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REVIEW

Photosystems under high light stress: throwing light on mechanism and adaptation

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Abstract

High light stress decreases the photosynthetic rate in plants due to photooxidative damage to photosynthetic apparatus, photoinhibition of PSII, and/or damage to PSI. The dissipation of excess energy by nonphotochemical quenching and degradation of the D1 protein of PSII and its repair cycle help against photooxidative damage. Light stress also activates stress-responsive nuclear genes through the accumulation of phosphonucleotide-3'-phosphoadenosine-5'-phosphate, methylerythritol cyclodiphosphate, and reactive oxygen species which comprise the chloroplast retrograde signaling pathway. Additionally, hormones, such as abscisic acid, cytokinin, brassinosteroids, and gibberellins, play a role in acclimation to light fluctuations. Several alternate electron flow mechanisms, which offset the excess of electrons, include activation of plastid or plastoquinol terminal oxidase, cytochrome b_6/f complex, cyclic electron flow through PSI, Mehler ascorbate peroxidase pathway or water–water cycle, mitochondrial alternative oxidase pathway, and photorespiration. In this review, we provided insights into high light stress-mediated damage to photosynthetic apparatus and strategies to mitigate the damage by decreasing antennae size, enhancing NPQ through the introduction of mutants, expression of algal proteins to improve photosynthetic rates and engineering ATP synthase.

Keywords: light stress; nonphotochemical quenching; photodamage; photosystem; reactive oxygen species; signaling.

Highlights

- High light (HL) stress-induced photoinhibition decreases the photosynthetic rate
- Nonphotochemical quenching and alternative electron flow are crucial for survival under HL stress
- HL damage is offset by chloroplast retrograde signaling and hormonal induction of antioxidant enzymes

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Abbreviations: ABA – abscisic acid; AOX – mitochondrial alternative oxidase; CEF – cyclic electron flow through PSI; CK – cytokinin; COX – cyanide-sensitive cytochrome *c* oxidase; EX1 – executor 1; Fd – ferredoxin; FLVs or FDPs – flavodiiron proteins; FNR – Fd:NADP(H) oxidoreductase; GA – gibberellins: HL – high light; JA – jasmonic acid; LHCSRs – light-harvesting complex stress-related proteins; m6A – N6-methyladenosine; MEcPP – methylerythritol cyclodiphosphate; MEP – methylerythritol phosphate; NPQ – nonphotochemical quenching; OEC – oxygen-evolving complex; OPDA – 12-oxo-phytodienoic acid; OXI1 – oxidative signal inducible 1; PAP/SAL1 – phosphonucleotide-3'-phosphoadenosine-5'-phosphate; PsbS – PSII subunit S; PTOX – plastoquinol terminal oxidase; PUB 4 – plant U-box 4; q_E – energy-dependent quenching; q_I – photoinhibitory quenching; q_T – photosynthetic state transition; ROS – reactive oxygen species; SOD – superoxide dismutase; WWC – water–water cycle; Yz^+ – oxidized tyrosine z; β-C – β-cyclocitral.
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Introduction

Light fluctuations affect the photosynthetic process in plants considerably (Morales and Kaiser 2020). The photosynthetic process comprises a series of reactions that are initiated with the excitation of chlorophyll in light-dependent reactions that culminate with the reduction of CO_2 in a 'light-independent' or dark reaction. The light reactions take place in the thylakoids of the chloroplast where electron transfer is carried out through a series of electron donors and acceptors present in the functional units known as photosystem (PS) I and II, thereby converting the harvested light energy to ATP and NADPH. Environmental variation in light regimes causes light to become a stress factor (Fiorucci and Fankhauser 2017) where low light intensity becomes insufficient to excite the chlorophyll molecules and high light (HL) intensity causes photoinhibition, photooxidation, photoinactivation, solarisation, photolability, and photodynamic reactions (Powles 1984). The photooxidative damage due to excess light energy is a consequence of the accumulation of multiple reactive oxygen species (ROS) generated due to an overflow of electrons in the photosystems of the light reactions (Tikkanen *et al.* 2014). HL stress causes the reaction centers to become light-saturated resulting in proteolytic degradation of D1 that can be repaired by newly synthesized D1 protein due to its property of high turnover (Tyystjärvi and Aro 1996). On the contrary, excess of D1 breakdown over repair causes photoinhibition of PSII (Murata *et al.* 2012). This inherent vulnerability of PSII to photon flux has a protective role towards PSI, as the degradation of damaged PSI protein and its subsequent replacement is a time and energy-intensive process. The damage from PSII to PSI is passed on irreversibly when PSI-linked electron acceptors lose the capacity to cope with the redox pressure (Kudoh and Sonoike 2002). The accumulation of electrons in PSI produces reactive oxygen radicals such as singlet oxygen and superoxide ions causing photooxidative stress (Munekage *et al.* 2002, Suorsa *et al.* 2012, Takagi *et al.* 2016).

Plants possess adaptive responses on different time scales to adjust to the damage caused by HL stress. The short-term response that occurs in a matter of seconds or minutes involves nonphotochemical quenching (NPQ). It is dependent on various factors such as the pH of the thylakoid lumen and the accumulation of zeaxanthin following its conversion from violaxanthin (Dietz 2015). The long-term response is associated with the change in gene expression of photosynthetic proteins such as plastid-encoded PSII (*psbA*) and PSI (*psaA*/*psaB*) core subunits, controlled by the redox state of the plastoquinone pool, increased PSII reaction center, cytochrome b_6/f complex, and ATP synthase in a reaction time of hours and days (Pfannschmidt *et al*. 1999, Spetea *et al.* 2014, Schuster *et al.* 2020). HL also increases the biosynthesis of certain hormones such as abscisic acid (ABA) and jasmonic acid (JA) while suppressing auxin and cytokinin synthesis (Suzuki *et al.* 2013, Dietz 2015). In addition to the above-mentioned adaptive mechanisms, the damage due to excess excitation pressure may be offset by various alternative mechanisms such as reversible phosphorylation of light-harvesting complexes (LHC), nonphotochemical chlorophyll fluorescence quenching (NPQ), activation of plastid or plastoquinol terminal oxidase (PTOX), cytochrome b_6/f complex, cyclic electron flow through PSI (CEF), Mehler ascorbate peroxidase (MAP) pathway or water–water cycle (WWC) and mitochondrial alternative oxidase pathway (AOX) (Mekala *et al.* 2015, Huang *et al.* 2019, Bolte *et al.* 2020, Sun *et al.* 2021). This review entails the chain of events initiated in response to HL stress and the metabolic adaptations for photoprotection to sustain yield under damaging light regimes. We also suggest potential approaches for improving photosynthetic efficiency under HL stress conditions.

High light stress-induced photoinhibition of photosynthetic capacity

The upper leaves of plants exposed to direct sunlight can dissipate nearly 75% of the absorbed light energy as heat which would otherwise lead to the formation of chlorophyll triplets and ROS (Friedland *et al.* 2019, Wu *et al.* 2021). In contrast, the shade plants or plants growing in the understorey of tropical forests are deprived of light. In both cases, intense illumination or HL conditions through the canopy gaps can disturb the redox homeostasis of the photosynthetic electron transport chain by creating an imbalance between the harvested light energy and the capacity to deal with excited electrons (Gollan and Aro 2020). It can lead to a loss in the efficiency of energy conversion and reduce the photosynthetic capacity through the process of photoinhibition.

Photoinhibition limits the photosynthetic activity either due to photoinactivation or photodamage (Huang *et al.* 2016, Li *et al.* 2018). In the twin processes of photoinactivation and photodamage, the former involves no alteration in the chemical structure of photosystems while the latter causes alteration in the chemical structure which is usually seen in both PSI and PSII, although PSII remains as the dominant site (Vass 2012, Li *et al.* 2018). Photoinhibition may be caused either by visible or UV light and the inhibitory effect increases with decreasing wavelength through blue to UV light (Sarvikas *et al.* 2006).

Photoinhibition is further classified as dynamic and chronic depending on its ability to revert to normal efficiency upon removal of stress conditions. Dynamic photoinhibition is caused by the diversion of absorbed light energy towards photoprotective heat dissipation where quantum yield decreases but the maximum photosynthetic rate remains unchanged. This decrease is often transient and quantum yield can return to its initial higher value when photon flux density decreases below the saturation levels. In contrast, chronic photoinhibition is relatively long-lasting and persists for weeks or months due to continuous exposure to high levels of excess light that damages the photosynthetic system and decreases both instantaneous quantum yields and maximum photosynthetic rate. This happens when light stress condition persists due to the inability of photoprotection mechanisms. The effect of chronic photoinhibition is

a consequence of loss of activity at the Mn cluster and damage to the D1 protein from the reaction center of PSII (Werner *et al.* 2002). The degradation of the photo-damaged D1 protein is affected by membrane-bound proteases and stromal proteases. The 33-kDa subunit of the oxygen-evolving complex (OEC) in PSII regulates the formation of cross-linked D1 protein during donor-side photoinhibition. The efficient turnover of the D1 protein is contributed by various proteases and protein components in chloroplasts, thus maintaining the quality of the PSII.

Photodamage to PSII by visible and UV light

Photodamage refers to irreversible damage to the structure of PSII that leads to its inactivation (Li *et al.* 2018). Both the UV-B and visible spectrums can damage the PSII complex at Mn clusters of water oxidation resulting in the modification of the water-oxidizing complex and the release of ROS (Vass 2012). UV absorption by Mn (III) and Mn (IV) ions is the primary sensitizer of UV-induced damage of the water-oxidizing machinery (Szilárd *et al.* 2007). The damage of the catalytic Mn cluster results in the inhibition of electron transfer to Tyr-Z^+ and P_{680}^+, and the reaction center protein (Larkum *et al.* 2001). The UV-B exposure induces modification or loss in the function of the Q_A and Q_B quinone electron acceptors (Vass *et al.* 1999, 2005). In the quinone pool, the UV-B radiations have a stronger effect on Q_B due to the specific destruction of the reduced quinone by the UV-B light or structural changes in the Q_B binding site (Vass *et al.* 1999, Dobrikova *et al.* 2013). The ROS generated by UV leads to the degradation of photosynthetic pigments, Rubisco, lipids, and amino acids as well as complex enzymes of the photosynthetic apparatus (Kataria *et al.* 2014, Czégény *et al.* 2016).

The visible-light-induced damage results from the modification of Q_A and Q_B acceptors in addition to their effect on Mn clusters and ROS production (Vass *et al.* 1992). The visible-light-induced damage to the PSII in plants is a result of modifications to the Q_A and Q_B acceptors, as well as the impact on Mn clusters and the production of ROS. Mechanisms of photoinhibition were grouped into two categories by Zavafer and Mancilla (2021): (*1*) excessive excitation-dependent photodamage and (*2*) excessive excitation-independent photodamage. These mechanisms have been explained in the following sections.

Excess excitation-dependent photodamage: Photo-damage occurs when the energy absorbed by the PSII complex is higher than the energy utilized in photochemistry or dissipated in photoprotection (Vass 2012). The PSII complex consists of two functional sides, *i.e.*, the acceptor and donor side, and their role in photodamage is discussed.

Acceptor-side limitation: The long-lived reduced quinone A ($Q^*_A^-$) can be produced in PSII due to the slow transfer of electrons from $Q^*_A^-$ to Q_B or $Q^*_B^-$ or interruption caused because of a vacant Q_B site. In this condition, $Q^*_A^-$ and $Q^*_B^-$ can combine with the redox states (S2 and S3) of the water-oxidizing complex and can form $S2Q^*_A^-$, $S3Q^*_A^-$, and $S3Q^*_B^-$ (Messinger and Renger 2008, Muh and Zouni 2011). These states may generate ROS which can react with components of the thylakoid membranes.

Donor-side limitation: Photoinactivation due to donor-side limitation may occur due to the inability of providing electrons at the rate of withdrawal of electrons from P_{680}. This may cause a prolonged build-up of oxygen radicals on the donor side which may result in photodamage (Andersson and Styring 1991, Aro *et al.* 1993). Blubaugh *et al.* (1991) proposed that the event of impairment of electron flow between Tyrosine Z and P_{680} occurs first, followed by a loss of oxidized Tyrosine Z (Yz^+) formation which is attributed to direct damage of tyrosyl residue or amino acids in the immediate vicinity. The impairment of electron flow may be attributed to the production of stable oxidizing radicals on the donor side of PSII when the supply of electrons from the Mn cluster is low. Both oxidizing agents P_{680}^+ and Yz^+ potentially oxidize the adjacent pigments and amino acids resulting in a photoinactivated reaction center (Blubaugh *et al.* 1991, Telfer and Kunkel 1991, Jegerschoeld *et al.* 1995). Donor-side photoinactivation also results in irreversible damage to D1 protein through its degradation. The targets for these damaging oxidizing species are not known at present, but the illumination of purified PSII reaction center particles in the presence of an artificial electron acceptor led to irreversible photobleaching of β-carotene and accessory chlorophyll, designated as Chl-670 (Telfer and Kunkel 1991).

Excess excitation-independent photodamage: The excess excitation-independent photodamage is induced due to the disruption of Mn–oxo bridges in the Mn_4Ca clusters leading to the release of Mn into the lumen (Wei *et al.* 2011, Zavafer *et al.* 2015). Further damage to PSII can be inflicted by exogenously generated ROS at iron–sulfur centers and cytochromes of thylakoids (Jung and Kim 1990, Suh *et al.* 2000).

Photodamage of PSI by HL stress

PSI is considered to be more stable than PSII under various environmental stresses, however, it is prone to photodamage on the transfer of excess electrons from PSII. Excess light kinetically limits the electron transfer from P_{700} to downstream electron acceptors such that the transient state of the excited P_{700} chlorophyll in PSI is de-excited to the triplet state ($^3P_{700}$) through the charge separation between P_{700} and A_o (chlorophyll *a* molecule which is primary electron acceptor of PSI), causing $^3P_{700}$ to react with O_2 to produce singlet $^1O_2$ (Rutherford *et al.* 2012) and cause photoinhibition of PSI (Cazzaniga *et al.* 2012). Light stress causes the nonavailability of oxidized ferredoxin at PSI such that the excess electrons are passed from the iron–sulfur (FeS) clusters at the stromal side of PSI to oxygen, forming superoxide ($O_2^{\bullet-}$) at the A_1 (phylloquinone) site within the thylakoid membranes (Kozuleva and Ivanov 2010, Kozuleva *et al.* 2014) and is

further dismutated to hydrogen peroxide (H_2O_2) (Asada 1999). At the PSI FeS clusters, this H_2O_2 is converted into $^{\bullet}OH$ through the Fenton reaction (Sonoike *et al.* 1995, Ivanov *et al.* 1998, Sonoike 2011) leading to oxidative destruction of the PSI center. Although photooxidative damage by PSI under HL was established from various studies, Lima-Melo *et al.* (2019) reported that it did not result in the accumulation of ROS in the whole leaves of *Arabidopsis* as the inactivation of PSI prevented further addition of ROS to the stromal pool.

Chloroplast signaling under light stress

Chloroplasts can communicate their status to the nucleus through retrograde signaling to regulate nuclear stress-responsive genes. The SAL1/phosphonucleotide-3'-phosphoadenosine-5'-phosphate (PAP), methylerythritol cyclodiphosphate (MEcPP), and ROS pathways act as important components of the chloroplast retrograde signaling pathway (Song *et al.* 2021) (Fig. 1). MEcPP, an intermediate of the methylerythritol phosphate (MEP) pathway for plastid isoprenoid biosynthesis, functions as another retrograde signal to activate stress-responsive nuclear gene expression (Xiao *et al.* 2012). When exposed to HL stress, plants accumulate MEcPP and regulate the expression of a series of nuclear genes (Benn *et al.* 2016). Also, the activity of SAL1 is inhibited resulting in the accumulation of PAP (Watson *et al.* 2018). The ROS can also function as a retrograde signal and modify the nuclear transcriptome to cope with these adverse stresses. The transcriptome of *A. thaliana* cell suspension culture under high light conditions showed that the transcription factors that regulate ROS scavenging were upregulated during early transcriptional responses to high light stress (González-Pérez *et al.* 2011).

A role for N6-methyladenosine (m6A) modification of transcripts of genes that affect the photosystem function is seen under HL stress. There is evidence that m6A modification has an important role in acclimation to high light. This modification positively regulates photosynthesis by reducing the activity of the photosystem and also by reducing the protein abundance during light stress. The genes involved encode proteins that have the photoprotection function (HHL1, MPH1, and STN8). This mechanism is an important way by which plants maintain photosynthetic activity under HL stress (Zhang *et al.* 2022).

The accumulated ROS lead to the ubiquitination of envelope proteins possibly by activating a cytoplasmic E3 ligase such as PLANT U-BOX 4 (PUB4). These ubiquitination moieties may be recognized by cellular degradation machinery that transports the damaged

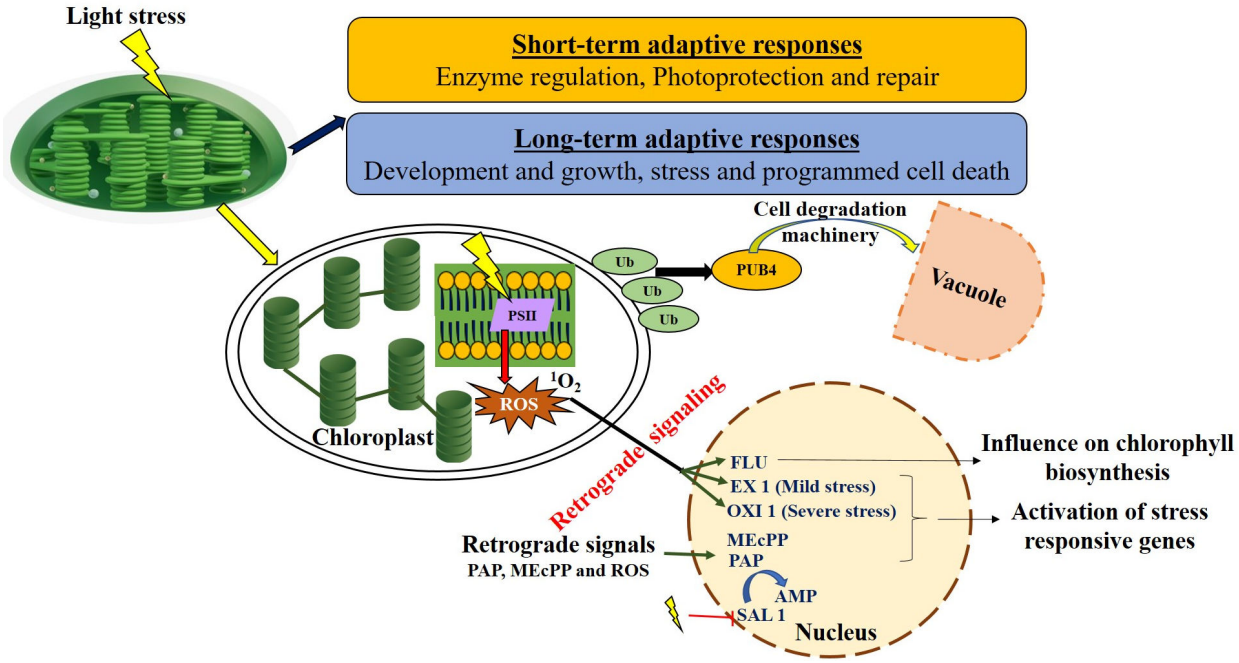

Fig. 1. Chloroplast signaling under light stress. Light stress on chloroplast activates various short-term and long-term adaptive responses. Light stress is perceived by the thylakoid membrane in chloroplast leading to chloroplast retrograde signaling which typically includes methylerythritol cyclodiphosphate (MEcPP), SAL1/phosphonucleotide-3'-phosphoadenosine-5'-phosphate (PAP), and ROS. Under light stress, singlet oxygen ($^1O_2$) is generated at PSII of the electron transport chain in the thylakoid membrane which mediates the transcriptional response of Flu protein that negatively regulates chlorophyll biosynthesis. $^1O_2$ accumulates in damaged chloroplasts leading to the ubiquitination of envelope proteins, possibly by activating a cytoplasmic E3 ligase such as PLANT U-BOX 4 (PUB4). $^1O_2$-derived signals can signal the nucleus to induce the expression of stress genes through EXECUTOR 1 (EX1) and OXIDATIVE SIGNAL INDUCIBLE 1 (OXI1) pathways. MEcPP and PAP also regulate the expression of a series of nuclear genes in response to light stress.

chloroplast to the central vacuole for turnover. At the same time, ROS-derived signals can signal the nucleus to induce the expression of stress genes through the EXECUTOR 1 (EX1) (mild light stress and enzymatic lipid peroxidation) and OXIDATIVE SIGNAL INDUCIBLE 1 (OXI1) (severe light stress and nonenzymatic lipid peroxidation) pathways. As $^1O_2$ is unlikely to leave the chloroplast, secondary messengers such as the β-carotene oxidation product β-cyclocitral (β-C) may travel to the cytoplasm or nucleus. However, under milder stress, the cell can choose to reduce ROS production in a chloroplast by quickly downregulating the import of photosystem components. In this case, the chloroplast envelope-localized E3 ligase SUPPRESSOR OF PLASTID PROTEIN IMPORT 1 (PPI1) LOCUS 1 (SP1) can ubiquitinate the plastid protein imports (translocon on the outer chloroplast membrane, TOC) machinery leading to their turnover through the 26S proteasome (Woodson 2016).

Hormonal control of HL response

The interaction between ROS, antioxidants, and phytohormones coordinates a complex signaling network in response to environmental stress conditions, in which abscisic acid (ABA) and jasmonic acid (JA) appear to be the master regulators of photosynthesis. Reports on HL stress resulting in ABA-induced stomatal closure have been documented (Raven 2014, Merilo *et al.* 2015). Apart from ABA, the biosynthesis of growth-related phytohormones, such as auxins and cytokinins (CK), was suppressed after long-term exposure to HL which led to the inhibition of plant growth (Huang *et al.* 2019). CK-receptor and insufficient CK-signaling mutants showed better PSII function than wild-type plants and were found to be more susceptible to HL stress due to photodamage of D1 protein along with poor enzymatic and nonenzymatic photoprotective mechanisms (Cortleven *et al.* 2014, Janečková *et al.* 2018).

Jasmonic acid (JA) and 12-oxo-phytodienoic acid (OPDA)-responsive genes suggested a relationship with $^1O_2$-type signaling that may be triggered by an increase in excitation and/or reduction pressure on PSII (Shumbe *et al.* 2016). Notably, oxylipin signaling is well known to interact with other signaling pathways, especially with GA signaling through antagonistic interaction between the JA Zinc Finger Inflorescence Meristem (ZIM)-domain family proteins (JAZ) and DELLA transcription suppressors. Although *JAZ* and *DELLA* genes were upregulated by HL stress, a prominent role in GA signaling was not evident in the analysis of the genes induced by HL and recovery. Ethylene mutants (*eto1-1* and *crt1-3*) repressed the expression and activation of violaxanthin de-epoxidase (VDE) and increased ROS production (Chen and Gallie 2015).

Photoprotection from HL-induced photodamage

Plants have devised various protective mechanisms for the dissipation of HL-induced excitation pressure. These metabolic pathways act as 'safety valves' in plants as a combination of nonphotochemical and photochemical quenching.

Photosynthetic state transition and spillover: Light-harvesting complexes (LHCs) are pigment–protein complexes, the majority of which are bound to LHCII trimers, forming a shared light-harvesting system for PSI and PSII (Rantala *et al*. 2017). To ensure maximum electron transport efficiency, the distribution of absorbed light energy between the two photosystems in plants must be balanced. Therefore, the subset of the LHCII protein complex is relocated between the two photosystems to ensure equitable light distribution and is known as photosynthetic state transition (Minagawa 2011). The phenomenon of state transition and spillover are difficult to separate and both involve equitable energy distribution between the two photosystems (Allen and Forsberg 2001). Spillover is widely distributed in both algae and land plants and requires both photosystems to be close (Yokono *et al.* 2015, Slavov *et al.* 2016). LHCII trimers are mostly found in the grana core and are made up of the proteins LHCB1, LHCB2, and LHCB3, the former two are phosphorylated reversibly to regulate efficient energy distribution (Damkjaer *et al.* 2009, Pietrzykowska *et al.* 2014, Wu *et al.* 2021). Adjustment of photosynthetic apparatus to changing light intensity requires cooperation between protein phosphorylation of PSII core complex and LHCII. Under HL conditions, the first component involves a conformational change in LHCII that allows it to dissipate excess energy as heat, thereby reducing the amount of energy transferred to the photosystem. The second component is the pH gradient across the thylakoid membrane, which becomes more acidic under HL conditions leading to the formation of a protonated form of zeaxanthin (Mekala *et al.* 2015). This protonation triggers the conversion of violaxanthin to zeaxanthin *via* the xanthophyll cycle, which results in the dissipation of excess energy as heat. The third important component is the PsbS protein, a protein that plays a critical role in regulating nonphotochemical quenching (NPQ). To induce LHCII aggregation, NPQ requires a combination of low pH in the thylakoid lumen and activation of PSII subunit S (PsbS) (Nicol and Croce 2021). Under HL conditions, PsbS can bind to the LHCII complex and trigger the conformational changes necessary for dissipating excess energy as heat. In addition, PsbS can regulate the activity of the enzyme responsible for the conversion of violaxanthin to zeaxanthin, thereby controlling the rate of energy dissipation (Simkin *et al.* 2022, Ghosh *et al.* 2023).

Nonphotochemical quenching (NPQ): heat dissipation channel: NPQ has been regarded as a safety valve to dissipate excess excitation energy not utilized during photochemistry and prevent the formation of ROS (Nath *et al*. 2013, Rochaix 2014, Tikkanen *et al.* 2014, 2015). NPQ is made up of several components that vary in time scales of activation under excess excitation energy and subsequent relaxation upon restoration of a normal light

(Ruban *et al.* 2012). The thermal dissipation by NPQ called q_E (energy-dependent quenching) is the fastest and activates in 0.1 to 1 s and relaxes within 1–2 min upon restoration to normal light (Li *et al.* 2000, Ruban *et al.* 2012). The site of its occurrence is mainly LHCII and PSII core (Nicol *et al.* 2019, Ruban and Wilson 2021). It necessitates a low thylakoid lumen pH which is generated by the increased proton transport to the lumen *via* saturated linear electron flow and PGR5/PGRL1-dependent cyclic electron transport, which is controlled by NADPH-dependent thioredoxin reductase C (Naranjo *et al.* 2021) (Fig. 2). As q_E de-excites a singlet excited chlorophyll, it is also called 'feedback de-excitation' (Külheim et al. 2002). The importance of q_E as an adaptive trait under HL stress is known in plants growing in excess light environments that have higher q_E capacities and xanthophyll pools. Excess light causes the de-epoxidation of violaxanthin to zeaxanthin which is catalyzed by a thylakoid lumen enzyme, violaxanthin de-epoxidase (VDE). Along with the presence of zeaxanthin in the low thylakoid lumen, the PsbS subunit of PSII and/or light-harvesting complex stress-related proteins (LHCSRs) activates the binding of zeaxanthin to facilitate energy transfer and de-excitation of chlorophyll (Ivanov *et al.* 2006). Thus, PsbS allows the exchange of xanthophylls in thylakoid membranes contributing to the synergistic effect of PsbS and zeaxanthin under HL intensity (Welc *et al.* 2021, Nosalewicz *et al.* 2022). Acidification of the lumen also activates specific proteins that act as q_E effectors which, in turn, increase the capacity of the LHCII to dissipate energy. Studies with mutants of genes encoding violaxanthin de-epoxidase (*npq1*) and PsbS (*npq4*) showed the increased extent of photoinhibition and production of singlet oxygen under HL intensities (Li *et al.* 2002, Roach and Krieger-Liszkay 2012). The redistribution of excitation energy between the two photosystems, known as photosynthetic state transition (q_T), is another strategy

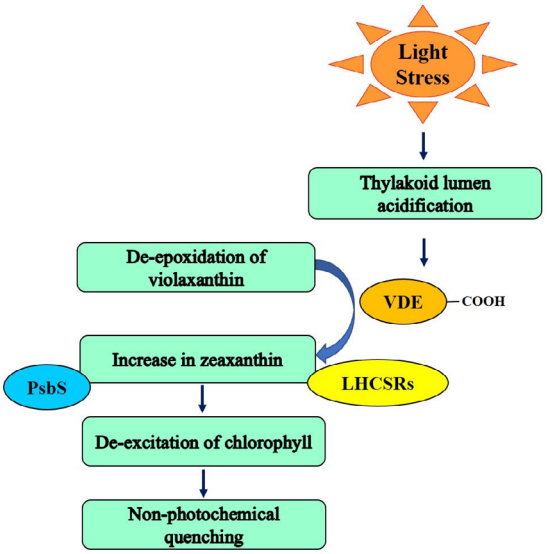

Fig. 2. Mechanism of PSII photoprotection under light stress.

for constantly adapting to light imbalances (Minagawa 2011). The state transition component prevents the over-reduction and overoxidation of both the photosystems and has been described in the previous section. Another key component of NPQ includes photoinhibitory quenching (q_I) that involves the reduction in the number of active PSII reaction centers due to photooxidative damage to the D1 protein of PSII (Edelman and Mattoo 2008, Derks *et al.* 2015). q_I component has slow relaxation kinetics and is considered a long-term mechanism of energy dissipation (Zulfugarov *et al.* 2014, Malnoë *et al.* 2018).

Although NPQ has a protective role, it continues to operate under shaded conditions or sun-flecking within the crop canopy, reducing photochemical efficiency (Ruban *et al.* 2012, Ghosh *et al.* 2023). To improve NPQ efficiency, a construct called VPZ, which consists of violaxanthin de-epoxidase, zeaxanthin epoxidase, and PsbS was overexpressed in tobacco and soybean. The transgenic plants showed improved photosynthetic efficiency, biomass accumulation, and grain yield primarily through an increase in seed number which suggested that modification of NPQ kinetics is a viable strategy for increasing crop yields (Kromdijk *et al.* 2016, De Souza *et al.* 2022). Further, exploration of zeaxanthin epoxidase and NPQ kinetics and its association with the lutein epoxide cycle that operates in many crops opens a way to reconfigure light reactions to develop stress-resilient crops (Ghosh *et al.* 2023).

PSII photoinhibition-repair cycle: a regulator of photosynthetic electron transfer chain: The maintenance of PSII activity is one of the most difficult challenges for organisms performing oxygenic photosynthesis under light stress because of the high vulnerability of the D1 protein (Townsend *et al.* 2018, Fagerlund *et al.* 2020, Tian *et al.* 2021). High light intensity increases phosphorylation of the D1 core protein of PSII that causes the unpacking and mobility of PSII-LHCII preventing strict separation between PSII-LHCII and PSI. This process causes uncontrolled excitation energy transfer from PSII-LHCII to PSI. During the PSII photoinhibition-repair cycle, the photodamaged D1 core protein is degraded and replaced by *de novo* synthesis to maintain protein homeostasis in the thylakoid membrane (Baena-González and Aro 2002). The repair cycle starts with the monomerization of the phosphorylated dimeric PSII complex in grana stacks (Aro *et al.* 2005). The photodamaged D1 protein is collectively removed by proteases [Zn-dependent filamentation temperature sensitive H (FtsH) metalloprotease and Deg protease] followed by its replacement with newly synthesized peptides (Nelson *et al.* 2014, Kato *et al.* 2015). The thylakoid insertion of the newly-synthetized D1 proteins, dimerization and religation of pigments and cofactors, and reactivation of the oxygen-evolving complex and electron transport in the non-appressed domains of the thylakoid membrane are involved in the repair cycle (Lu *et al.* 2011, Suorsa *et al.* 2014). The entire process consumes 1,300 ATP per D1 molecule synthesized (Wang *et al.* 2016, 2021; Murata and Nishiyama 2018). Several

other auxiliary proteins are also needed for translational insertion of the newly synthesized D1 protein, *e.g.*, LPA1 (*Chlamydomonas* REP27), CYP38, and PAM68 (*Synechocystis* Sll0933) which are required for efficient translation of psbA, its maturation and correct assembly of PSII complex (Mulo *et al.* 2012).

Protection of PSI during light stress: Under excess light, the reaction center chlorophyll of PSI, P_{700}, is kept oxidized to maintain the balance between light utilization and dissipate the excess photoexcitation energy in PSI. The oxidation state of P_{700} is regulated by several molecular mechanisms on both the electron donor and acceptor sides of PSI (Lima-Melo *et al.* 2019). The electron donor side control of PSI photodamage is achieved through 'photosynthetic control' where electron supply is limited at the Cyt b_6f (Tikkanen *et al*. 2014, Chaux *et al.* 2015) in response to high lumen acidification created by the oxidation of H_2O at the luminal side of PSII and the Q-cycle in Cyt b_6f (Tikkanen *et al.* 2015, Colombo *et al*. 2016). On the acceptor side of PSI, electrons are excited to reduce ferredoxin (Fd), and are used by the enzyme Fd:NADP(H) oxidoreductase (FNR) and flavodiiron proteins (FLVs or FDPs) to generate NADPH and support P_{700} oxidation (Shin *et al.* 1963). FNR and FLV are critical components of the post-PSI electron transfer cascade, their abundance and location on the membrane (close to PSI), might contribute to PSI protection (Burlacot *et al.* 2018).

Photorespiration can function as an O_2-dependent alternative electron sink to dissipate excess light energy (Kozaki and Takeba 1996, Takahashi *et al.* 2007). In this process, both reduced Fd and ATP are required for the regeneration of 3-phosphoglycerate from 2-phosphoglycolate in the photorespiratory carbon oxidation cycle. Thus, P_{700} oxidation is maintained by relieving the limitation of PSI on the acceptor side. Numerous reports suggest that photorespiration functions as the largest alternative electron flow to O_2 and are responsible for P_{700} oxidation and the protection of PSI against photoinhibition in C_3 plant leaves (Takagi *et al.* 2016, Wada *et al.* 2018).

Besides nonphotochemical quenching (NPQ) through zeaxanthin and protonation of the PsbS protein, two PSI cyclic electron flow (CEF) systems, *i.e.*, the NADH dehydrogenase-like complex-dependent pathway and the ferredoxin-plastoquinone reductase pathway cause the enhancement of NPQ by generating the electrochemical potential difference of H^+ across the thylakoid membrane (Kono *et al.* 2014).

Cyclic electron flow (CEF) around PSII and PSI: CEF works by the concerted action of both PSII and PSI and is implicated in the photoprotection of both the photosystems as well as providing ATP to fix atmospheric CO_2 (Nawrocki *et al.* 2019). The photosynthetic regulation by CEF varies between the immature and mature leaves as it contributes towards photoprotection in immature leaves and more towards ATP synthesis in mature leaves (Huang *et al.* 2017). Immature leaves have a reduced ability to utilize light energy as evidenced by lower electron flow from PSII, so the ability to dissipate excess light energy becomes critical for photoprotection of both photosystems (Rott *et al.* 2011). Further, the light-saturation point of electron transport from PSII is much lower, limiting photosynthetic CO_2 assimilation and increasing ROS production (Murata *et al.* 2007). To limit ROS production during the repair process, CEF-mediated strong acidification of the thylakoid lumen facilitates Ca^{2+} sequestration in the lumen, which helps stabilize the water-splitting complex against photodamage (Krieger and Weis 1993). On the other hand, mature leaves grown under HL intensity dissipate excess light energy by activating the zeaxanthin pigment-dominated nonphotochemical quenching (Li *et al.* 2002).

Mehler ascorbate peroxidase (MAP) pathway or water–water cycle (WWC): Under HL conditions, the excess electrons are accepted by the molecular oxygen causing O_2 reduction at the acceptor side of PSI or at phylloquinone A1 site to generate superoxide ($O_2^{\bullet-}$). This process is called Mehler reaction and the superoxide produced in the process can undergo dismutation either spontaneously or by chloroplastic superoxide dismutase (SOD) to yield H_2O_2 (Kozuleva *et al.* 2020) which is further detoxified to H_2O and O_2. This process is also called water–water cycle (WWC) as the electrons flow from water in PSII to form water in PSI (Asada 1999). In chloroplast, several antioxidant enzymes are present to reduce the ROS contents, namely stromal ascorbate peroxidase (sAPX), thylakoid-bound ascorbate peroxidase (tAPX), 2-Cys peroxiredoxins (2CPA and 2CPB), one peroxiredoxin Q (PrxQ), one type II peroxiredoxin (PrxIIE), and two glutathione peroxidase-like proteins (GpxL1 and GpxL7) (Chang *et al.* 2009, Dietz 2016). PSI was found to be highly reduced in *Bletilla striata*, *Arabidopsis thaliana*, and *Camellia* species after a sudden transition from low light to HL accompanied by a relatively low proton gradient insufficient to downregulate the electron flow from PSII. The presence of WWC favored electron exit from PSI to O_2, resulting in the rapid oxidation of PSI thereby protecting it from photoinhibition (Huang *et al.* 2019, Sun *et al.* 2020). However, WWC is more effective at protecting PSI from photoinhibition at room temperature than at low temperature (4°C) when light intensity is suddenly increased (Huang *et al.* 2021).

Plastid or plastoquinol terminal oxidase (PTOX): PTOX is a non-heme-diiron carboxylate protein found in photosynthetic organisms that oxidize plastoquinol (PQH_2) to plastoquinone (PQ) and reduces O_2 to H_2O (Josse *et al.* 2003). High light intensity is frequently experienced by alpine plant species such as *Ranunculus glacialis* growing at high altitudes. The plastoquinone reoxidation rate in leaves of these plants exposed to the sun is faster than that of shade leaves (Laureau *et al*. 2013). When HL intensity results in a highly reduced pool of plastoquinone, PTOX acts as a safety valve to keep the acceptor side of PSII oxidized and prevent photoinhibition (Feilke *et al*. 2016, Ahmad *et al.* 2020). Localization of PTOX in the chloroplast is highly dependent on the proton motive force (Bolte *et al*. 2020). Experiments with dark-to-light

transitions revealed that the conserved C-terminus domain of PTOX contains cysteine residues that are oxidized in the dark and reduced in low light intensity (Rog *et al.* 2022). High light intensity increases the magnitude of the proton gradient and facilitates PTOX attachment to the thylakoid membrane followed by its subsequent oxidation, which would otherwise result in triplet chlorophyll formation and ROS production (Ahmad *et al.* 2020, Bolte *et al.* 2020).

Mitochondrial alternative oxidase (AOX) pathway: The cyanide-sensitive cytochrome *c* oxidase (COX) and the cyanide-insensitive alternative oxidase are two terminal oxidases in the mitochondrial electron transport chain that compete for electrons from the ubiquinone pool, with only COX coupled with proton translocation (Ribas-Carbo *et al.* 1995). Mutant studies in *A. thaliana* under increasing light intensity [50, 250, and 700 µmol(photon) m^{-2} s^{-1}] revealed that AOX helps to maintain the redox status of the photosynthetic electron transport chain by sustaining a higher quantum yield of PSII. It maintains a high ratio of open reaction centers of PSII and prevents the over-reduction of chloroplastic electron transporters (Vishwakarma *et al.* 2015). In addition, HL-mediated generation of reducing equivalents (NADPH) above the Calvin cycle requirement is transported as malate through the malate–oxaloacetate shuttle which is dissipated by the AOX pathway to maintain chloroplast electron transporters in the oxidized state (Zhang *et al.* 2010, Vishwakarma *et al.* 2015). The ROS concentrations can also be lowered by AOX through the activation of the antioxidant defense system (Strodtkötter *et al.* 2009). Fig. 3 illustrates the process of alternative electron flow during light reactions under light stress.

Strategies to improve photosynthetic efficiency under HL intensity

To conclude we suggest the following approaches to optimize photosynthetic light reactions and improve photosynthetic efficiency under high light.

Enhancing light capture by decreasing antenna size: Light distribution across the leaves of the plant in a dense

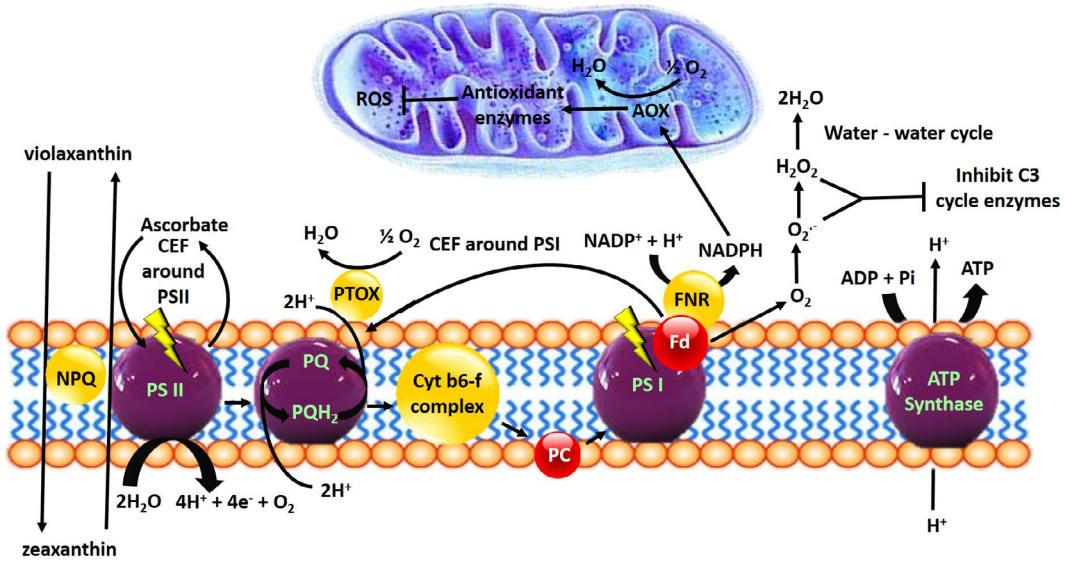

Fig. 3. Alternative electron flow during light reactions under light stress. Excess of excitation energy leads to lumen acidification that activates a safety valve called NPQ which dissipates excess energy as heat to prevent the formation of chlorophyll triplets and ROS. Lumen acidification favors the attachment of PTOX that competes for electrons under excess excitation pressure with PSI to reduce O_2 to H_2O. CEF around PSII, recently characterized in oxygenic phototrophs and desert microalgae results in a backward flow of electrons from plastoquinol is responsible for PSII-CEF. CEF-mediated strong acidification of thylakoid lumen dissipates excess excitation energy *via* NPQ, which promotes the PSII repair process, limiting ROS production. Furthermore, stronger acidification of the thylakoid lumen facilitates Ca^{2+} sequestration in the lumen, which aids in the photoprotection of the water-splitting complex. Under HL conditions, the excess excitation energy is dissipated as heat *via* NPQ, with the remainder resulting in the production of superoxide radical ($O_2^{\bullet-}$) with electron donors being the FeS centre in the PSI complex, Fd at PSI, and flavoproteins in chloroplasts. $O_2^{\bullet-}$ thus produced undergoes dismutation either spontaneously or catalyzed by chloroplastic SOD to yield H_2O_2 and O_2. Both $O_2^{\bullet-}$ and H_2O_2 inhibit thiol group containing enzymes of the Calvin cycle (NADP-glyceraldehyde-3-phosphate dehydrogenase, fructose-1,6-bisphosphatases, sedoheptulose-1,7-bisphosphatases, ribulose-5-phosphate kinase) to hamper photosynthesis. Subsequently, peroxidases then reduce H_2O_2 to water. As a result, O_2 produced by the water-splitting complex is reduced to water by electrons from PSI, a process known as the water–water cycle, which protects photosynthetic apparatus photooxidation and prevents photoinhibition. Furthermore, excess NADPH generated by light is transported as malate *via* the malate–oxaloacetate shuttle and dissipated by the AOX pathway to keep chloroplast electron transporters oxidized. The protective mechanisms during excess light initiate signaling pathways that are closely knitted into the regulatory network and may also interact with one another. Signals perceived by chloroplast can convey the information to the nucleus through retrograde signaling resulting in the remodeling of the photosynthetic apparatus to re-establish photostasis and energy balance.

crop canopy is not uniform as leaves of the upper canopy absorb most of the photosynthetic active radiation (PAR) resulting in a high photosynthetic rate as compared to the lower canopy. The overall photosynthesis in the entire plant can be improved by introducing mutants with a decreased cross-section of the LHC antennae, thereby allowing PAR to travel deeper and uniformly into the crop canopy (Kirst *et al.* 2017). Jin *et al.* (2016) developed a mutant of *A. thaliana* with affected regulation of chlorophyll synthesis which showed improvements in light use as evidenced by a 50% increase in the amounts of accumulated glucose and fructose, as well as more than 10% dry-mass biomass in mature plants. Gu *et al.* (2017) performed pot and field experiments using rice with up to 50% less chlorophyll content and showed up to 40% increase in photosynthetic rate, elevated concentrations of ribulose-1,5-bisphosphate carboxylase/oxygenase, and faster growth rates, which translated into similar yields to the wild-type but in less time. A yellow-green line with truncated light-harvesting antennae in the model plant *N. tabacum* resulted in 25% higher biomass accumulation per unit absorbed light (Kirst *et al.* 2017).

Improving nonphotochemical quenching features increased biomass accumulation in dynamic light conditions: To prevent or mitigate the damaging effects of light stress-induced ROS production in plants, there is the activation of NPQ mechanisms through which excess excitation energy can be dissipated as heat in the light-harvesting complexes (Ruban 2016). As long as the plants are exposed to HL intensities, NPQ components are quickly initiated to dissipate excess excitation energy as heat, thereby preventing photoinhibition of the photosynthetic machinery. The fastest NPQ component q_E (energy-dependent quenching) initiates within seconds to minutes after acidification of the thylakoid lumen, which is subsequently further enhanced through xanthophyll cycle activation, *i.e.*, the conversion of violaxanthin (V) into photoprotective zeaxanthin (Z) *via* antheraxanthin (A). Simultaneously, NPQ leads to the inhibition of photosynthetic efficiency which ultimately drops to very low levels under HL conditions. NPQ relaxes upon the shifting of HL to low light or dark conditions and pigment–protein PSII efficiency recovers through the reconversion of zeaxanthin to violaxanthin. However, full relaxation of NPQ after HL stress is a rather slow process (30–60 min or longer), during which photosynthetic capacity is still inhibited to some extent under otherwise optimal conditions, thereby possibly losing time for biomass production. Kromdijk *et al.* (2016) developed transgenic VPZ tobacco plants by overexpressing the lumenal pH sensor protein PsbS and the xanthophyll-converting enzymes, which displayed faster NPQ relaxation under changing light conditions and thus faster recovery of photosynthesis resulted in higher biomass accumulation compared to control plants.

Translating strategies from lower plants/microalgae/cyanobacteria into higher plants: Evolutionary studies suggest that oxygenic photosynthesis was first of all developed in cyanobacteria and subsequently in microalgae and plants. Research is in progress to translate the potentially advantageous mechanisms associated with ancestral proteins from lower organisms into higher plants to improve photosynthetic performance in crops. Various researchers have reported that expression of algal cytochrome c_6 (cyt c_6) protein in *Arabidopsis* and tobacco resulted in an improved photosynthetic electron transfer and biomass accumulation under field conditions (Chida *et al.* 2007, Yadav *et al.* 2018, López-Calcagno *et al.* 2020). Chida *et al.* (2007) inserted a *cyt* c_6 gene from the red alga *Porphyra yezoensis* into *Arabidopsis* and Yadav *et al.* (2018) introduced a *cyt* c_6 gene from the green macroalga *Ulva fasciata* (sea lettuce) into tobacco. Both studies reported enhanced growth phenotypes during the first eight weeks of plant growth, following increased chlorophyll and photosynthetic metabolite contents, although other photosynthetic parameters were only slightly improved.

Another class of photosynthetic flavoproteins, which disappeared in angiosperms throughout evolution is flavodiiron proteins (FDPs), that can be promising tools for the bioengineering of future crops (Mullineaux 2016). FDPs serve as photoprotective excess electron valves in the so-called 'Mehler-like reaction' or water–water cycle of photosynthesis (Ilík *et al.* 2017, Alboresi *et al.* 2019) across a large part of the green lineages from cyanobacteria up to gymnosperms. In angiosperms, in which FDPs are absent, the introduction of FDPs could therefore possibly replace several ROS-scavenging enzymes and reactions, thus saving energy and nitrogen sources or adding extra protection. Transgenic lines of tobacco, *Arabidopsis* and barley expressing cyanobacterial Flv1/3 or Flv2/4 proteins in chloroplasts showed that FDPs can act as additional electron sinks in plants as well, particularly under fluctuating light stress, thereby improving photosynthetic performance (Gómez *et al.* 2018, Tula *et al.* 2020, Shahinnia *et al.* 2021, Vicino *et al.* 2021).

Enhanced production of ATP: Cyclic electron flow around PSI is mainly responsible for the production of ATP per NADPH as compared to linear electron flow. The number of c subunits in the c ring of the F_o complex of ATP synthase determines the stoichiometry of ATP produced per H^+ translocated through the complex (Walker 2013). Thus, engineering ATP synthase to have a smaller ring would automatically boost the amount of ATP produced per NADPH in linear electron flow to offer an advantage under conditions of constant illumination (Cardona *et al.* 2018). Fig. 4 presents various strategies to improve crop yields through the optimization of photosynthetic light reactions.

Conclusion

Photoinhibition, photoinactivation, photooxidation, solarization, and photodynamic reactions reduce the efficiency of photosynthetic light reactions when excitation pressure is too high. The existence of photoprotective and alternative electron flow mechanisms are thus prerequisites for the survival of plants under fluctuating

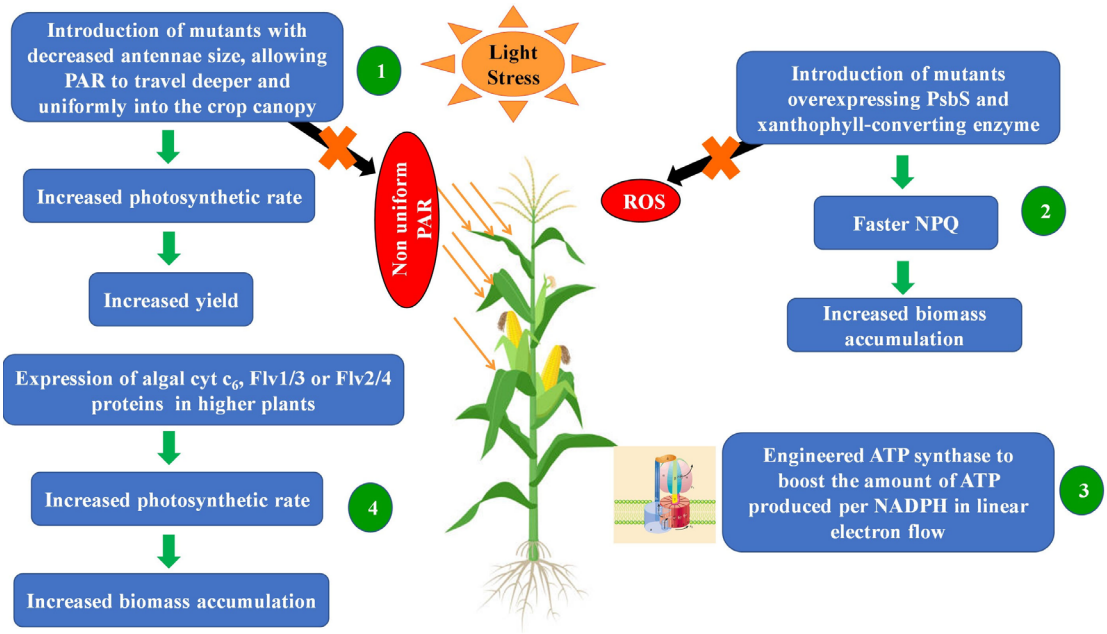

Fig. 4. Various strategies to improve crop yields through optimization of photosynthetic light reactions. 1 – enhancing light capture by decreasing antenna size; 2 – improving nonphotochemical quenching features increased biomass accumulation in dynamic light conditions; 3 – translating strategies from lower plants/microalgae/cyanobacteria into higher plants; 4 – producing more ATP.

light environments. Dissipation of excess excitation energy by NPQ, the photoinhibition-repair cycle of PSII, slowing down the electron flow from the donor side of PSI, and enhanced electron flow on the acceptor side to keep PSI in the oxidized state are key adaptive mechanisms under HL stress. In addition, activation of several stress-responsive genes *via* chloroplast retrograde signaling and hormonal induction of antioxidant enzymes mitigate light stress-induced damage. To prevent the reduction of molecular oxygen, several alternative electron sinks are available, *i.e.*, plastid or plastoquinol terminal oxidase (PTOX), cytochrome b_6/f complex, cyclic electron flow through PSI (CEF), Mehler–ascorbate peroxidase (MAP) pathway or water–water cycle (WWC), mitochondrial alternative oxidase pathway (AOX), and photorespiration.

To improve the efficiency of light reactions, genetic engineering approaches can be utilized to enhance light capture by decreasing antenna size and improving nonphotochemical quenching. Transgenes from lower plants/microalgae/cyanobacteria can also be integrated into higher plants. ATP synthase can be genetically engineered to produce more ATP under HL stress. Thus, optimization of photosynthetic light reactions resulting in increased photosynthetic efficiency provides an effective long-term solution to boost and sustain crop productivity under HL stress.

We have reviewed the current status of photosystems under HL stress here and we feel that still there are a lot of unanswered questions on the molecular mechanisms of photodamage and recovery. In the past decade, there has been renewed interest by researchers and the field has been kept vibrant and active in terms of new knowledge that is being gained. We trust that our coverage of the subject here will help formulate questions and that several gaps need to be filled, especially with the advancement in technology both the photosystems can be explored in more detail in the future.

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