VNU Journal of Science: Natural Sciences and Technology, Vol. 42, No. 2 (2026) 66-83

VNU Journal of Science: Natural Sciences and Technology

Journal homepage: https://js.vnu.edu.vn/NST

Original Article

Photosynthesis Regulation by State Transitions

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Received 31st March 2025
Revised 05th Febuary 2026; Accepted 10th March 2026

Abstract: Climate change poses serious threats to agriculture worldwide, including drought, salinity, and unfavorable light conditions that affect plant growth. Photosynthesis, a physiological process of great significance for crop yield, occurs in chloroplasts and includes light-dependent reactions that produce ATP and NADPH, followed by the Calvin cycle that fixes CO_2 into glucose. This process is highly sensitive to abiotic stresses. Crops have evolved critical regulatory mechanisms to modulate photosynthesis, enabling adaptation to adverse environmental conditions. One such regulatory mechanism is state transitions, where plants adjust the excitation energy balance between Photosystem I (PSI) and Photosystem II (PSII) in response to fluctuating light conditions. This allows plants to optimize the utilization of absorbed light energy by dynamically reallocating energy between the two photosystems as needed. State transitions enable plants to acclimate to varying light environments, maintain efficient conversion of light energy into photosynthetic products, and protect the photosynthetic apparatus from photodamage. This mechanism enhances photosynthetic efficiency, improves crop productivity, and bolsters environmental stress resilience. Elucidating the molecular basis of state transitions offers promising biotechnological strategies to engineer crops with enhanced stress tolerance and productivity under suboptimal environmental conditions.

Keywords: Climate change, crop productivity, photosynthesis, state transitions.

1. Introduction

The human population is growing rapidly, leading to increasing standards of living. Additionally, agricultural systems are affected by changes in land use, economy, and climate. Shortly, the demand for food and energy will be enormous. The stocks of unsustainable resources are diminishing, and our only possibility of preserving peace and welfare is to significantly reduce the consumption of limited resources and enhance the production of sustainable resources. Food, fossil fuels, and all other organic energy and matter in general are based on converting light energy into chemical form through the photosynthesis of plants. In this context, we must maximize the capacity to utilize photosynthetic production sustainably for the benefit of humanity. Moreover,

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https://doi.org/10.25073/2588-1140/vnunst.5885

photosynthesis regulation is the best defense against climate change. One of the main goals must be the improvement of more efficient and more tolerant photosynthetic organisms that can utilize solar energy with maximum yield and minimal negative environmental impact. Therefore, a deep understanding of the structure, function, and regulation mechanisms of the photosynthetic machinery is crucial for maintaining photosynthetic activity in the face of environmental change. Improvements in the capture and conversion of light energy have been a central focus of many researchers worldwide. This review aims to summarize the current understanding of the structure and function of the photosynthetic apparatus, particularly photosynthetic efficiency regulation by state transitions, as studied mainly in the unicellular green alga *Chlamydomonas reinhardtii* (C. *reinhardtii*) and the model plant *Arabidopsis thaliana*, and explore potential future applications in agriculture and biotechnology.

1.1. The Photosynthesis Apparatus, from Structure to Function

Light absorption and energy conversion occur within specific and highly organized structures found in the thylakoid membrane, the photosynthetic apparatus. This apparatus consists of four principal protein supercomplexes: two photosystems (PSI and PSII), the cytochrome b6f (Cyt b6f), and the ATP synthase complex. These four complexes perform the processes of light harvesting, electron transport, and photophosphorylation, catalyzing the conversion of light energy into chemical energy in the form of ATP and NADPH (see Figure 1). Changes in one of these complexes due to specific environmental conditions can lead to modifications in photosynthetic activity, ultimately affecting plant productivity.

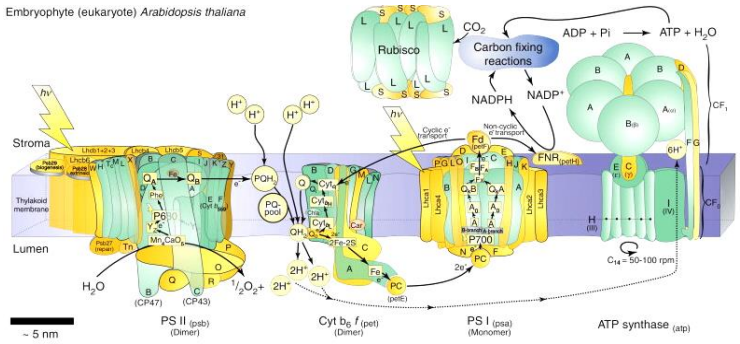

Figure. 1. Photosynthetic apparatus organisation and the electron transport chain in a higher plant exemplified by *Arabidopsis thaliana* [1]. Key protein complexes in the chloroplast of *Arabidopsis thaliana* include photosystem II (PSII), cytochrome b6f (Cyt b6f), photosystem I (PSI), ATP synthase, and Rubisco. Subunit names are abbreviated with single letters, omitting complex-specific prefixes: "psa" for PSI, "psb" for PSII, "pet" for cytochrome b6f and related carriers, "atp" for ATP synthase, and "rbc" for Rubisco. Subunits encoded by the chloroplast genome are shown in green; nuclear-encoded subunits are in yellow [1].

1.2. Photosystems: Light Energy Absorption, Excitation Energy Transfer and Conversion into Chemical Energy

Localized in thylakoid membranes, photosystems are highly organized supercomplexes composed of integral and peripheral membrane proteins. Photosystems (PSs) contain antenna complexes, which bind most of the pigments and are therefore responsible for a large portion of light capture, and the core complex, which contains the reaction center (RC) where charge separation and electron transport take place. In oxygenic photosynthetic organisms, photosystems are

classified into two types based on differences in the core complex: Type I (PSI) and Type II (PSII), which contain iron–sulfur complexes and quinone–protein complexes, respectively. In plants, they have distinct thylakoid localizations: PSII is mainly found within the stacked grana membranes, while PSI is located in the unstacked stroma lamellae and external grana.

Photosystem I

The role of PSI is the conversion of light energy into chemical energy contained in the high-energy compound NADPH. This is catalyzed by the preliminary reduction of ferredoxin by PSI. Ferredoxin is a redox mobile protein localized in the stroma that reduces NADP+ in a reaction catalyzed by ferredoxin-NADP-reductase. Electrons to PSI are provided by the redox lumenal protein plastocyanin. In plants and green algae, PSI is constituted by a core complex and peripheral Lhca (Light-harvesting complex of photosystem I, type a) antennas located on one side of the complex (Figure 2A). The PSI core complex is formed by two main subunits, PsaA and PsaB, which are coded by the plastid genes *psaA* and *psaB*, respectively; 13 other protein subunits, 2 phylloquinones (A1), and 3 Fe_4S_4 clusters (FX, FA, and FB) are found in the core. The PSI reaction center contains 6 chlorophyll molecules organized in three dimers bound with core proteins; two Chls form a special pair (P700) with an absorption maximum at 700 nm, and they are the electron donor for charge separation; two other Chls are the primary electron acceptor (A0); two others localized between P700 and A0, named A, are accessory chlorophylls (Figure 2B). These six chlorophyll molecules are separated into three pairs linked with PsaA and PsaB on the two sides (Figure 2B).

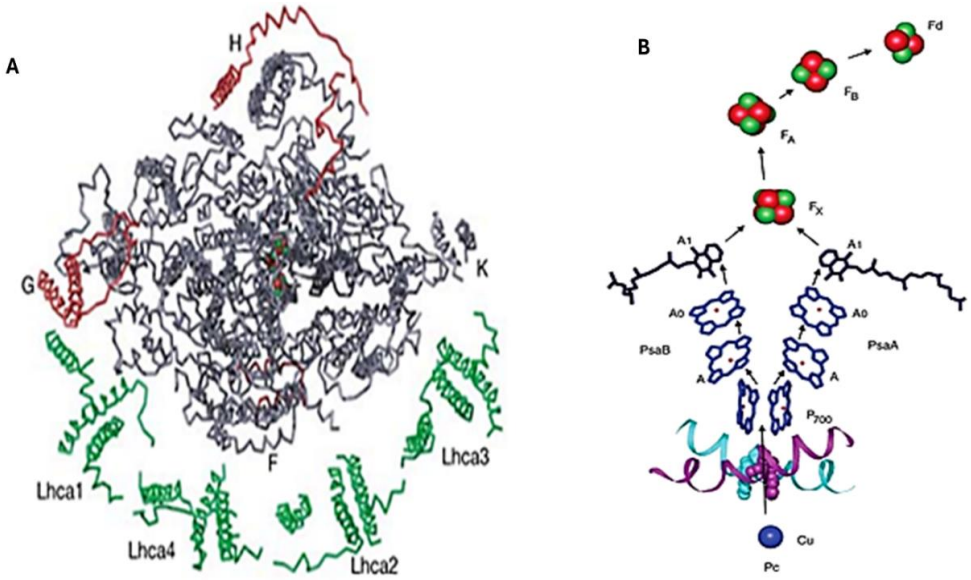

Figure 2. A) The structure of photosystem I (PSI) is depicted based on the crystal structure reported in [2]. The plant PSI structural model, resolved at 4.4 Å, is shown as a Cα backbone. The four light-harvesting proteins (Lhca1–4) are highlighted in green. Novel structural features within the reaction center (core), which are absent in the cyanobacterial equivalent, are colored red, while conserved elements of the reaction center are shown in grey. B) A structural model illustrating the pathway of light-driven electron transport from plastocyanin to ferredoxin in photosystem I is presented. Chlorophyll molecules are shown in blue, quinones in black, the copper atom of plastocyanin (PC) in blue, and the iron (red spheres) and sulfur (green spheres) atoms of the three Fe_4S_4 clusters as well as the Fe_2S_2 cluster of ferredoxins (Fd) are depicted. Additionally, two tryptophan residues, represented as light-blue and light-pink space-filling models, which may participate in electron transfer from PC to P700, are shown within their secondary structural context [3].

The PSI peripheral antenna, called LHCI (or Lhca) (Light-Harvesting Complex of PSI), is composed of four different light-harvesting membrane proteins with polypeptides of about 20–24 kDa. These proteins are encoded by the nuclear genes *lhca1, lhca2, lhca3,* and *lhca4*. LHCI binds chlorophyll a and b, violaxanthin, and lutein for light energy capture, excitation energy transfer to the core complex, and photoprotection [5, 7].

PSI binds approximately 173 chlorophyll molecules in total [8]. About 20 chlorophylls are located between LHCI and the core complex; these intermediary pigments are involved in energy transfer from the LHCI peripheral antenna to the PSI reaction center [5]. Moreover, some phosphorylated trimeric LHCII (Light-Harvesting Complex of PSII) binds to PSI in a phenomenon called "state transitions" which allows energy balancing between the two photosystems [9, 10].

Photosystem II

Like PSI, PSII is composed of two moieties: the core complex containing the reaction center and a peripheral antenna system.

The reaction center of PSII (RCII) consists of two proteins, D1 (PsbA) and D2 (PsbD), encoded by the plastidial genes *psbA* and *psbD*, respectively. It coordinates six chlorophyll molecules, two pheophytins (PhD1, PhD2), two β-carotene molecules (CarD1, CarD2), two plastoquinones (QA, QB), and other essential cofactors for the electron transport chain [11, 12]. PSII is primarily found in a dimeric form, with each monomer containing at least 27 to 28 subunits per core complex [13].

Two of the six chlorophylls in RCII form the special pair (P680), where charge separation occurs, acting as the primary electron donor. Two other chlorophyll molecules function as "accessory" chlorophylls, similar to those in the PSI core, though their roles remain unclear. The remaining two chlorophylls are located near the internal antenna complexes CP43 and CP47, acting as linker chlorophylls to facilitate energy transfer from the internal antennas to the special pair, P680 [12]. These linker chlorophylls are also involved in protecting the PSII reaction center from photoinduced damage [14].

Additionally, two β-carotene molecules may participate in a putative electron transfer mechanism when the Mn(4)Ca cluster is damaged, protecting the reaction center by transferring and/or quenching ChlZD1 triplet states and quenching singlet oxygen [15, 16]. The two plastoquinone molecules serve as the terminal electron acceptors of PSII on the stromal side of the complex: QA is stably bound to PSII through hydrogen bonds with the main-chain amide group of D2, while QB is the exchangeable quinone. When QB is doubly reduced by QA, it binds two stromal protons (forming PQH2) and is released into the membrane.

The two pheophytin molecules, associated with D1, D2, and P680, act as primary electron acceptors, receiving electrons from P680 and transferring them to QA. Cytochrome b559 can be directly reduced by pheophytin and may prevent photoinhibitory damage to PSII by decreasing the probability of charge recombination between the primary radical pair $P680^{+}$–$Pheo^{-}$, which would otherwise lead to the formation of the P680 triplet state [17]. Furthermore, cytochrome c550 stabilizes the binding between Cl^{-} and Ca^{2+} ions in the oxygen-evolving complex and protects the Mn(4)Ca cluster from reduction under unfavorable conditions [18].

The core complex contains two internal antenna proteins, CP47 (PsbB) and CP43 (PsbC), which bind approximately 16 and 14 chlorophyll molecules, respectively, as well as some β-carotene molecules. These β-carotene molecules may be involved in quenching chlorophyll triplets and singlet oxygen produced by P680 triplets due to the recombination of $P680^{+}$ and $Pheo^{-}$ [12, 20].

Most of the energy utilized by PSII is absorbed by the PSII light-harvesting complex (Lhcb proteins), encoded by homologous nuclear genes belonging to the *lhc* gene family [21].

Three monomeric proteins - CP29, CP26, and CP24 - products of the *lhcb4, lhcb5,* and

lhcb6 genes, respectively, are arranged near CP43 (CP26) and CP47 (CP24, CP29). These proteins participate in light capture, energy transfer from the peripheral antenna to the core complex, and photoprotection by dissipating excess energy as heat. LHCII, the major light-harvesting complex of PSII, is a heterotrimeric complex composed of the *Lhcb1*, *Lhcb2*, and *Lhcb3* isoforms [21]. Up to four LHCII trimers per monomeric core are present in the membranes. Lhcb proteins bind numerous chlorophyll and carotenoid molecules (Figure 3) [19, 22].

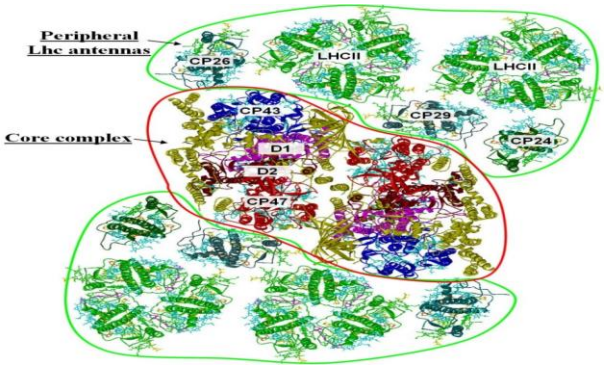

Figure 3. Structural model of the PSII–LHCII supercomplex [4]. D1, D2, CP43, and CP47 are core subunits of PSII, highlighted in blue, cyan, salmon, and pink, respectively, while the extrinsic subunit PsbO is shown in yellow. The Lhcb proteins, including trimeric LHCII and monomeric Lhcb, are represented in dark green and light green, respectively. Lhcb3 and the minor antenna complexes-CP24, CP26, and CP29-are schematically outlined in dark green, light blue, magenta, and orange, respectively. Green and pink asterisks mark areas with similarly high densities of trimeric and monomeric LHCII, respectively. Pink tripod-shaped lines denote regions excluded from staining corresponding to the LHCII trimers.

In particular, each LHCII monomeric subunit coordinates eight chlorophyll a, six chlorophyll b, and four xanthophyll molecules [23]. Carotenoid molecules such as lutein, violaxanthin, and neoxanthin play essential roles not only in light absorption [24] but also in photoprotection by facilitating the thermal dissipation of excess energy through a process known as Non-Photochemical Quenching (NPQ), extensively discussed by numerous authors [25–27]. Additionally, carotenoids contribute to chlorophyll triplet quenching [28] and reactive oxygen species (ROS) scavenging [29]. NPQ relies on the xanthophyll cycle and the PsbS protein, a grana-localized protein whose exact position remains under investigation [30-34].

LHCII antenna trimers associate with the PSII core at different positions: strongly associated trimers (S-trimers) are located between CP26 and CP29, moderately associated trimers (M-trimers) interact with CP29 and CP24, while loosely bound trimers (L-trimers) have an unknown position [13, 18]. The largest PSII supercomplex purified to date is the C2S2M2 supercomplex, consisting of a dimeric core with S-LHCII and M-LHCII on both sides of PSII [19]. Structural analyses indicate that Lhcb antennas, which bind most of the PSII chlorophylls (~80%), are organized to ensure highly efficient energy transfer to the reaction center, with approximately 90-95% of absorbed energy reaching P680 [35, 36].

1.3. Oxygen Evolution Complex and Water Photooxidation Process

The source of oxygen on earth is the water oxidation at the level of the Oxygen Evolving Complex (OEC) in PSII. The OEC includes four Mn ions located at the corners of a tetrahedron and a Ca^{2+} ion located in the center region, forming a Mn4Ca cluster with each metal ion in this cluster connecting to another Mn ion by three-μ-oxo bridges [5]. This Mn4Ca

cluster is connected to D1 through the tyrosine Z (Yz), CP43 and at least three nuclear-encoded proteins, PsbO(33kDa), PsbP (23 kDa) and PsbQ (16 kDa) participating in its stabilization. Although the exact oxygen evolution mechanism is still debated, it is known that oxygen evolution is done by a 5-step oxidation of two water molecules through five S oxidation states of the manganese cofactor from S0 to S4, and light provides the energy for the reaction [6]. The whole process is described as $2\ H_2O \rightarrow (S0 \rightarrow S1 \rightarrow S2 \rightarrow S3 \rightarrow S4 \rightarrow S0) \rightarrow O_2 + 4\ H^+$. In each passage from S0 to S4, one electron and one proton are produced and the S4 state decomposes spontaneously to release O_2 and come back the S0 state [3]. Electrons are transferred from the Mn4Ca cluster through the redox-active Tyrosine (Y_Z) to the oxidized $P680^+$ [7] and integrate into the electron transfer chain of PSII (Fig. 1). Released protons are used for ATP synthesis by the chloroplast ATPase complex.

1.4. The Cytochrome b6f Complex in the Electron Transport Chain of Oxygenic Photosynthesis

The cytochrome b_6f complex is localized in a central position in the photosynthetic electron transport chain, between PSII and PSI. It assures the electron transfer connection between the two photosystems by oxidizing plastoquinol coming from PSII (PQH_2) and reducing plastocyanin, which transfer electrons to PSI. At the same time, it catalyses the transfer of protons from the chloroplast stroma across the thylakoid membrane into the lumen, thus contributing to the creation of a trans-membrane ΔpH. The cytochrome b_6f complex is a dimer and each monomer includes redox-active subunits as cytochrome *f*, cytochrome *b* and the Rieske-type *2Fe-2S* protein containing prosthetic groups and other small subunits [8]. In the linear electron transport chain, plastoquinol (PQH_2) transfers two electrons from oxidized PSII to Cyt b_6f in two pathways: one to the Rieske protein (FeS_R) and one other to Cyt *b*. The cytochrome *f* accepts an electron from (FeS_R) and then transfers this electron to plastocyanin (PC), a soluble protein that can reach a lumenal site of PSI and reduce the oxidized $P700^+$.

The Cyt *b* transfers an electron to a second Cyt *b* that reduces plastoquinone on a stromal site. This quinone cycle allow picking up two more protons from the stroma for each electron transferred in the linear electron chain (PSII-Cyt-PSI). Two total protons are transferred into the lumen for each electron transferred from PSII to PSI, and these protons creates the electrochemical potential driving ATP formation in chloroplasts (see Figure 4).

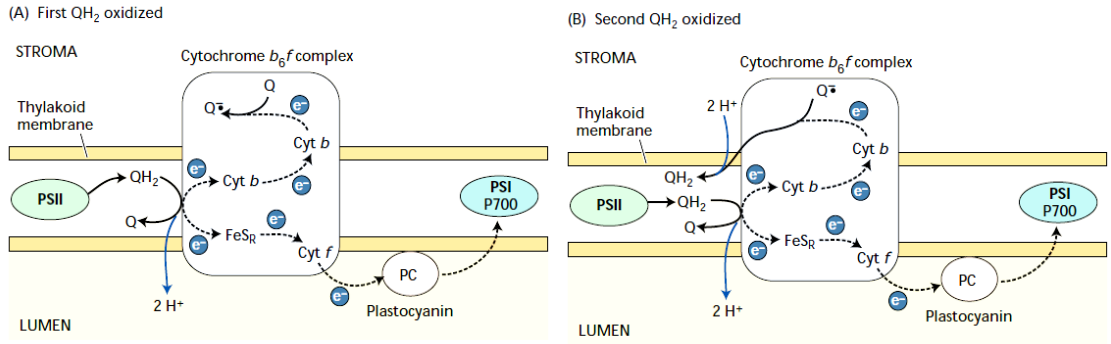

Figure 4. The electron transfer in cytochrome b_6f complex [9].

The Figure 4 illustrates the mechanism of electron and proton transfer within the cytochrome b6f complex. This complex is composed of two b-type cytochromes (Cyt b), a c-type cytochrome (Cyt c, also known as cytochrome f), a Rieske iron-sulfur protein (FeSR), and two quinone oxidation-reduction sites. (A) In the noncyclic (linear) pathway, a

plastohydroquinone (PQH2) molecule generated by photosystem II is oxidized on the lumenal side of the complex. During this process, PQH2 donates two electrons: one to the Rieske Fe–S protein and one to a b-type cytochrome, while simultaneously releasing two protons into the lumen. The electron transferred to FeSR is passed on to cytochrome f (Cyt f) and then to plastocyanin (PC), which ultimately reduces P700 in photosystem I (PSI). Meanwhile, the reduced b-type cytochrome transfers an electron to another b-type cytochrome, which then reduces plastoquinone (PQ) to plastosemiquinone (PQ*–). (B) In the cyclic pathway, a second PQH2 molecule is oxidized. One electron moves from FeSR to plastocyanin and then to P700, while the other passes through the two b-type cytochromes to reduce plastosemiquinone back to plastohydroquinone, concurrently taking up two protons from the stroma. In total, for every two electrons delivered to P700, four protons are translocated across the membrane.

1.5. Photosynthetic Electron Transfer in Thylakoid Membranes

Light energy absorbed by pigment molecules in the antenna systems are transferred to the reaction centre of the respective photosystem by a resonance mechanism that allows the transfer of excitation energy from Chl to Chl. The pigment absorbs light energy in the visible region from about 350 to 700 nm. The excitation energy is used to excite the chlorophyll special pairs P680 (1.2V) and P700 (0.49V) into the strong reductant $P680^*$ (-0.75V) and $P700^*$ (-1.2V). The special pairs are the primary electron donors that reduce in a very short time (few ps) pheophytin (in PSII) and two chlorophyll molecules (Ao in PSI). These electron transfers from excited chlorophylls in the reaction centers to primary acceptors are the primary photochemical events that convert excitation energy into chemical energy. After charge separation, $P680^+$ and $P700^+$ become oxidants. $P680^+$ is a very strong oxidant that is able to oxidize water by keeping electrons from the OEC through the tyrosine Yz. $P700^+$ keeps electrons from the lumenal protein plastocyanin.

After charge separation in PSII, the electron transport chain starts. The complete linear electron transport involves several protein and molecules that finally allow the global redox reaction of water oxidation and $NADP^+$ reduction in NADPH:

$H_2O \rightarrow [Mn4CaCl] \rightarrow Yz \rightarrow P680 \rightarrow Pheo \rightarrow Q_A \rightarrow Q_B/Q_BH2 \rightarrow cyt b_6f \rightarrow PC \rightarrow PSI \rightarrow Fd \rightarrow NADP^+$

Half-times of electron transfer respective:

1ms →10µs→1µs→ps→200ps→10-100µs→1-10ms→1ms→10-100µs→100ns to µs→100µs [3].

Reduction of Q_A by $Pheo^-$ occurs in 250–300 ps, and electron transfer from Q_A to Q_B has a half-time of approximately 100 µs [3]. Electron transfer from Cyt b_6f to PC to PSI is described above. Electrons from PC are then used to reduce the photooxidised primary electron donor of PSI, the $P700^+$. In the PSI reaction centre, where charge separation takes place, the electron from the primary acceptor Ao passes a series of membrane bound iron-sulfur Fe4S4 clusters (F_X, F_A, and F_B) till the soluble ferredoxin (Fd). The electron transfer from Fx to Fd requires about 500 ns. Under the action of the flavoprotein ferredoxin–$NADP^+$ reductase (FNR), ferredoxin reduces $NADP^+$ to NADPH completing the sequence of the "linear electron transport" started from the water photooxidation reaction. This process is schematized in Fig. 1. From PSI, a second electron transfer pathway exists, where ferredoxin does not reduce $NADP^+$ into NADPH, but transfers its electron directly to Cyt f of the Cyt b_6f complex, and ultimately to the PSI reaction centre through PC. This "cyclic electron transport" increases proton transfer into the thylakoid lumen and therefore ΔpH. Lumenal protons (produced from water splitting reaction and electron transfer at the level of Cyt b_6f) create a pH gradient, which drives ATP synthesis to produce ATP via ATP synthase from ADP and inorganic phosphate (Pi). In addition, low pH in the lumen is necessary to regulate an important

photoprotective mechanism, the ^{1}Chl* de–excitation via NPQ mechanism by PsbS protonation and activation of the xanthophyll cycle in high light condition.

2. Photosynthetic Efficiency Regulation by State Transitions

In the photosynthetic apparatus organization of plants, the two photosystems (PSII and PSI) are localized in two distinct regions of thylakoids. Whereas PSII is mostly confined to the grana regions, PSI is mainly located in the stroma lamellae and the electron transfer between photosystems is mediated by the electron carriers plastoquinone, the cytochrome b_6f and the plastocyanin. Each photosystem contains an antenna system that is responsible for light harvesting and a reaction centre that performs the charge separation and converts excitation energy transferred from antenna complexes into chemical energy. Even if both photosystems bind Chls *a*, *b* and carotenoids, the absorption spectra of PSI and PSII are slightly different, mainly due to the different antenna proteins that coordinate different amounts of Chl *a* and *b* and modulate the specific absorption of the bound pigments. PSI is enriched in chlorophyll *a* absorbing in the far-red region (≥700 nm), whereas PSII is enriched in chlorophyll *b*, whose maximum absorption is at shorter wavelengths in the red region (~650 nm). The two photosystems work in series, therefore, their excitation energy must be balanced in order to optimize photosynthetic performance. However, in certain cases the absorbed light may result in an unbalanced excitation of the two photosystems. Thus, the overall efficiency of the resulting electron flow decreases [41]. In this case, the plant activates a short-term adaptation mediated mainly by the reversible phosphorylation of the major light-harvesting complex of photosystem II (LHCII), which causes its migration from PSII to PSI in a phenomenon called "state transitions" (Figure 5).

State transitions had been discovered more than forty years ago by Bonaventura and Myers [42]. It has been detected as a redistribution of excitation energy between PSII and PSI by changes in light absorption cross-section and excitation energy spillover between the two photosystems depending on light quality and quantity. It was also observed in nature by Tavish [43]. This redistribution is known to be reversible and exhibits a short-time kinetics of induction/relaxation of about 15 to 30 min [44, 45]. When the conditions causing this unbalanced photosystem excitation persist for a longer time (hours to days), other photosynthetic mechanisms are activated, such as photosystem stoichiometry adjustment. This kind of acclimatization is denoted as a long-term response [46-48]. The STN7 (State Transition 7) kinase protein, which is indispensable for state transitions (see below), is also involved in the shift between short-term and long-term acclimation to fluctuating light conditions [48].

State transitions induce the relocation of a part of LHCII between the two photosystems, a phenomenon driven by LHCII phosphorylation/dephosphorylation during light variations [41, 49-52]. Under illumination conditions favorable for PSII excitation (red/blue light), LHCII phosphorylation is activated, and a portion of phosphorylated LHCII migrates to PSI. This decreases PSII absorption while conversely increasing PSI absorption.

On the contrary, under light conditions that preferentially excite PSI (far-red light), LHCII dephosphorylation is activated, promoting LHCII migration back to PSII. "State II" denotes an adaptive state under light preferentially absorbed by PSII (characterized by high LHCII phosphorylation and migration to PSI), while "State I" denotes an adaptive state under light preferentially absorbed by PSI.

Mobile LHCII between PSII and PSI has been found to represent about 80% of total LHCII in the green alga *C. reinhardtii* [53], but only 20–25% in higher plants [49]. In both higher plants and *Chlamydomonas*, imbalances in excitation energy distribution between the two photosystems are sensed by the redox state of the intersystem electron transfer components,

particularly the plastoquinone (PQ) pool and the Cyt b6f complex in the thylakoid membranes [50, 52, 54, 55]. When one photosystem is excited more than the other, it affects the redox state of the PQ pool [52, 56].

Previous studies indicated that LHCII is phosphorylated when the PQ pool is reduced, meaning PSII has higher activity compared to PSI [50, 52, 56, 57]. A reduced PQ pool induces the State II transition by activating an LHCII kinase, leading to P-LHCII displacement from PSII to PSI, thereby increasing PSI light absorption and decreasing PSII light absorption. Conversely, when the PQ pool is oxidized, the LHCII kinase is inactivated, and LHCII is dephosphorylated by an LHCII phosphatase. This promotes the State I transition, where dephosphorylated LHCII returns from PSI to PSII, redistributing absorbed energy in favor of PSII.

The binding of PQH_2 to the Qo-site of Cyt b6f activates the LHCII kinase(s) [50]. It has also been proposed that LHCII phosphorylation is regulated by the chloroplast ferredoxin-thioredoxin system [58, 59].

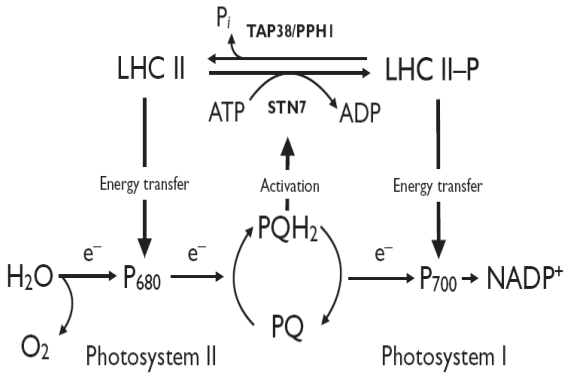

Figure 5. State transitions regulation, from Puthiyaveetil [10].

The Figure 5 illustrates the light reactions of photosynthesis as the electron transport from H_2O to $NADP^+$ through two photosystems linked by the cytochrome b6f complex, which oxidizes plastoquinol (PQH_2) to plastoquinone (PQ). When light is primarily absorbed by photosystem II (PSII), the light-harvesting complex II (LHC II) kinase STN7 detects PQH_2 and triggers the transition to state 2. During this transition, phosphorylation of LHC II reduces the light-harvesting antenna size of PSII while increasing that of photosystem I (PSI). Conversely, when the PQ pool becomes mostly oxidized, such as under light conditions favoring excitation of PSI, the phospho-LHC II phosphatase TAP38/PPH1-operating independently of light and redox signals-catalyzes dephosphorylation of phospho-LHC II. This results in a decrease in the antenna size of PSI and an increase in that of PSII.

State transitions are therefore based on the reversible phosphorylation of LHCII, and in particular of the Lhcb1 and Lhcb2 isoforms [11]. In *Arabidopsis* an LHCII kinase protein family, called TAKs (Thylakoid-Associated Kinases), has been isolated from thylakoid membranes by Snyders, Kohorn, [12, 13] . In the green alga *C. reinhardtii*, a thylakoid-associated serine/threonine protein kinase named STT7 (state transition-deficient mutant 7) was identified by Depege et al., [14] The deficiency of this protein in the *Chlamydomonas stt7* mutant leads to the non-phosphorylation of LHCII and lock thylakoids in State I [14]. Similarly, inactivation of the homologous *stn7* gene of in *Arabidopsis* blocks state transitions [15]. Additional biochemical and genetic analysis of the *stt7/stn7* mutants demonstrated their LHCII kinase role [16–18]. Four phosphorylated residues have been found near the C-terminal end of the STN7 kinase in the case of Arabidopsis [19], but not in the orthologous STT7 kinase of *C. reinhardtii* [14]. Topological studies suggest that STT7 has a single transmembrane domain with the N-terminal localized in lumen and the kinase domain exposed in stroma [16].

Co-immunoprecipitation analyses [16] supported the direct interaction of STT7 with LHCII, with the Rieske iron–sulphur protein of the Cyt b_6f complex and with PSI during state transitions [16].

Furthermore, the STN8 kinase protein, a paralogue of STN7, was found to be responsible for phosphorylation of PSII core proteins as D1, D2, CP43, and PsbH in

Arabidopsis [18, 20], and the STL1 protein for phosphorylation of CP43 and D2 in *C. reinhardtii* [14]. However, since PSII core phosphorylation is not selectively activated by PSII-specific light as for LHCII [21], a direct activation of STN8 from the Qo site as STN7 is unlikely [10] and PSII core phosphorylation seems independent of Cyt b_6f [22]. The PSII core phosphorylation was shown to be unaffected in the absence of STN7 [18]. The phosphorylation of the D1 protein has been suggested to have a key function during the PSII repair cycle [53-56], but the significance of D2 and CP43 phosphorylation is not clear. However, some authors proposed that phosphorylation of both minor Lhcb proteins and PSII core subunits may facilitate the dissociation of peripheral antenna via coulomb repulsion [27, 28]. In the green alga *C. reinhardtii* it has been found that the minor antenna protein CP29 is also phosphorylated and play an important role in state transitions [35, 39, 57, 58. Although Tokotsu and co-workers [29] proposed that CP26 is not necessary for state transitions in *Chlamydomonas*, however this protein and Lhcbm5, a particular isoform of LHCII, were found linked with PSI in State II [31, 32]. The minor antenna proteins CP26 and CP29 localize in an intermediate position between the PSII core and the LHCII antenna, and their migration from PSII to PSI indicates a large reorganization of PSII during state transitions in *Chlamydomonas*, which is not the case in plants [30, 31, 33].

A second enzyme, a P-LHCII phosphatase, is required to promote the transition from State II to State I [34, 35]. The thylakoid-associated TAP38 phosphatase (Thylakoid-Associated Phosphatase of 38 kDa), also called PPH1, has recently been found to be responsible for the dephosphorylation of the LHCII protein [36, 37]. In *Arabidopsis* plants inactivated for PPH1 function, P-LHCII is not dephosphorylated, and thylakoid are blocked in State II and enriched in PSI-LHCII complex [36, 37]. *In vitro* dephosphorylation assay clearly indicated that PPH1 can directly dephosphorylate P-LHCII [36]. This phosphatase is required for the dephosphorylation of the major trimeric Lhcb1 and Lhcb2 proteins, but it is not able to dephosphorylate the PSII core proteins D1, D2 and CP43 [36, 37]. Dephosphorylation of PSII core proteins was indeed not affected in PPH1 knock-out mutants [36], suggesting that other chloroplast phosphatase is responsible for dephosphorylation of these core proteins [10]. It is proposed that the dephosphorylation of PSII core proteins is catalyzed by a PP2A-type protein [38], while this PPH1 protein is a PP2C-type protein phosphatase [37].

The phosphorylation/dephosphorylation action of STN7 and PPH1 are opposed and they need to be finely regulated. The observation that PPH1 overexpressor plants (increasing 20-fold the amount of PPH1 protein) still show LHCII phosphorylation [36], indicates that LHCII dephosphorylation by PPH1 does not inhibit STN7 phosphorylation and a regulation mechanism of these phosphorylation/dephosphorylation may be independent. As discussed before, LHCII phosphorylation is triggered by a reduced plastoquinone pool state, while the thylakoid phosphatase proteins are known to be not redox sensitive and kinetically heterogeneous [39].

LHCII is a trimeric complex composed of a combination of three highly homologous Lhcb isoforms, Lhcb1-3, encoded by a large multigene family [40]. In plants, three different types of LHCII trimers are named S, M and L, based on their strong (S), moderate (M) and loose (L) association with the PSII complex. The positions of the S and M trimers are well defined in the supercomplex [41], while the locations of loosely bound trimers are still debated. The absence of the Lhcb3 isoform in stroma membranes under State II conditions [42] suggested that trimer M, which is particularly enriched in this subunit [43, 44], is not involved in state transitions. Other results would indicate that trimer M is involved instead [45, 46] and that Lhcb3 plays a role in modulating the rate of state transitions [46]. However, the investigations on trimers

associated to PSI in State II indicate that mobile trimers have a different composition as compared to trimers S and M, thus it is proposed that S and M trimers are (almost) not involved in state transitions, while the peripheral trimers loosely bound to PSII are the mobile pool of LHCII during state transitions in plants [33].

When a trimeric LHCII binds to PSI in State II, it transfers excitation energy very efficiently [33], thus increasing PSI absorption cross section of about 21%. At least 50% of PSI forms a PSI-LHCII complex in State II conditions, therefore decreasing excitation of PSII reaction centers. The very recent Roberto Bassi groups inactivated the entire Lhcb1 and Lhcb2 gene clades in *Arabidopsis thaliana* and then reintroduced either the wild-type isoforms Lhcb1.3 and Lhcb2.1, or versions lacking the N-terminal phosphorylatable residues, which are believed to mediate state transitions. While substituting Lhcb2.1 at Thr-40 prevented the formation of the PSI-LHCI-LHCII complex, replacing Thr-38 in Lhcb1.3 did not affect the formation of this supercomplex, nor did it influence the magnitude or timing of PSII fluorescence quenching during the state 1 to state 2 transition [47]. This indiced that phosphorylation of Lhcb2 at Thr-40 by STN7 accounted for approximately 60% of PSII fluorescence quenching during state transitions. In contrast, the Thr-38 phosphosite in Lhcb1.3 was not necessary for the formation of the PSI-LHCI-LHCII supercomplex or for the re-equilibration of the plastoquinone redox state. The Lhcb2 phosphomutant retained about 40% residual fluorescence quenching, suggesting that an as-yet unidentified, STN7-dependent component of state transitions, which is unrelated to Lhcb2 Thr-40 phosphorylation and the formation of the PSI-LHCI-LHCII supercomplex, plays a role in balancing PSI/PSII excitation pressure when plastoquinone is over-reduced [47].

It has been found that the block of state transitions in the *Arabidopsis stn7* mutant results in a more reduced plastoquinone pool [15]. Moreover growth of plant is impaired under fluctuating light for quality and quantity [14, 15, 24], indicating a clear role of STN7 and state transitions in response to light changes. However, the lack of the STN7 kinase causes relative over excitation of PSII under low light but not under high light, leading to disturbed maintenance of an efficient electron flow under fluctuating light intensities [24]. Indeed, state transitions are considered a low light short-term regulation [48], while at high light other photosynthetic regulation seems more important, such as excitation energy dissipation as heat NPQ [55, 71]. NPQ controls the energy use efficiency at the level of the PSII antenna system in relation to light intensity [50, 51], thereby leading to the redox balance between light reactions and downstream electron acceptors, whereas the STN7 kinase-dependent LHCII protein phosphorylation balances the excitation between PSII and PSI. In conditions of low light exciting preferentially PSII, NPQ is low and, in the absence of the STN7 kinase, most of the energy absorbed by LHCII is transferred to PSII leading to overexcitation of PSII relatively to PSI. This leads to over-reduction of the electron transfer chain and less photochemistry in PSII, which finally causes a decreased growth of the *stn7* mutant [24]. In high light conditions, NPQ induces a large thermal dissipation of the absorbed energy at the level of PSII antennas, which reduces the surcharge for PSII reaction centre, thus the energy redistribution by movement of LHCII to PSI become less important [24, 49]. Indeed, it is proposed that the STN7 kinase function is synchronized with the PsbS and xanthophylls cycle dependent NPQ [52]. On the contrary, the absence of PPH1 results in the constitutive presence of the PSI-LHCII complex (thus in a constitutive increase of the PSI antenna size). Interestingly, it was found the *pph1* mutants have a particular and unexpected growth phenotype in low light. Indeed, it has been reported that LHCII hyperphosphorylation due to inactivation of PPH1 could improve the photosynthetic performance and plant growth under low light conditions [36].

In our previous work, we produced the *pph1/npq4* double mutant and characterized some photosynthetic, growth, and reproduction properties in comparison with wild-type and single mutant plants under both high and low light conditions. The results obtained indicated that under high light, the *pph1* mutant maintains good photoprotection ability, while *npq4* plants show greater susceptibility to photodamage. The *pph1/npq4* double mutant exhibited resistance to high light stress like that of the single *npq4* mutant. Under low light conditions, the *pph1/npq4* mutant showed a significant increase in growth and flowering compared to the single mutants and wild-type plants [53]. This suggests that optimizing photosynthesis to improve crop growth and productivity may be possible (at least in controlled low light conditions) through the regulation of state transitions and modification of NPQ [53]. This work complements earlier genetic and physiological studies of PPH1's role in LHCII dephosphorylation and state transitions [36] but uniquely links genetic mutants with growth and reproduction phenotypes under different light intensities. It bridges molecular mechanisms to whole-plant physiology, filling a vital gap in understanding PPH1's practical impact on plant fitness.

Phospho-proteomic studies have reinforced that phosphorylation is a widespread regulatory mechanism for protein function in chloroplasts, including the enzymes involved in starch metabolism. Beyond photosynthetic machinery, protein kinases such as STN7 and STN8, as well as their corresponding phosphatases like PPH1), along with several other kinases and phosphatases, have now been localized to the chloroplast [54]. Mapping phosphorylation sites on proteins of chloroplast starch metabolism to illustrate the potential and largely unknown role of protein phosphorylation in the regulation of starch biology [54]. Study state transitions to understand how plants adapt to different light conditions.

In addition to the above analytical research results, over the past five years, international research has focused on elucidating the physiological roles, molecular regulators, and adaptive significance of state transitions in photosynthesis across plants and algae. Molecular mechanisms: The critical roles of STN7 kinase (in higher plants) and STT7 kinase (in green algae) in LHCII phosphorylation and regulating antenna migration have been precisely identifie [55-57]. Besides phosphorylation, N-terminal acetylation of LHCII proteins is necessary for efficient antenna migration [58]. Physiological function: State transitions is confirmed as a key factor balancing energy distribution between photosystems to enhance CO_2 assimilation efficiency and maintain photosynthetic performance under fluctuating light conditions [55, 56, 59]. Novel Discoveries: Advanced techniques, such as spatially resolved imaging, mutant analysis, and fluctuating light models, have quantitatively demonstrated the effects of ST on photosynthesis rates, biomass accumulation, and stress tolerance conditions [55-57]. Artificial manipulation of state transitions has opened new avenues for improving photosynthetic efficiency [60]. Demonstrated artificial regulation of state transitions using synthetic polymers and electrochemical luminescence techniques, which significantly enhanced light harvesting efficiency and ATP/NADPH generation compared to controls, suggesting new strategies for increasing plant productivity [60].

2.1. Evaluation of the Reliability, Novelty, and Significance of the Cited Studies

Reliability: The body of literature referenced is overwhelmingly composed of peer-reviewed, high-impact publications from reputable journals such as Nature, Science, Plant Cell, Biochimica et Biophysica Acta, and Proceedings of the National Academy of Sciences (see reference list). Many foundational studies by Allen JF and colleagues [11, 21, 61-68] have been extensively cited and experimentally validated over decades, establishing a solid and reproducible framework for understanding protein phosphorylation and state transitions. Structural studies [69–71]

provide high-resolution crystallographic data that underpin mechanistic models, adding robustness to the biochemical and physiological interpretations. The proteomic and phosphoproteomic analyses [19, 72] employ advanced mass spectrometry techniques, enhancing the reliability of protein kinase/phosphatase identification and their roles in state transitions. In summary, the methodologies used across these studies are rigorous, reproducible, and supported by complementary approaches (biochemistry, genetics, structural biology, and biophysics), ensuring high reliability of the findings.

Novelty: While many of the cited works represent seminal discoveries dating back several decades [34, 73], the list also includes cutting-edge research that advances the field: The elucidation of the STN7 kinase's dual role in both short-term state transitions and long-term photosystem stoichiometry adjustment [15, 74] represents a significant conceptual advance, linking rapid acclimation to gene expression regulation. Genome editing studies [47] provide novel insights into the molecular components of state transitions beyond classical models, including quenching components independent of PSI-LHCI-LHCII supercomplex formation. The integration of redox signaling with thioredoxin systems regulating LHCII phosphorylation [75, 76] offers innovative perspectives on the crosstalk between photosynthetic electron transport and regulatory phosphorylation. Recent structural and functional characterizations of photosystem supercomplexes [4, 33] reveal dynamic rearrangements during state transitions, deepening understanding of the molecular basis of energy redistribution. Thus, the collection balances foundational knowledge with recent breakthroughs, reflecting both the historical development and current frontiers of the field.

Significance: These studies profoundly advance our comprehension of how plants dynamically regulate light harvesting, optimizing photosynthetic efficiency and preventing photodamage under fluctuating environmental conditions. These profound insights extend their reach across various domains. In fundamental biology, they precisely clarify the molecular mechanisms governing energy balancing between Photosystem I and Photosystem II, a core principle of photosynthetic regulation. For agricultural and biotechnological applications, insights into state transitions and related acclimation mechanisms lay a solid foundation for engineering crops with improved tolerance to fluctuating light conditions, thereby enhancing yield stability. This is achieved through a deeper understanding of complex kinase/phosphatase signaling networks and pigment–protein interactions, which opens promising avenues for synthetic biology approaches aimed at optimizing photosynthetic performance [53, 77, 78]. From ecological relevance, the dynamic flexibility elucidated by this research provides key explanations for plant adaptation to natural light fluctuations, contributing significantly to ecosystem productivity and resilience.

2.2. Comparative Analysis and Discussion of Consensus and Contradictions Consensus

Consensus: State transitions are widely acknowledged as a rapid and dynamic acclimation mechanism that enables photosynthetic organisms to balance excitation energy distribution between Photosystem II (PSII) and Photosystem I (PSI). This balancing act optimizes photosynthetic efficiency and mitigates photodamage under fluctuating light conditions. The underlying molecular process primarily involves the reversible phosphorylation of light-harvesting complex II (LHCII), facilitating its migration between PSII and PSI. In green algae such as *C. reinhardtii*, state transitions regulate up to approximately 80% of LHCII complexes, reflecting a flexible and extensive antenna remodeling. Conversely, in higher plants like *Arabidopsis thaliana*, roughly 15% of LHCII is involved [59, 79], indicating a more restrained yet functionally significant role. Recent insights have refined this picture: In C. reinhardtii, phosphorylated LHCII detached from PSII does not fully

reattach to PSI; instead, it undergoes quenching to protect PSII from overexcitation. This indicates a direct photoprotective role of state transitions, especially relevant when NPQ mechanisms are delayed or less efficient [79]. In higher plants, rapid NPQ mediated via the PsbS protein acts synergistically with state transitions during high light stress, underscoring an integrated protective network Cyanobacteria present a mechanistically distinct yet functionally analogous adaptation. Their phycobilisome antenna systems mediate state transitions differently, but fluorescence lifetime imaging microscopy studies reveal uniform PSII quenching during state II despite heterogeneous PSI/PSII distribution, highlighting a conserved strategy for excitation balance [80]. At the systems level, consensus maintains that state transitions are a broadly conserved photoprotective mechanism complementing others such as the xanthophyll cycle and NPQ [81]. However, interesting evolutionary exceptions emerge; for example, Bryopsidales macroalgae and kleptoplastic sea slugs lack canonical state transitions and the xanthophyll cycle, relying instead on alternative pigment- and lipid-based photoprotection strategies [81]. These findings emphasize evolutionary diversity in photoprotective approaches.

Contradictions and unresolved aspects: Despite broad acceptance of state transitions as a ubiquitous energy-balancing and photoprotective mechanism, recent evidence challenges its universality. In green algae, a substantial photoprotective role for state transitions is well-supported [81], whereas in higher plants, this function appears more auxiliary and context dependent [53]. Novel research shows that Bryopsidales macroalgae completely lack state transitions [81]. This finding is particularly notable because these algae also do not employ other widespread photoprotection mechanisms such as the xanthophyll cycle or energy-dependent dissipation of excess light. Moreover, this deficiency extends to the kleptoplasts-stolen chloroplasts-within Sacoglossa sea slugs that consume these macroalgae. The absence of state transition capability in kleptoplasts results from structural remodeling, notably enforced chloroplast sphericity during incorporation into the animal host, facilitating prolonged retention [81]. This profound functional loss in both the original algae and their kleptoplasts offers a critical perspective on the necessity and prevalence of state transitions. It further invites exploration of alternative, yet uncharacterized, photoprotective strategies in these unique biological systems. Molecular diversity: While LHCII phosphorylation by STN7 kinase is firmly established as the canonical trigger for state transitions-inducing lateral migration of phosphorylated LHCII from PSII-rich grana stacks to PSI-enriched stromal lamellae-the regulatory complexity extends beyond phosphorylation alone. Recent findings reveal that loss of N-terminal acetylation impairs LHCII mobility and antenna migration, highlighting a multilayered regulatory network modulating state transition [58]. However, the universality of these molecular modifications across diverse taxa, and the hierarchical relationships among phosphorylation, acetylation, and potentially other posttranslational modifications, remain open areas of active investigation.

2.3. Limitations of Current Studies and Future Research Directions

Limitations of current studies: Limited field studies and natural fluctuating light experiments limit generalizability to agricultural and ecological contexts. Technical challenges in time- and space-resolved quantification of state transitions levels contribute to data inconsistency across labs. Molecular complexity remains to be fully decoded, especially for interacting kinases/phosphatases and other regulatory proteins. Application of genome editing to modulate state transitions is still nascent, with few translational outcomes.

Future research directions: Conduct more in situ and field experiments under natural fluctuating light regimes to evaluate the agronomic significance of state transitions.

Employ integrative genomics, proteomics, and phosphoproteomics to map the comprehensive regulatory network controlling state transitions. Combine biotechnology, genome editing (e.g., CRISPR), and novel materials for synthetic control of state transitions to enhance crop photosynthetic productivity, as proposed by [53, 77, 78]. Conduct cross-taxa comparative studies spanning cyanobacteria, algae, and higher plants to understand evolutionary diversification of state transitions mechanisms.

Author Contribution Statements

K. T. T. H wrote the manuscript in consultation with S.C and C.R.

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