photosystem I by repetitive flashes and chilling stress in cucumber leaves, *Biochim. Biophys. Acta Bioenerg*, **1865**, 149490, https://doi.org/10.1016/j.bbabio.2024.149490.
34. Sonoike, K. (1996) Degradation of psaB gene product, the reaction center subunit of photosystem I, is caused during photoinhibition of photosystem I: possible involvement of active oxygen species, *Plant Sci.*, **115**, 157-164, https://doi.org/10.1016/0168-9452(96)04341-5.
35. Sonoike, K., Kamo, M., Hihara, Y., Hiyama, T., and Enami, I. (1997) The mechanism of the degradation of psaB gene product, one of the photosynthetic reaction center subunits of Photosystem I, upon photoinhibition, *Photosynth. Res.*, **53**, 55-63, https://doi.org/10.1023/A:1005852330671.
36. Sonoike, K. (2011) Photoinhibition of Photosystem I, *Physiol. Plant.*, **142**, 56-64, https://doi.org/10.1111/j.1399-3054.2010.01437.x.
37. Tiwari, A., Mamedov, F., Grieco, M., Suorsa, M., Jajoo, A., Styring, S., Tikkanen, M., and Aro, E.-M. (2016) Photodamage of iron-sulphur clusters in Photosystem I induces non-photochemical energy dissipation, *Nat. Plants*, **2**, 16035, https://doi.org/10.1038/nplants.2016.35.
38. Tiwari, A., Mamedov, F., Fitzpatrick, D., Gunell, S., Tikkanen, M., and Aro, E.-M. (2024) Differential FeS cluster photodamage plays a critical role in regulating excess electron flow through Photosystem I, *Nat. Plants*, **10**, 1592-1603, https://doi.org/10.1038/s41477-024-01780-2.
39. Furutani, R., Wada, S., Ifuku, K., Maekawa, S., and Miyake, C. (2023) Higher reduced state of Fe/S-signals, with the suppressed oxidation of P700, causes PSI inactivation in *Arabidopsis thaliana*, *Antioxidants*, **12**, 21, https://doi.org/10.3390/antiox12010021.
40. Subramanyam, R., Joly, D., Gauthier, A., Beauregard, M., and Carpentier, R. (2005) Protective effect of active oxygen scavengers on protein degradation and photochemical function in Photosystem I submembrane fractions during light stress, *FEBS J.*, **272**, 892-902, https://doi.org/10.1111/j.1742-4658.2004.04512.x.
41. Petrova, A. A., Boskhomdzhieva, B. K., Milanovsky, G. E., Koksharova, O. A., Mamedov, M. D., Cherepanov, D. A., and Semenov, A. Yu. (2017) Interaction of various types of Photosystem I complexes with exogenous electron acceptors, *Photosynth. Res.*, **133**, 175-184, https://doi.org/10.1007/s11120-017-0371-1.
42. Shi, Q., Sun, H., Timm, S., Zhang, S., and Huang, W. (2022) Photorespiration alleviates photoinhibition of Photosystem I under fluctuating light in tomato, *Plants*, **11**, 195, https://doi.org/10.3390/plants11020195.
43. Yamamoto, H., Takahashi, S., Badger, M. R., and Shikanai, T. (2016) Artificial remodelling of alternative electron flow by flavodiiron proteins in Arabidopsis, *Nat. Plants*, **2**, 16012, https://doi.org/10.1038/nplants.2016.12.
44. Tan, S.-L., Huang, X., Li, W.-Q., Zhang, S.-B., and Huang, W. (2021) Elevated CO2 concentration alters photosynthetic performances under fluctuating light in *Arabidopsis thaliana*, *Cells*, **10**, 2329, https://doi.org/10.3390/cells10092329.
45. Tikkanen, M., Mekala, N. R., and Aro, E.-M. (2014) Photosystem II photoinhibition-repair cycle protects Photosystem I from irreversible damage, *Biochim. Biophys. Acta Bioenerg.*, **1837**, 210-215, https://doi.org/10.1016/j.bbabio.2013.10.001.
46. Messant, M., Hani, U., Lai, T.-L., Wilson, A., Shimakawa, G., and Krieger-Liszkay, A. (2024) Plastid terminal oxidase (PTOX) protects photosystem I and not photosystem II against photoinhibition in *Arabidopsis thaliana* and *Marchantia polymorpha*, *Plant J.*, **117**, 669-678, https://doi.org/10.1111/tpj.16520.
47. Kozuleva, M. A., and Ivanov, B. N. (2023) Superoxide anion radical generation in photosynthetic electron transport chain, *Biochemistry (Moscow)*, **88**, 1045-1060, https://doi.org/10.1134/S0006297923080011.

48. Naydov, I., Kozuleva, M., Ivanov, B., Borisova-Mubarakshina, M., and Vilyanen, D. (2024) Pathways of oxygen-dependent oxidation of the plastoquinone pool in the dark after illumination, *Plants*, **13**, 3479, https://doi.org/10.3390/plants13243479.
49. Zhou, Q., Yamamoto, H., and Shikanai, T. (2022) Distinct contribution of two cyclic electron transport pathways to P700 oxidation, *Plant Physiol.*, **192**, 326-341, https://doi.org/10.1093/plphys/kiac557.
50. Kono, M., and Terashima, I. (2016) Elucidation of photoprotective mechanisms of PSI against fluctuating light photoinhibition, *Plant Cell Physiol.*, **57**, 1405-1414, https://doi.org/10.1093/pcp/pcw103.
51. Kramer, M., Rodriguez-Heredia, M., Saccon, F., Mosebach, L., Twachtmann, M., Krieger-Liszkay, A., Duffy, C., Knell, R. J., Finazzi, J., and Hanke, G. T. (2021) Regulation of photosynthetic electron flow on dark to light transition by ferredoxin:NADP(H) oxidoreductase interactions, *eLife*, **10**, e56088, https://doi.org/10.7554/eLife.56088.
52. Rodriguez-Heredia, M., Saccon, F., Wilson, S., Finazzi, G., Ruban, A. V., and Hanke, G. T. (2022) Protection of Photosystem I during sudden light stress depends on ferredoxin:NADP(H) reductase abundance and interactions, *Plant Physiol.*, **188**, 1028-1042, https://doi.org/10.1093/plphys/kiab550.
53. Tikhonov, A. N. (2024) The cytochrome b_6f complex: plastoquinol oxidation and regulation of electron transport in chloroplasts, *Photosynth. Res.*, **159**, 203-227, https://doi.org/10.1007/s11120-023-01034-w.
54. Kurisu, G., Zhang, H., Smith, J. L., and Cramer, W. A. (2003) Structure of the cytochrome b_6f complex of oxygenic photosynthesis: tuning the cavity, *Science*, **302**, 1009-1014, https://doi.org/10.1126/science.1090165.
55. Malone, L. A., Qian, P., Mayneord, G. E., Hitchcock, A., Farmer, D. A., Thompson, R. F., Swainsbury, D. J. K., Ranson, N. A., Hunter, C. N., and Johnson, M. P. (2019) Cryo-EM structure of the spinach cytochrome b_6f complex at 3.6 Å resolution, *Nature*, **575**, 535-539, https://doi.org/10.1038/s41586-019-1746-6.
56. Pintscher, S., Pietras, R., Mielecki, B., Szwalec, M., Wójcik-Augustyn, A., Indyka, P., Rawski, M., Koziej, L., Jaciuk, M., Ważny, G., Glatt, S., and Osyczka, A. (2024) Molecular basis of plastoquinone reduction in plant cytochrome b_6f, *Nat. Plants*, **10**, 1814-1825, https://doi.org/10.1038/s41477-024-01804-x.
57. Martinez, S. E., Huang, D., Ponomarev, M., Cramer, W. A., and Smith, J. L. (1996) The heme redox center of chloroplast cytochrome *f* is linked to a buried five-water chain, *Protein Sci.*, **5**, 1081-1092, https://doi.org/10.1002/pro.5560050610.
58. Ponamarev, M. V., and Cramer, W. A. (1998) Perturbation of the internal water chain in cytochrome *f* of oxygenic photosynthesis: loss of the concerted reduction of cytochromes *f* and b_6, *Biochemistry*, **37**, 17199-17208, https://doi.org/10.1021/bi981814j.
59. Sainz, G., Carrell, C. J., Ponamarev, M. V., Soriano, G. M., Cramer, W. A., and Smith, J. L. (2000) Interruption of the internal water chain of cytochrome *f* impairs photosynthetic function, *Biochemistry*, **39**, 9164-9173, https://doi.org/10.1021/bi0004596.
60. Hasan, S. S., Yamashita, E., Baniulis, D., and Cramer, W. A. (2013) Quinone-dependent proton transfer pathways in the photosynthetic cytochrome b_6f complex, *Proc. Natl. Acad. Sci. USA*, **110**, 4297-4302, https://doi.org/10.1073/pnas.1222248110.
61. Crofts, A. R., Hong, S., Wilson, C., Burton, R., Victoria, D., Harrison, C., and Schulten, K. (2013) The mechanism of ubihydroquinone oxidation at the Qo-site of the cytochrome bc1 complex, *Biochim. Biophys. Acta Bioenerg.*, **1827**, 1362-1377, https://doi.org/10.1016/j.bbabio.2013.01.009.
62. Tikhonov, A. N. (2014) The cytochrome b_6f complex at the crossroad of photosynthetic electron transport pathways, *Plant Physiol. Biochem.*, **81**, 163-183, https://doi.org/10.1016/j.plaphy.2013.12.011.
63. Zito, F., Finazzi, G., Joliot, P., and Wollman, F. A. (1998) Glu78, from the conserved PEWY sequence of subunit IV, has a key function in cytochrome b_6f turnover, *Biochemistry*, **37**, 10395-10403, https://doi.org/10.1021/bi980238o.
64. Szwalec, M., Bujnowicz, Ł., Sarewicz, M., and Osyczka, A. (2022) Unexpected heme redox potential values implicate an uphill step in cytochrome b_6f, *J. Phys. Chem. B*, **126**, 9771-9780, https://doi.org/10.1021/acs.jpcb.2c05729.
65. Hope, A. B. (1993) The chloroplast cytochrome bf complex A critical focus on function, *Biochim. Biophys. Acta Bioenerg.*, **1143**, 1-22, https://doi.org/10.1016/0005-2728(93)90210-7.
66. Ustynyuk, L. Y., and Tikhonov, A. N. (2022) Plastoquinol oxidation: rate-limiting stage in the electron transport chain of chloroplasts, *Biochemistry (Moscow)*, **87**, 1084-1097, https://doi.org/10.1134/S0006297922100029.
67. Sarewicz, M., Szwalec, M., Pintscher, S., Indyka, P., Rawski, M., Pietras, R., Mielecki, B., Koziej, Ł., Jaciuk, M., Glatt, S., and Osyczka, A. (2023) High-resolution cryo-EM structures of plant cytochrome b_6f at work, *Sci. Adv.*, **9**, eadd9688, https://doi.org/10.1126/sciadv.add9688.
68. Finazzi, G. (2002) Redox-coupled proton pumping activity in cytochrome b_6 *f*, as evidenced by the pH dependence of electron transfer in whole cells of *Chlamydomonas reinhardtii*, *Biochemistry*, **41**, 7475-7482, https://doi.org/10.1021/bi025714w.
69. Soriano, G. M., Guo, L.-W., de Vitry, C., Kallas, T., and Cramer, W. A. (2002) Electron transfer from the Rieske iron-sulfur Protein (ISP) to cytochrome *f in vitro*, *J. Biol. Chem.*, **277**, 41865-41871, https://doi.org/10.1074/jbc.M205772200.
70. Arantes, G. M. (2025) Redox-activated proton transfer through a redundant network in the Qo site

of cytochrome bc1, *J. Chem. Inf. Model*, **65**, 2660-2669, https://doi.org/10.1021/acs.jcim.4c02361.

71. Soriano, G. M., Ponamarev, M. V., Carrell, C. J., Xia, D., Smith, J. L., and Cramer, W. A. (1999) Comparison of the cytochrome bc1 complex with the anticipated structure of the cytochrome b_6f complex: De Plus Ça Change de Plus C'est la Même Chose, *J. Bioenerg. Biomembr.*, **31**, 201-214, https://doi.org/10.1023/A:1005463527752.
72. Rumberg, B., and Siggel, U. (1969) pH changes in the inner phase of the thylakoids during photosynthesis, *Naturwissenschaften*, **56**, 130-132, https://doi.org/10.1007/BF00601025.
73. Tikhonov, A. N., Khomutov, G. B., Ruuge, E. K., and Blumenfeld, L. A. (1981) Electron transport control in chloroplasts. Effects of photosynthetic control monitored by the intrathylakoid pH, *Biochim. Biophys. Acta Bioenerg.*, **637**, 321-333, https://doi.org/10.1016/0005-2728(81)90171-7.
74. Hope, A. B., Valente, P., and Matthews, D. B. (1994) Effects of pH on the kinetics of redox reactions in and around the cytochrome bf complex in an isolated system, *Photosynth. Res.*, **42**, 111-120, https://doi.org/10.1007/BF02187122.
75. Finazzi, G., and Rappaport, F. (1998) *In vivo* characterization of the electrochemical proton gradient generated in darkness in green algae and its kinetic effects on cytochrome b_6f turnover, *Biochemistry*, **37**, 9999-10005, https://doi.org/10.1021/bi980320j.
76. Tikhonov, A. N. (2015) Induction events and short-term regulation of electron transport in chloroplasts: an overview, *Photosynth. Res.*, **125**, 65-94, https://doi.org/10.1007/s11120-015-0094-0.
77. Finazzi, G., Minagawa, J., and Johnson, G. N. (2016) The cytochrome b_6f complex: a regulatory hub controlling electron flow and the dynamics of photosynthesis? in *Cytochrome Complexes: Evolution, Structures, Energy Transduction, and Signaling* (Cramer, W. A., and Kallas, T., eds) Springer Netherlands, pp. 437-452, https://doi.org/10.1007/978-94-017-7481-9_22.
78. Genty, B., and Harbinson, J. (1996) Regulation of light utilization for photosynthetic electron transport, in *Photosynthesis and the Environment*, (Baker, N. R., eds) Springer Netherlands, pp. 67-99, https://doi.org/10.1007/0-306-48135-9_3.
79. Kramer, D. M., Sacksteder, C. A., and Cruz, J. A. (1999) How acidic is the lumen? *Photosynth. Res.*, **60**, 151-163, https://doi.org/10.1023/A:1006212014787.
80. Cooley, J. W. (2013) Protein conformational changes involved in the cytochrome *bc*1 complex catalytic cycle, *Biochim. Biophys. Acta Bioenerg.*, **1827**, 1340-1345, https://doi.org/10.1016/j.bbabio.2013.07.007.
81. Schoepp, B., Brugna, M., Riedel, A., Nitschke, W., and Kramer, D. M. (1999) The Qo-site inhibitor DBMIB favours the proximal position of the chloroplast Rieske protein and induces a pK-shift of the redox-linked proton, *FEBS Lett.*, **450**, 245-250, https://doi.org/10.1016/s0014-5793(99)00511-6.
82. Roberts, A. G., Bowman, M. K., and Kramer, D. M. (2004) The inhibitor DBMIB provides insight into the functional architecture of the Qo site in the cytochrome b_6f complex, *Biochemistry.*, **43**, 7707-7716, https://doi.org/10.1021/bi049521f.
83. Vilyanen, D., Pavlov, I., Naydov, I., Ivanov, B., and Kozuleva, M. (2024) Peculiarities of DNP-INT and DBMIB as inhibitors of the photosynthetic electron transport, *Photosynth. Res.*, **161**, 79-92, https://doi.org/10.1007/s11120-023-01063-5.
84. Jahns, P., Graf, M., Munekage, Y., and Shikanai, T. (2002) Single point mutation in the Rieske iron-sulfur subunit of cytochrome b_6f leads to an altered pH dependence of plastoquinol oxidation in Arabidopsis, *FEBS Lett.*, **519**, 99-102, https://doi.org/10.1016/s0014-5793(02)02719-9.
85. Vilyanen, D., Naydov, I., Ivanov, B., Borisova-Mubarakshina, M., and Kozuleva, M. (2022) Inhibition of plastoquinol oxidation at the cytochrome b_6f complex by dinitrophenyl ether of iodonitrothymol (DNP-INT) depends on irradiance and H^+ uptake by thylakoid membranes, *Biochim. Biophys. Acta Bioenerg.*, **1863**, 148506, https://doi.org/10.1016/j.bbabio.2021.148506.
86. Ivanov, B. N. (1993) Stoichiometry of proton uptake by thylakoids during electron transport in chloroplasts, in *Photosynthesis: Photoreactions to Plant Productivity*, (Abrol, Y. P., Mohanty, P., and Govindjee, eds) Springer Netherlands, pp. 109-128, https://doi.org/10.1007/978-94-011-2708-0_4.
87. Schansker, G. (2022) Determining photosynthetic control, a probe for the balance between electron transport and Calvin-Benson cycle activity, with the DUAL-KLAS-NIR, *Photosynth. Res.*, **153**, 191-204, https://doi.org/10.1007/s11120-022-00934-7.
88. Joliot, P., and Johnson, G. N. (2011) Regulation of cyclic and linear electron flow in higher plants, *Proc. Natl. Acad. Sci. USA*, **108**, 13317-13322, https://doi.org/10.1073/pnas.1110189108.
89. Ott, T., Clarke, J., Birks, K., and Johnson, G. (1999) Regulation of the photosynthetic electron transport chain, *Planta*, **209**, 250-258, https://doi.org/10.1007/s004250050629.
90. Harbinson, J., and Hedley, C. L. (1989) The kinetics of $P\text{-}700^+$ reduction in leaves: a novel in situ probe of thylakoid functioning, *Plant Cell Environ.*, **12**, 357-369, https://doi.org/10.1111/j.1365-3040.1989.tb01952.x.
91. Laisk, A., and Oja, V. (1994) Range of photosynthetic control of postillumination $P700^+$ reduction rate in sunflower leaves, *Photosynth. Res.*, **39**, 39-50, https://doi.org/10.1007/BF00027141.
92. Kanazawa, A., Ostendorf, E., Kohzuma, K., Hoh, D., Strand, D. D., Sato-Cruz, M., Savage, L., Cruz, J. A.,

Fisher, N., Froehlich, J. E., and Kramer, D. M. (2017) Chloroplast ATP synthase modulation of the thylakoid proton motive force: implications for Photosystem I and Photosystem II photoprotection, *Front. Plant Sci.*, **8**, 719, https://doi.org/10.3389/fpls.2017.00719.

93. Takagi, D., Amako, K., Hashiguchi, M., Fukaki, H., Ishizaki, K., Goh, T., Fukao, Y., Sano, R., Kurata, T., Demura, T., Sawa, S., and Miyake, C. (2017) Chloroplastic ATP synthase builds up a proton motive force preventing production of reactive oxygen species in photosystem I, *Plant J.*, **91**, 306-324, https://doi.org/10.1111/tpj.13566.
94. Wang, C., and Shikanai, T. (2019) Modification of activity of the thylakoid H^+/K^+ antiporter KEA3 disturbs ΔpH-dependent regulation of photosynthesis, *Plant Physiol.*, **181**, 762-773, https://doi.org/10.1104/pp.19.00766.
95. Wu, G., Ortiz-Flores, G., Ortiz-Lopez, A., and Ort, D. R. (2007) A point mutation in atpC1 raises the redox potential of the Arabidopsis chloroplast ATP synthase γ-subunit regulatory disulfide above the range of thioredoxin modulation, *JBC*, **282**, 36782-36789, https://doi.org/10.1074/jbc.M707007200.
96. Degen, G. E., Jackson, P. J., Proctor, M. S., Zoulias, N., Casson, S. A., and Johnson, M. P. (2023) High cyclic electron transfer via the PGR5 pathway in the absence of photosynthetic control, *Plant Physiol.*, **192**, 370-386, https://doi.org/10.1093/plphys/kiad084.
97. Nandha, B., Finazzi, G., Joliot, P., Hald, S., and Johnson, G. N. (2007) The role of PGR5 in the redox poising of photosynthetic electron transport, *Biochim. Biophys. Acta Bioenerg.*, **1767**, 1252-1259, https://doi.org/10.1016/j.bbabio.2007.07.007.
98. Penzler, J.-F., Naranjo, B., Walz, S., Marino, G., Kleine, T., and Leister, D. (2024) A pgr5 suppressor screen uncovers two distinct suppression mechanisms and links cytochrome b_6f complex stability to PGR5, *Plant Cell*, **36**, 4245-4266, https://doi.org/10.1093/plcell/koae098.
99. Degen, G. E., Pastorelli, F., and Johnson, M. P. (2024) Proton Gradient Regulation 5 is required to avoid photosynthetic oscillations during light transitions, *J. Exp. Bot.*, **75**, 947-961, https://doi.org/10.1093/jxb/erad428.
100. Avenson, T. J., Cruz, J. A., Kanazawa, A., and Kramer, D. M. (2005) Regulating the proton budget of higher plant photosynthesis, *Proc. Natl. Acad. Sci. USA*, **102**, 9709-9713, https://doi.org/10.1073/pnas.0503952102.
101. Kozuleva, M. A., Petrova, A. A., Mamedov, M. D., Semenov, A. Yu., and Ivanov, B. N. (2014) O2 reduction by Photosystem I involves phylloquinone under steady-state illumination, *FEBS Lett.*, **588**, 4364-4368, https://doi.org/10.1016/j.febslet.2014.10.003.
102. Kozuleva, M., Goss, T., Twachtmann, M., Rudi, K., Trapka, J., Selinski, J., Ivanov, B., Garapati, P., Steinhoff, H., Hase, T., Scheibe, R., Klare, J. P., and Hanke, G. T. (2016) Ferredoxin:NADP(H) oxidoreductase abundance and location influences redox poise and stress tolerance, *Plant Physiol.*, **172**, 1480-1493, https://doi.org/10.1104/pp.16.01084.
103. Taylor, R. M., Sallans, L., Frankel, L. K., and Bricker, T. M. (2018) Natively oxidized amino acid residues in the spinach cytochrome b_6f complex, *Photosynth. Res.*, **137**, 141-151, https://doi.org/10.1007/s11120-018-0485-0.
104. Sang, M., Ma, F., Xie, J., Chen, X.-B., Wang, K.-B., Qin, X.-C., Wang, W.-D., Zhao, J.-Q., Li, L.-B., Zhang, J.-P., and Kuang, T.-Y. (2010) High-light induced singlet oxygen formation in cytochrome b_6f complex from *Bryopsis corticulans* as detected by EPR spectroscopy, *Biophys. Chem.*, **146**, 7-12, https://doi.org/10.1016/j.bpc.2009.09.012.
105. Baniulis, D., Hasan, S. S., Stofleth, J. T., and Cramer, W. A. (2013) Mechanism of enhanced superoxide production in the cytochrome b_6f complex of oxygenic photosynthesis, *Biochem.*, **52**, 8975-8983, https://doi.org/10.1021/bi4013534.
106. Sarewicz, M., Bujnowicz, Ł., Bhaduri, S., Singh, S. K., Cramer, W. A., and Osyczka, A. (2017) Metastable radical state, nonreactive with oxygen, is inherent to catalysis by respiratory and photosynthetic cytochromes bc_1/b_6f, *Proc. Natl. Acad. Sci. USA*, **114**, 1323-1328, https://doi.org/10.1073/pnas.1618840114.
107. Borisova-Mubarakshina, M. M., Naydov, I. A., and Ivanov, B. N. (2018) Oxidation of the plastoquinone pool in chloroplast thylakoid membranes by superoxide anion radicals, *FEBS Lett.*, **592**, 3221-3228, https://doi.org/10.1002/1873-3468.13237.
108. Kozuleva, M. (2022) Recent advances in the understanding of superoxide anion radical formation in the photosynthetic electron transport chain, *Acta Physiol. Plant.*, **44**, 92, https://doi.org/10.1007/s11738-022-03428-0.
109. Pesaresi, P., Pribil, M., Wunder, T., and Leister, D. (2011) Dynamics of reversible protein phosphorylation in thylakoids of flowering plants: The roles of STN7, STN8 and TAP38, *Biochim. Biophys. Acta Bioenerg.*, **1807**, 887-896, https://doi.org/10.1016/j.bbabio.2010.08.002.
110. Schönberg, A., Rödiger, A., Mehwald, W., Galonska, J., Christ, G., Helm, S., Thieme, D., Majovsky, P., Hoehenwarter, W., and Baginsky, S. (2017) Identification of STN7/STN8 kinase targets reveals connections between electron transport, metabolism and gene expression, *Plant J.*, **90**, 1176-1186, https://doi.org/10.1111/tpj.13536.
111. Grieco, M., Tikkanen, M., Paakkarinen, V., Kangasjärvi, S., and Aro, E.-M. (2012) Steady-state phosphorylation of light-harvesting complex II proteins preserves Photosystem I under fluctuating white light, *Plant Physiol.*, **160**, 1896-1910, https://doi.org/10.1104/pp.112.206466.

112. Shapiguzov, A., Chai, X., Fucile, G., Longoni, P., Zhang, L., and Rochaix, J.-D. (2016) Activation of the Stt7/STN7 kinase through dynamic interactions with the cytochrome b_6f complex, *Plant Physiol.*, **171**, 82-92, https://doi.org/10.1104/pp.15.01893.
113. Hoh, D., Froehlich, J. E., and Kramer, D. M. (2024) Redox regulation in chloroplast thylakoid lumen: the pmf changes everything, again, *Plant Cell Environ.*, **47**, 2749-2765, https://doi.org/10.1111/pce.14789.
114. Balashov, N. V., Borisova-Mubarakshina, M. M., and Vetoshkina, D. V. (2025) The influence of hydrogen peroxide on the redistribution of antenna complexes between photosystems in higher plants, *Biochemistry (Moscow)*, **90**, 943-955, https://doi.org/10.1134/S0006297925601443.
115. Fernyhough, P., Horton, P., and Foyer, C. (1984) Regulation of light harvesting chlorophyll a/b binding protein (LHCP) phosphorylation in intact maize mesophyll chloroplasts, in *Advances in Photosynthesis Research*, Springer, Dordrecht, pp. 299-302, https://doi.org/10.1007/978-94-017-4973-2_68.
116. Vershubskii, A. V., Trubitsin, B. V., Priklonskii, V. I., and Tikhonov, A. N. (2017) Lateral heterogeneity of the proton potential along the thylakoid membranes of chloroplasts, *Biochim. Biophys. Acta Bioenerg.*, **1859**, 388-401, https://doi.org/10.1016/j.bbamem.2016.11.016.