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Review

# Photoinhibition or photoprotection of photosynthesis? Update on the (newly termed) sustained quenching component qH☆

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ABSTRACT

Non-photochemical quenching (NPQ) of chlorophyll fluorescence is a valuable feature for the study of photosynthetic organisms' light utilization and dissipation. However, all too often NPQ is simply equated with the harmless dissipation of excess absorbed light energy as heat. This is not always the case as some processes cause NPQ without thermal dissipation. Photoinhibitory quenching, qI, is sustained NPQ that continuously depresses the commonly used fluorescence parameter "quantum yield of photosystem II (PSII)", or $F_v/F_m$, and is often viewed as a result of PSII core inactivation due to D1 damage. Inactivated PSII cores might have a photoprotective role but that is not the topic of the present review. Instead, this review focuses on a sustained photoprotective antenna quenching component, which we have termed qH, and summarizes the recently uncovered molecular players of this sustained form of NPQ.

## 1. Introduction

Photosynthesis is a biological process of primary importance, as it provides the energy source for food, feedstock and biofuel production and also mitigates climate change. As light in excess of photosynthetic capacity can lead to cellular damage (Li et al., 2009), protective mechanisms against damage have evolved, including ways to minimize light absorption, detoxify reactive oxygen species generated by excess light, and dissipate excess absorbed light energy as heat (Horton et al., 1996; Logan et al., 2014). Together, these processes constitute photoprotection. The present review will focus on photoprotection by thermal excess energy dissipation (known as non-photochemical quenching, NPQ, see below) and the distinction between the contributions of photosystem II (PSII) inactivation and slowly relaxing NPQ mechanisms to the phenomenon of photoinhibitory quenching (qI). This distinction has been previously discussed by (Demmig-Adams and Adams, 2006) for overwintering plants. The aim of this review is to clarify and update the definition of qI, which refers to NPQ due to photoinhibition (decreased $CO_2$ fixation). qI has rightfully been called "ill-defined" (Nilkens et al., 2010) and the source of confusion in the field comes from the term "photoinhibition" as (Maxwell and Johnson, 2000) explained: "it is important to note that this term, when applied to fluorescence analysis, generally refers to both protective processes and to damage to the reaction centers of PSII (Osmond, 1994), whilst in more molecular studies, it is specifically the latter that is referred to as photoinhibition." Furthermore, viewing photooxidation of the PSII core subunit D1 and the oxygen evolving center (OEC), and the resulting inactivation of PSII photochemistry, as photodamage has the connotation of a passively occurring process that should be avoided. Instead, using the term 'photoinactivation' may better capture the cell's control of D1 status (as discussed in Section 1.3) to prevent more widespread damage. The present review will emphasize that photoinhibitory quenching can be due to PSII core inactivation and degradation as well as slowly relaxing NPQ mechanisms, and can therefore be photoprotective.

### 1.1. Non-photochemical quenching (NPQ) of chlorophyll fluorescence

Photosynthetic organisms possess pigments (chlorophyll (Chl) and carotenoids) that absorb light energy. The fate of absorbed light energy is to ultimately power photochemistry, which generates chemical energy in the form of ATP and NADPH. Light energy in excess is dissipated as heat. Some of the energy that is neither used for photochemistry nor dissipated as heat is emitted as fluorescence. Chlorophyll fluorescence is thus a good proxy to assay these processes (Baker, 2008; Eberhard et al., 2008; Horton et al., 1996). When measured at room temperature, chlorophyll fluorescence largely comes from the light-harvesting proteins, or antennae, of PSII. A decrease in fluorescence due to photochemistry (defined here as charge separation within PSII) is called *photochemical quenching*. Accordingly, a decrease in fluorescence that is not due to photochemistry is called *non-photochemical quenching*. The term NPQ is commonly used to refer to the dissipation of excess absorbed light energy as heat; however, this shortcut can be confusing to a non-specialist as not all processes

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contributing to non-photochemical quenching of chlorophyll fluorescence lead to thermal dissipation of excess excitation energy. The next subsections will briefly review what is known about the different NPQ processes and distinguish them by whether or not they actively promote thermal dissipation.

#### *1.1.1. NPQ processes that do not lead to thermal dissipation*

A decrease in fluorescence can be due to fewer fluorescing molecules arising from decreased light absorption rather than active thermal dissipation. Such is the case, for example, when chloroplasts are relocated within the cell and shade each other. Chloroplast movement occurs under white or blue actinic light and requires the phototropin receptor PHOT2 (Cazzaniga et al., 2013; Dall'Osto et al., 2014). This fluorescence decrease or quenching *sensu stricto* has been termed qM, for movement, but this component does not effectively promote thermal dissipation. Because this light-avoidance response is not induced by red light, use of red light might be preferable to measure NPQ processes unrelated to chloroplast movement.

Energy re-distribution occurring between the photosystems during the monitoring of chlorophyll fluorescence at room temperature can also result in a fluorescence decrease. This quenching component is called qT, for state transition, and is due to the movement of phosphorylated antenna proteins away from PSII (Quick and Stitt, 1989). Phosphorylated PSII antennae collect light energy that is transferred to photosystem I (PSI) where charge separations occurs, which leads to fluorescence quenching as recently demonstrated by (Nawrocki et al., 2016) in microalgae. In algae and moss, LHCSR1 was found to mediate such PSI quenching in excess light (Kosuge et al., 2018; Pinnola et al., 2015). Another model assumes that detached antennae thermally dissipate light energy (Unlu et al., 2014). In this latter model, qT would belong in the next subsection (Section 1.1.2). In plants exposed to saturating light, qT does not contribute to NPQ (Nilkens et al., 2010). For reviews regarding state transitions and NPQ, see (Tikkanen et al., 2012) and (Papageorgiou and Govindjee, 2014). When $Q_A$ is reduced, excitation-energy spillover from PSII to PSI photochemical units that are excitonically coupled will also lead to fluorescence quenching from PSI charge separation (see, e.g., (Kowalczyk et al., 2013)) or can lead to thermal dissipation if P700 is oxidized (see below). In plants, spillover is not believed to occur due to strict spatial segregation of the photosystems. However this idea is being revised as megacomplexes comprising both photosystems have been biochemically isolated (Rantala et al., 2017; Yokono et al., 2015). Whether these complexes or “photosystome” (in analogy to the respirasome) represent a physiological state enabling spillover remains to be explored.

#### *1.1.2. NPQ processes that lead to thermal dissipation*

Apart from the cases described above, a decrease in fluorescence means an increase in thermal dissipation of excess light energy. Heat is difficult to measure directly as it dissipates on time and space scales beyond the resolution of available instrumentation. Furthermore, deconvoluting the heat effect of the light source used to induce NPQ to the heat emitted by the biological system makes the problem all the more complex, which is why direct measurement of heat has not been the method of choice to study this phenomenon. Few studies using microcalorimetry (e.g., Gruszecki et al., 1996) or photoacoustic spectroscopy (Havaux and Tardy, 1997; Mullineaux et al., 1994; Yahyaoui et al., 1998) have been conducted and have found direct correlations between fluorescence quenching and heat dissipation.

Historically, NPQ components have been separated according to their relaxation kinetics and their sensitivities to chemical inhibitors (Quick and Stitt, 1989). However as discussed previously by Demmig-Adams et al. (2014), the relaxation kinetics of a given NPQ component can vary widely in nature and I therefore advise a distinction between NPQ components by the molecular players involved (Fig. 1; see also recent review by Pinnola and Bassi, 2018).

The flexible mode of energy dissipation, or energy-dependent quenching qE (Krause et al., 1982), has been the most studied NPQ component; it is turned on and off rapidly (seconds to minutes) and its key molecular players have been identified. In vascular plants, the protein PsbS gets protonated at glutamate residues in its lumenal loops upon light exposure and, together with the xanthophyll pigments zeaxanthin (and/or lutein), PsbS is necessary to catalyze formation of a quenching site (Demmig et al., 1987; Johnson and Ruban, 2011; Li et al., 2000; Niyogi et al., 1997; Sylak-Glassman et al., 2014). Exactly where and how PsbS acts is a topic of intensive investigation using a wealth of approaches, including structural (Fan et al., 2015; Krishnan et al., 2017), modeling (Zaks et al., 2012), biochemical (Correa-Galvis et al., 2016; Gerotto et al., 2015; Sacharz et al., 2017), and imaging approaches (Kereïche et al., 2010). Two membrane proteins localized to the grana core (RIQ1, RIQ2) were found to result in a *reduced induction of NPQ* when mutated and are proposed to regulate qE (Yokoyama et al., 2016). The term “economic photoprotection” has been used to describe the observation that qE competes efficiently with closed reaction centres ($Q_A$ reduced) but not with open ones thereby preserving productive electron transfer (Belgio et al., 2014). Recently it was further shown that this qE state is accompanied by a far red fluorescence band only when reaction centers are closed and that open reaction centers lower the quenching rate (Farooq et al., 2018).

A zeaxanthin-dependent quenching was characterized by Dall'Osto et al. (2005) and later termed qZ (Nilkens et al., 2010), moving the field away from a distinction of quenching components based on relaxation kinetics but rather focused on molecular players and mechanisms. The latter mechanism is PsbS- and ΔpH-independent, requires zeaxanthin, and involves a conformational change of at least the minor antenna protein CP26. This quenching component is induced and reversed on a timescale of minutes to tens of minutes.

Photoinhibitory quenching qI (Krause, 1988), comprises processes that relax slowly (hours or longer) – hence the term “sustained quenching”. Such processes result in photoinhibition, which is best defined as the light-induced decrease of the quantum yield of photosynthetic carbon fixation (Baker, 1996). qI can be due to inactivation and/or degradation of the D1 protein of PSII. However, not all of qI is due to PSII core inactivation/degradation, as there are slowly relaxing fluorescence quenching processes that are independent of D1 abundance (Demmig and Björkman, 1987) or D1 function (Chow et al., 1989). Some of these slowly relaxing processes are now described such as qZ and the recently termed qH (Malnoë et al., 2017; for more details, see Section 2). A possible new definition for qI could be quenching due to photoinactivation of D1 rather than due to photoinhibition, as qZ and qH are also photoinhibitory.

When PSII cores are photoinactivated, reaction-center (RC) quenching occurs due to charge recombination between $Q_A^-$ and $P680^+$ within PSII or possibly through other thermal traps whose nature is unknown (Finazzi et al., 2004; Ivanov et al., 2006; Matsubara and Chow, 2004). (Staleva et al., 2015) have recently found in cyanobacteria a pigment-protein complex that associates to the RC during assembly and enables photoprotective quenching. Similarly to PSII photoinactivation, (Tiwari et al., 2016) have proposed that inactivation of the PSI acceptor side associated with more LHCII antenna coupled to PSI leads to thermal dissipation of excess energy via oxidized P700 ($P700^+$) (Schlodder et al., 2005), which results in fluorescence quenching (named ‘PSI-NPQ’). Such thermal dissipation by $P700^+$ also occurs when PSII is excitonically coupled with PSI (spillover) and protects lichens (Slavov et al., 2013) and desert cyanobacteria from desiccation (Bar-Eyal et al., 2015) as well as coral symbionts from heat stress (Slavov et al., 2016).

### *1.2. Diversity of energy dissipation routes and mechanisms*

While there is a consensus that electronic interactions between Chl and carotenoid in the antenna of PSII regulate light harvesting, the biophysical mechanism(s) underlying NPQ processes is/are still a matter of debate (Walla et al., 2014). Several mechanisms have been

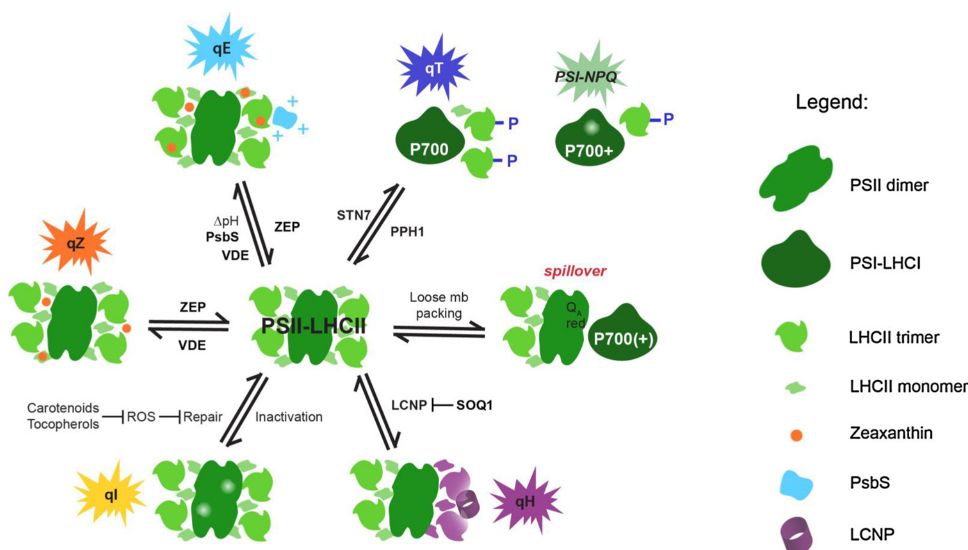


**Fig. 1. Model for the different NPQ states.** Top view of PSII-LHCII complexes and reversible changes occurring during the different NPQ states. ΔpH, pH gradient formed as a result of light reactions; +, protonated residues activate PsbS; violaxanthin de-epoxidase (VDE) and zeaxanthin epoxidase (ZEP) are enzymes of the xanthophyll cycle; −P, phosphorylation regulated by STN7 kinase and PPH1 phosphatase; during PSI-NPQ, inactivation of PSI occurs at FeS cluster $F_A/F_B$ (whitened); during spillover, loose membrane (mb) packing results in loss of photosystems spatial segregation and excitation-energy transfer from PSII to PSI when $Q_A$ is reduced (red); during qI, inactivation of PSII occurs at D1 subunit (whitened); ROS, reactive oxygen species; qM, fluorescence decrease or quenching due to chloroplast movement, not represented here because does not directly affect energy distribution at level of photosystems. Adapted from (Erickson et al., 2015).

proposed involving either Chl-carotenoid energy transfer (Ma et al., 2003), Chl-carotenoid charge-transfer (Holt et al., 2005), and/or Chl-Chl charge transfer (Miloslavina et al., 2008). For qE, Dall'Osto et al. (2017) recently concluded that one mechanism, occurring in trimeric LHCII, depends on zeaxanthin but not lutein, and does not involve charge transfer (qE1). A second mechanism occurs in monomeric LHCII, depends on zeaxanthin and lutein, and involves formation of a carotenoid cation radical (qE2). The signal that has been attributed to a Chl-carotenoid interaction or to a carotenoid cation radical in previous ultrafast spectroscopy experiments has been revisited in a study by (van Oort et al., 2018) on isolated LHCII. The authors stress the importance of controlled ultrafast spectroscopy experiments at different laser power/repetition rates and of reporting this information to assess whether strong laser pulses led to the artefactual formation of Chl-carotenoid species. However, due to the short-lived nature of these species (~10 ps) combined with their slow formation (hundreds of picoseconds), the use of strong laser pulses enables their observation and therefore should not be considered an artefact (G. R. Fleming, personal communication). The role of carotenoids in NPQ has thus been a matter of discussion, i.e., whether xanthophyll molecules are excitation-energy traps ("quenchers") or facilitate dissipation indirectly by changing the protein-chlorophyll environment ("allosteric regulators"), with recent findings supporting both possibilities: energy transfer between Chl and carotenoid (Liguori et al., 2017) or charge transfer between Chl and carotenoid (Park et al., 2017) and zeaxanthin acting between complexes rather than internally (Xu et al., 2015), see also discussion by Kress and Jahns (2017).

Across photosynthetic lineages, however, both the molecular players involved in NPQ processes and the organization of pigment-protein complexes differ (for reviews, see Magdaong and Blankenship, 2018; Niyogi and Truong, 2013). It is interesting that some mechanisms converged in terms of involving carotenoids, and it is therefore possible that the routes for de-excitation of excited Chl are similar. In the future, questions of dynamics and efficiency should be explored: can all routes for Chl de-excitation occur at the same time? Under which conditions is one route promoted over others? How much energy is dissipated through these different routes? How much excitation energy is going to heat dissipation? Using a revised NPQ parameter is necessary to address questions of quantum yield of different NPQ routes (Ahn et al., 2009). Simulating excitation energy routes will help tackle these questions that have thus far been addressed using either the lake model (assuming infinite diffusion) or the puddle model (assuming local diffusion). Amarnath et al. (2016) proposed a new model that takes into account the diffusion length of excitation energy as determined by the extent of quenching and thereby improves predictions of fluorescence lifetimes in a given quenching condition; see also Morris and Fleming in the present issue.

### 1.3. When all else fails? Loss of function of the D1 subunit of PSII

The expression "too much of a good thing" is often used to refer to photosynthetic organisms' relationship to light. Too much absorbed light, particularly in the presence of other stress factors, can lead to damage and eventually cell death (Barber and Andersson, 1992). PSII is especially sensitive to inactivation because it generates oxidants. Inactivation is caused by reactive oxygen species (ROS) either at the donor side of PSII, where oxygen is released, or at the acceptor side of PSII where plastoquinones accept electrons generated by water oxidation (Aro et al., 1993). It had long been thought that ROS are unintended, damage-causing by-products of photoreactions. This idea is, however, being revised (across all fields, not only plant biology) as ROS are becoming recognized as signals that regulate gene expression (Foyer et al., 2017; Kim et al., 2012), and in the case of PSII, inhibiting its repair (Nishiyama et al., 2006; Takahashi and Badger, 2011). Furthermore, the view that D1 loss of function is due to increased damage (potentially *when all* other photoprotective mechanisms *fail*) has been critically discussed by Adams et al. (2013) who proposed regulated repression of photosynthetic genes by increased source or decreased sink activity as a cause of photoinhibition. Loss of function of D1 would thereby prevent superoxide and singlet oxygen formation and diminish general cellular damage (Adams et al., 2006). In addition it would indirectly prevent damage to PSI by limiting electron transfer to PSI (Tikkanen et al., 2014).

Non-functional PSIIs result in quenching of chlorophyll fluorescence (qI component, see above). The following factors involved in repair/turnover of PSII cores have thus been identified by screening for enhanced qI or altered photochemical quenching: two thylakoid membrane proteins (Low Quantum Yield of PSII, LQY1; Lu et al., 2011; Hypersensitive to High Light, HHL1; Jin et al., 2014) and a lumenal protein (Maintenance of Photosystem II under High light, MPH2; Liu and Last, 2017). In addition, Rühle et al. (2018) recently reviewed the photosynthesis-related factors that were identified using chlorophyll fluorescence. According to Sarvikas et al. (2010), inactivated centers would marginally contribute to energy dissipation as heat and photoprotection, while other authors have suggested that inactivated D1 are photoprotective (Lee et al., 2001).

## 2. qH, a form of sustained quenching that is photoprotective

I review here what is known about a form of antenna quenching that is slow to relax (sustained) and is photoprotective. We named this component, qH, to distinguish it from qI (Malnoë et al., 2017); in analogy to these letters' respective positions in the alphabet, the photoprotective quenching "H" comes before quenching "I" that is due to loss of PSII core function (and possibly other yet-to-be-discovered slowly relaxing photoinhibitory NPQ processes). A sustained ΔpH-independent form of NPQ has been described in evergreens, for example (Demmig-Adams et al., 2014), and we are now uncovering the molecular players involved in qH, which is a good candidate for the latter mode of photoprotection. I would like to emphasize here that the introduction of "qH" is not to add yet another quenching component to a list of quenching components that might already be too long! The intention instead is to raise awareness that the slowly relaxing "qI" component comprises multiple processes some of which photoprotective and occurring in the antenna. It is well possible that the underlying mechanism responsible for non-photochemical fluorescence quenching by qH is the same as that of qE or qZ, but that the molecular players that modulate this dissipative state differ and might provide for adaptation to the range of conditions photosynthetic organisms experience.

### 2.1. Molecular players of qH

To obtain molecular insights on slowly relaxing quenching processes, Brooks et al. (2013) mutagenized a qE-deficient line (PsbS mutant) and isolated a mutant with high and slowly reversible NPQ. The mutated gene was named suppressor of quenching 1 (*SOQ1*) to describe its function. When present, SOQ1 negatively regulates (suppresses) a form of slowly reversible quenching (qH). SOQ1 is a chloroplast-localized thylakoid membrane protein of 104 kDa that contains multiple domains, including a haloacid dehalogenase-like hydrolase (HAD) domain on the stromal side of the thylakoid membrane, a transmembrane helix, a thioredoxin-like (Trx) domain, and a β-propeller NHL repeat (named after NCL-1, HT2A and Lin-41) on the lumenal side of the thylakoid membrane. While the stromal domain is dispensable for SOQ1 to negatively regulate qH, the lumenal domains are required (Brooks et al., 2013). The nature of the thioredoxin-like domain begs the question of whether qH is redox-regulated. Whether this domain is redox active and may act by reducing or oxidizing its target(s) remain to be determined. The lumenal space is an oxidizing environment (Buchanan and Luan, 2005). What are the electron donors and acceptors for redox modulation of lumenal proteins, and how do they get to the lumenal space (see Järvi et al. (2013) for a review of candidates)? On the other side of the membrane, the stromal HAD domain is dispensable for SOQ1's ability to negatively regulate qH, as demonstrated by the wild type NPQ phenotype of the *soq1* mutant complemented with the truncated SOQ1-deltaHAD (Brooks et al., 2013). As the HAD domain does not play a role in negatively regulating qH, it might, conversely, positively regulate qH. Under stress conditions when qH is active, the negative regulation of qH by SOQ1 should be alleviated, and this might occur through its HAD domain. This would mean that the HAD domain has an inhibiting effect on SOQ1 function. The topology of SOQ1, with domains on both sides of the membrane, raises the attractive possibility of a putative metabolic sensor in the stroma that would regulate the function of the lumenal domain to fine-tune light harvesting for metabolic needs. Rather than feedback de-excitation through pH sensing, the latter response might involve feedback from stromal redox power and metabolism.

qH is independent of known factors required for other types of NPQ, such as PsbS, zeaxanthin formed from violaxanthin, ΔpH formation, or the STN7 kinase (Brooks et al., 2013). We mutagenized the double mutant lacking both PsbS and SOQ1 to assess molecular players required for qH. We found that the plastid lipocalin, LCNP, is required for qH to occur (Malnoë et al., 2017). LCNP is a soluble protein of 29 kDa localized in the thylakoid lumen, whose expression is increased during abiotic stress such as drought and high light (Levesque-Tremblay et al., 2009). Based on domain conservation, it is an eight-stranded antiparallel beta sheet that forms a barrel with high affinity for small hydrophobic molecules. In the genome of *Arabidopsis* and other land plants, there is another true lipocalin named temperature-induced lipocalin (TIL) (Frenette Charron et al., 2002). TIL is localized to different cell membranes and organelles (depending on growth conditions) but not to the chloroplast (Charron et al., 2005; Hernández-Gras and Boronat, 2015). TIL and LCNP play a role during abiotic stress and have overlapping functions in protecting against lipid peroxidation (Boca et al., 2014). The mode of action of plant lipocalins is unknown, i.e., to which molecules they bind and whether they have enzymatic activity. Two lipocalin-like proteins are involved in photoprotection through the xanthophyll cycle: violaxanthin de-epoxidase (VDE) and zeaxanthin epoxidase (ZEP) (Bugos et al., 1998). Compared to true lipocalins, VDE and ZEP have additional N-ter and C-ter domains, and have partial conservation of the structurally conserved regions of the lipocalin fold (Charron et al., 2005). So far, based on the NPQ phenotype of mutant combinations or addition of a reductant (dithiothreitol) that inhibits VDE activity, we can infer that LCNP would not bind violaxanthin, zeaxanthin, or lutein. However, we cannot totally exclude that qH relies on zeaxanthin formed from *β*-carotene, especially as Demmig-Adams et al. (1989) found a correlation between a decrease in *β*-carotene concentration and an increase in sustained NPQ in shade-grown evergreen leaves exposed to experimental high light. Determining the hydrophobic ligand/substrate of LCNP should prove valuable to decipher the mechanism of qH. Our present working hypothesis is that LCNP modifies a molecule in the vicinity or within the antenna proteins, thereby triggering a conformational change that converts antenna proteins from a harvesting to a dissipative state (Fig. 2). It remains to be elucidated whether this dissipative state is due to a charge-transfer or energy-transfer among chlorophylls and/or xanthophylls, and which specific molecules are involved in this mechanism.

We know that qH occurs in the peripheral antenna of PSII because qH is absent in a mutant background lacking chlorophyll *b* (Malnoë et al., 2017) and because minimal fluorescence $F_o$ (or $F_o'$) decreases when the corresponding quenching is activated in a *soq1* mutant (Brooks et al., 2013) or after a cold and high light treatment in wild type (Malnoë et al., 2017; see Section 2.4 for more comments on fluorescence parameters). The peripheral antenna of PSII is composed of the light-harvesting chlorophyll-binding Lhcb proteins, also referred to as LHCII, that is divided into minor (Lhcb4, 5, 6 or CP29, 26, 24, respectively) and major (Lhcb1, 2, 3) complexes. These proteins are organized mostly into oligomers, such as the trimeric form for the major LHCII, or monomeric form for the minor LHCII proteins that link trimeric LHCII proteins to dimeric PSII core complexes forming PSII–LHCII supercomplexes (Ballottari et al., 2012). The next question concerns in which specific antenna qH is localized (i.e. in trimeric or monomeric complexes) and whether or not oligomerization and/or aggregation is required. LCNP was found by mass spectrometry analysis to localize to the stroma lamellae and grana margin (Tomizioli et al., 2014). While LCNP localization might differ under favorable versus stress conditions, it is also possible that the lumenal space is too small for LCNP to diffuse around and form quenching sites within the grana. It might be that the formation of strong quenchers in the LHCII at the grana margin could quench excitation energy received by LHCII within the grana core.

*LCNP* and *SOQ1* genes are conserved among all land plants with sequenced genomes. Whether these genes are conserved across algal genomes is not clear; there is partial domain conservation in some algal lineages for *SOQ1* (Brooks, 2012) but an absence of homologs for *LCNP* (Charron et al., 2005). However, since the lipocalin superfamily is known to have poor sequence identity and high structural similarity

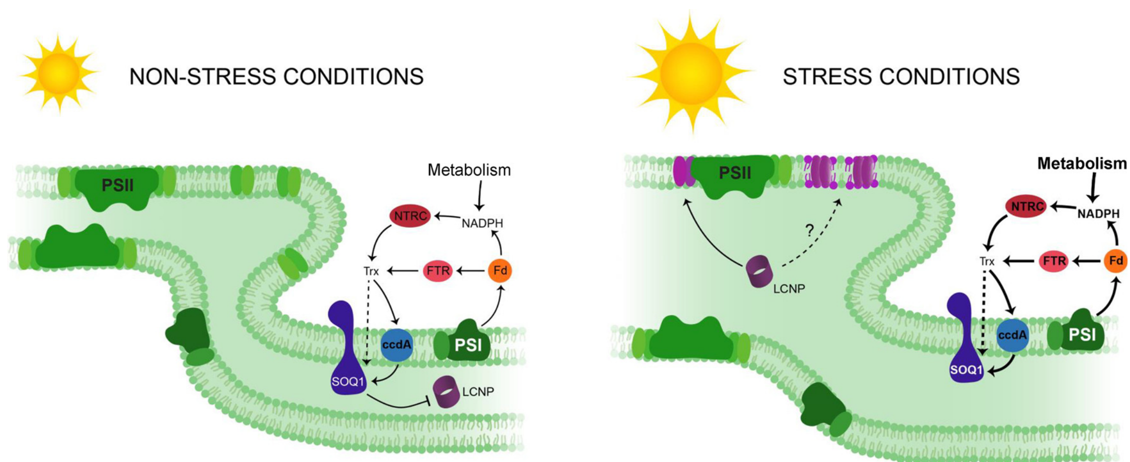


**Fig. 2. Model for qH.** Under non-stress conditions, SOQ1 inhibits LCNP activity. Under stress conditions, such as cold and high light, LCNP is active and quenching sites indicated by purple color are produced in the peripheral antenna of PSII. The formation of quenching sites may be directly mediated by LCNP (solid arrow) or indirectly (dashed arrow) through LCNP modification of LHCII hydrophobic environment. Redox regulation of qH might occur through stromal electron donors such as ferredoxin (Fd) by the ferredoxin-thioredoxin (Trx) reductase pathway (FTR) and/or the nicotinamide adenine dinucleotide phosphate (NADPH) thioredoxin reductase C pathway (NTRC). Thicker arrows represent more reducing power under stress conditions that would potentially alleviate SOQ1 inhibition of LCNP. Lumenal space expansion could also enable access for LCNP to LHCII. Adapted from (Brooks et al., 2013).

(Lakshmi et al., 2015), it is possible that *LCNP* is contained in algal genome and that the quenching mechanism associated with these genes, and its regulation, is broadly conserved, at least within the green lineage.

### 2.2. Regulation of qH

In plants grown under controlled laboratory conditions with standard continuous low light intensity of 120 μmol photons $m^{-2} s^{-1}$ 10 h day/14 h night, qH does not seem to occur, as judged by the similar fluorescence parameters in wild type versus the *lcnp* mutant. Wild type and *lcnp* plants grown under these conditions, and challenged to high light intensities (> 1000 μmol $m^{-2} s^{-1}$) for 10 min, do not show any differences in NPQ kinetics (Malnoë et al., 2017). However, the *soq1* mutant displays sustained NPQ, suggesting that SOQ1 functions to prevent qH under non-stress conditions (Fig. 2). In contrast, we found that qH operates in wild type under stress conditions, such as cold temperatures and high light intensity (Malnoë et al., 2017), indicating that SOQ1 negative regulation is overridden somehow under the latter conditions. One hypothesis is that LCNP becomes oxidized and activated under stress conditions, and that SOQ1 activity and/or quantity is not sufficient to inactivate LCNP under these conditions. Interestingly, LCNP is located in the thylakoid lumen, where the SOQ1-Trx domain is located; LCNP contains seven conserved cysteines and homologs of LCNP present both inter and intra molecular disulfide bridges (Urade et al., 1995; Yang et al., 1994). A possible biochemical interaction between SOQ1 and LCNP is further substantiated by the altered electrophoretic mobility of LCNP in the *soq1* mutant. Identifying the potential post-translational modification(s) of LCNP might be key to understand the regulation of LCNP activity by SOQ1.

Although LCNP was found in the soluble fraction of the thylakoid lumen (Levesque-Tremblay et al., 2009), we identified a role for LCNP in a quenching mechanism occurring in the peripheral antenna of PSII at the thylakoid membrane (Malnoë et al., 2017), which makes it likely that LCNP localizes to the membrane depending on light conditions. Membrane association of TIL was shown to be mediated by a proline-rich motif (Hernández-Gras and Boronat, 2015) that is partially conserved in LCNP. Under conditions in which qH is operating, we would expect LCNP to associate with the membrane. Furthermore, homologs of LCNP can dimerize (Bhatia et al., 2012), and so does VDE (Arnoux et al., 2009). As this dimerization is important to accommodate the ligand or substrate of these proteins, it is reasonable to propose that LCNP functions as a dimer. Conditional recruitment of LCNP to the thylakoid membrane and/or potential dimerization of LCNP might be regulated by SOQ1 since these properties would be essential for LCNP function and SOQ1 is a negative regulator of qH.

In the future, it will be interesting to explore how qH is regulated by the distribution of these molecular players in the thylakoid space and by their stoichiometry relative to each other and to LHCII. As SOQ1 is enriched at the grana margin (Brooks et al., 2013), does LCNP only act there? What is the trigger for qH induction? We do know that acidification of the lumen induces qE (Briantais et al., 1979), a reduced plastoquinone pool activates qT (Allen et al., 1981), the NADPH/ATP ratio fine-tunes cyclic electron flow (CEF; Alric et al., 2010), CEF is enhanced under reducing conditions (Takahashi et al., 2013), light quality regulates orange-carotenoid-protein activity in cyanobacterial NPQ (Wilson et al., 2006), and that calcium (Petroutsos et al., 2011), blue (Petroutsos et al., 2016) and UV light (Allorent et al., 2016) regulates expression of *Chlamydomonas* qE molecular players (Allorent and Petroutsos, 2017).

Finally, it will be interesting to understand how qH is sustained. This quenching component is slow to relax: a 10-min light induction of qH in the *soq1* mutant takes 3 h in the dark for relaxation back to the initial fluorescence level. What factors (gene expression, enzyme activity) does the relaxation of qH require? How can we accelerate it or slow it down?

### 2.3. Possible physiological significance

Sustained energy dissipation has been reported previously in comparative ecophysiological studies and found to be particularly prevalent in evergreens (Demmig-Adams and Adams, 2006). In contrast to deciduous species, which shed their photosynthetic leaves and needles during unfavorable conditions such as hot, dry summers or freezing winters, evergreens retain their green tissues for many years and throughout suboptimal growth seasons.

Two forms of sustained NPQ have been described: one relying on continuous maintenance of a trans-thylakoid ΔpH under cold temperatures in low light or darkness and intermittent freezing days in the fall (Gilmore and Björkman, 1995; Verhoeven et al., 1998) and the other ΔpH-independent (Demmig et al., 1987; Verhoeven et al., 1999). The first one is PsbS-dependent, and therefore a form of sustained qE. According to current terminology, the second might be classified as qZ and/or qH. Both ΔpH-dependent and independent processes are closely

associated with overnight zeaxanthin retention (Adams and Demmig-Adams, 1995; Demmig-Adams and Adams, 2006).

LCNP is highly expressed under high light or drought conditions. LNCP-overexpressing lines exhibit increased resistance to drought stress (Levesque-Tremblay et al., 2009) and the *soq1* mutant displayed increased survival in response to cold and high light stress (Malnoë et al., 2017). It is thus likely that qH plays an important role in protection against a range of abiotic stresses. In the future, it will be interesting to test the potential involvement in qH of genes whose expression has been positively correlated with the presence of sustained quenching in evergreens, such as genes from several distant relatives of the LHC protein family encoding the early light-induced proteins, ELIP, or high-light-induced proteins, HLIP (Zarter et al., 2006).

### 2.4. $F_v/F_m$, not a reliable assessment of loss of PSII core

The parameter $F_v/F_m = (F_m - F_o)/F_m$ provides a rapid way to assess plant health, but caution should be used (Adams and Demmig-Adams, 2004; Murchie and Lawson, 2013) as $F_v/F_m$ is often misinterpreted as a specific indicator of PSII photoinactivation due to D1 damage. Rather, $F_v/F_m$ represents the quantum yield of PSII that will be low not only when the PSII core is inactivated but also in the presence of processes competing with charge separation (thus by definition photoinhibiting), such as slowly relaxing NPQ. In other words, thermal dissipation and loss of PSII core function both result in $F_v/F_m$ decreases. The open-source photosynthesis instrument MultispeQ (Kuhlgert et al., 2016), designed for large-scale and widespread use, measures a new NPQ parameter to address this issue especially in the field where extensive dark adaptation are not practical (Tietz et al., 2017).

Consequently, $F_v/F_m$ is not an appropriate way to specifically assess loss of PSII core function if sustained quenching is also present. *soq1* exhibits a lower $F_v/F_m$ than wild type (Col-0) but less lipid peroxidation (Malnoë et al., 2017). The relative contributions of decreased PSII photochemistry or energy transfer to PSII cores versus thermal dissipation can be assessed from the yields of minimal ($F_o$) and maximal ($F_m$) fluorescence yields (Demmig and Björkman, 1987). Low $F_v/F_m$ can be due to a high $F_o$ as a result of either PSII core inactivation or antenna detachment; low $F_v/F_m$ can also be due to both low $F_o$ and $F_m$ in the presence of qH (Fig. 3). As several minutes are sufficient to relax qE, whereas several hours are required to relax qH, dark-adaptation for 10 min can reveal $F_o$ and $F_m$ quenching due to sustained energy dissipation.

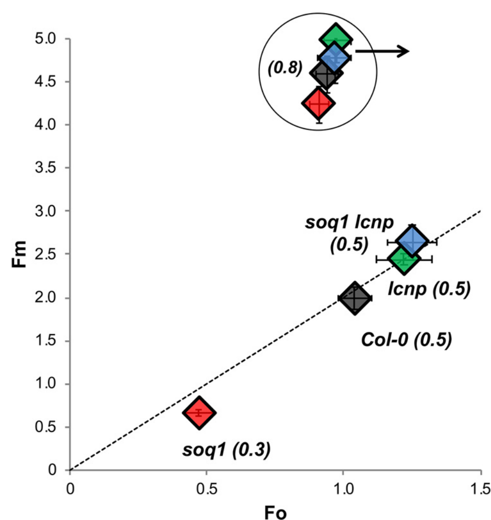


**Fig. 3. Caution regarding the $F_v/F_m$ parameter.** $F_m$ vs $F_o$ and $F_v/F_m$ value displayed in parenthesis before treatment (circled) and after a mild cold and high light treatment (8 h at 12 °C and 1070 μmol photons $m^{-2} s^{-1}$). The dashed line represents fluorescence values that lead to $F_v/F_m = 0.5$. Data represent means ± SD (n = 4 individuals). Col-0 and *lcnp* mutants have similar $F_v/F_m$ but *lcnp* mutants have decreased $F_m$ and increased $F_o$ (arrow) whereas Col-0 has mostly decreased $F_m$.

Studies on D1 degradation by FtsH (VAR2) and regulation through phosphorylation by the kinase STN8 (Kato and Sakamoto, 2014) further demonstrate that $F_v/F_m$ can be uncoupled from D1 accumulation and inactivation. After an experimental high light treatment, the double mutant *var2 stn8* exhibited higher $F_v/F_m$ than *var2*, but greater $H_2O_2$ production and less D1 accumulation than *var2* (Kato and Sakamoto, 2014). This finding indicates that phosphorylated PSII would result in more quenching than its non-phosphorylated counterpart and merits further investigation. Correlations among dark-sustained forms of D1 protein phosphorylation, fluorescence quenching, $F_v/F_m$ decreases, and zeaxanthin accumulation have previously been reported for an overwintering conifer (Ebbert et al., 2005).

### 2.5. Ways of differentiating between photodamage and photoprotection

Much of the focus in understanding photoprotection by thermal energy dissipation has been geared towards identifying molecular players and mechanisms. It is often assumed that NPQ is photoprotective but, as described above, it is not that simple. Not all NPQ processes are photoprotective, and the relative contribution of those that are is not well understood (Lambrev et al., 2012).

(Ruban and Murchie, 2012) proposed to use chlorophyll fluorescence to determine photoprotectiveness of NPQ with the $qP_d$ parameter, which stands for photochemical quenching (qP) in the dark immediately after an illumination period. The light intensity at which PSII photoinactivation starts occurring can be defined as the point when $qP_d < 1$ (Ruban, 2016). During a step-wise exposure to a series of increasing light intensities, the latter parameter is calculated based on the assumption that measured or actual minimal fluorescence in the dark ($F_o'_{act}$) deviate from corrected minimal fluorescence ($F_o'_{calc}$) solely due to PSII photoinactivation. Whether or not this assumption is justified remains to be determined.

Apart from chlorophyll fluorescence, several other methods have been established to directly determine loss of PSII function. Low levels of the PSII core protein D1 are interpreted as a sign of damage (Aro et al., 1993). However, D1 is an abundant protein with a high turnover, and its quantification can be challenging. Lincomycin, an inhibitor of protein synthesis in the chloroplast (D1 is encoded in the chloroplast genome), is used to block D1 repair and study D1 inactivation. Non-functional/damaged D1 can accumulate and biochemical quantification may thus miss functional impairment. Another sign of photodamage is modification of proteins, such as by protein carbonylation. The OxyBlot kit derivatizes carbonyl groups with a chemical and uses a primary antibody against those derivatized groups; the difficulty here is to prevent additional oxidation during the preparation. Lipids are another target of damage. Havaux et al. (2006) developed a technique that images *in vivo* lipid peroxidation. This technique has the advantage to be non-destructive. Awad et al. (2015) found that lipid peroxidation correlates well with protein carbonylation. The slow spontaneous decomposition of peroxidized lipids emits a faint light; a high level of luminescence corresponds to a high level of lipid peroxidation (Fig. 4). This approach has been used to demonstrate that NPQ is photoprotective (Havaux and Niyogi, 1999).

As an alternative to measuring oxidative damage by ROS, direct assessment of different ROS was developed using either dyes such as diaminobenzidine or nitroblue tetrazolium, fluorescence probes such as the Singlet-Oxygen Sensor Green (see for example (Lu et al., 2011), for use of these compounds), or by electron paramagnetic resonance spectroscopy using spin probes (Fufezan et al., 2002). Using the latter approach, (Roach and Krieger-Liszkay, 2012) demonstrated that more ROS are generated in the *npq4* mutant lacking PsbS.

Direct measurement of electron-transfer reactions to assess functionality of photosystems would be a method of choice as it is non-

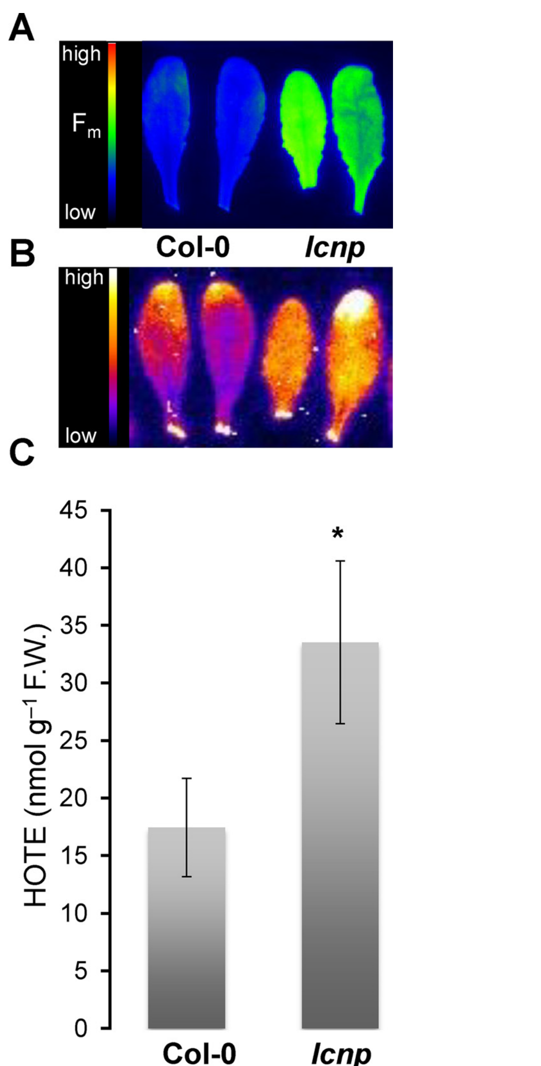


**Fig. 4. Lipid peroxidation as a measure of photoprotection.** When qH is active (Col-0 compared to qH inactive in *lcnp*), less lipid peroxidation are produced. Leaves were exposed to a cold and high light treatment for 3 h at 6 °C and 1500 μmol photons $m^{-2} s^{-1}$. Representative images of (**A**) maximum chlorophyll fluorescence, $F_m$, after a 10 min dark acclimation to relax qE and (**B**) autoluminescence originating from lipid peroxides. **C**) Quantification of lipid peroxidation expressed as HOTE level. Data represent means ± SD (n = 3 samples of pooled leaves from six individuals). Asterisks mark significant difference relative to Col-0 at $P < 0.05$ by Student's *t*-test. Adapted from (Malnoë et al., 2017).

invasive. *In-vivo* spectrophotometry measures light-induced absorption changes at different wavelengths, probing electron transfer through the different thylakoid membrane complexes (see, e.g., Bailleul et al., 2010 for a review on electrochromic shift and Joliot and Joliot, 2005 for PSI kinetics). Finazzi et al. (2004) used the latter approach to characterize a form of reaction-center quenching. Other methods include measuring oxygen evolution and $CO_2$ fixation under non-photorespiratory conditions, which correlate linearly with electron transfer (Genty et al., 1989; Harbinson et al., 1990). However, due to the gaseous nature of these molecules, achieving optimal, air-tight experimental conditions can be "something of an art" (Johnson and Murchie, 2011).

## 3. Manipulation of light-energy harvesting and thermal dissipation to improve crops yields

One of the proposed ways to optimize photosynthetic yield is to manipulate light harvesting and thermal dissipation (Zhu et al., 2010). Other approaches that are actively explored include optimizing carbon fixation by Rubisco (see the perspective by Ort et al., 2015). The relaxation of NPQ can sometimes be slower than the dynamics of light-intensity fluctuations in nature. Under moderate light conditions that promote rapid plant growth, qE is usually the major component of NPQ, but qZ, qH, and qI become more prominent during chronic light stress. Because NPQ decreases the quantum yield of photosynthesis (Demmig-Adams and Adams, 1992), the sustained engagement of slowly reversible NPQ components in fluctuating light has the potential to result in decreases in carbon gain by a plant canopy (Zhu et al., 2004). A decrease by 13–32% (depending on temperature) in carbon gain has been predicted to be due to slow relaxation of NPQ. Speeding up NPQ relaxation could therefore substantially increase photosynthetic energy conversion and was predicted to be a viable approach to increase plant productivity in favorable growth environments where there is less need for photoprotection (Long et al., 2006; Zhu et al., 2004).

### 3.1. Increasing NPQ capacity and accelerating zeaxanthin-removal kinetics

Manipulating the fast quenching response was recently demonstrated to increase leaf biomass by up to 20% (Kromdijk et al., 2016). The authors overexpressed the genes encoding for PsbS, and for the xanthophyll cycle enzymes VDE and ZEP (VPZ transformant) in the model crop tobacco. Upon light fluctuations, NPQ was both induced and relaxed more rapidly, thereby better matching fluctuations and promoting protection while preventing potential 'waste' of energy (Ruban, 2017). As plant growth and development are strongly modulated by the cellular redox balance, enhanced biomass production could also be related to increased ROS production, in plant lines with accelerated NPQ relaxation, due to increased excitation pressure (see Demmig-Adams et al., 2018).

Next, it will be important to test whether such increased leaf biomass translates to an increase in seed biomass. In the green revolution, increase of grain-crop yield has been achieved instead by inhibiting growth and biomass production via dwarf mutants (Demmig-Adams et al., 2017). Similar engineering of NPQ is under way for crops such as rice, cassava, sorghum, cow pea, maize, soybean and wheat (http://ripe.illinois.edu). It was recently shown that overexpression of PsbS in rice leads to both higher biomass and 26% higher grain yield (Hubbart et al., 2018). It was also found that enhanced qE by overexpression of PsbS in tobacco resulted in partial stomata closure that, in turn, optimized water use efficiency (Glowacka et al., 2018).

### 3.2. Dissipation of the trans-thylakoid pH gradient and the $K^+/H^+$ antiporter

Ion transporters localized in the thylakoid membrane have recently been identified. KEA3 is a $K^+/H^+$ antiporter that exchanges protons from the lumen for potassium ions from the stromal space. The *kea3* mutant shows enhanced qE as a larger proton gradient builds up in the absence of KEA3 (Armbruster et al., 2014). Overexpressing KEA3 would thus be expected to lead to faster dissipation of qE, therefore better matching light fluctuations, and possibly enabling higher biomass yield as was shown for the VPZ transformant described above. Indeed, Armbruster et al. (2014) found that, under fluctuating light, the *kea3* mutant displays a lower quantum yield of PSII and lower $CO_2$ fixation than wild type.

Another aspect of the impact of the trans-thylakoid membrane potential on photosynthetic output was described by (Davis et al., 2016). The latter authors found that a high $\Delta\Psi$ (difference in electric potential) across the thylakoid membrane prevents charge recombination at the level of PSII, thereby decreasing potential inactivation. Therefore, efforts aiming at manipulating $\Delta\Psi$ could mitigate loss of PSII function and enable higher biomass yield (Davis et al., 2017).

### 3.3. Truncated LHCs

A strategy to optimize conversion of absorbed photon to biomass has been to ensure that light distributes homogenously within a dense culture of photosynthetic organisms by artificially decreasing (truncating) their light harvesting antenna (Kirst and Melis, 2018; Ort and Melis, 2011). This approach has been successful in increasing biomass yield in algae (Mussgnug et al., 2007), cyanobacteria (Kirst et al., 2014), and recently crop plants (Kirst et al., 2017, 2018). The possibility that altered redox conditions may play a role in growth modulation of photosynthetic organisms by altered light-harvesting should also be explored.

Many crop plants are rapidly-growing annuals that evolved in open habitats, but today are often densely grown in agricultural contexts. One idea put forth in the smart canopy model described in (Ort et al., 2015) is to manipulate leaf angle to allow better light distribution throughout the plant canopy. It might also be worthwhile to look into natural strategies that evolved to optimize light collection and thermal dissipation in plants growing densely in nature. Furthermore, crop plant systems experience pronounced fluctuations in incident light intensity (Kaiser et al., 2018), and photosynthesis research is gearing up towards understanding such dynamics at the molecular level (Thormahlen et al., 2017).

## 4. Conclusions

The study of photoinhibition and, more specifically, of non-photochemical fluorescence quenching associated with photoinhibition has led to confusion between the original definition from biophysical studies (photoinhibition = decreased $CO_2$ fixation) compared to a newer definition from molecular studies (photoinhibition = D1 damage). The term qI was coined for sustained quenching due to photoinhibition and encompasses non-photochemical quenching processes that are sustained and decrease $CO_2$ fixation. Not all of qI is necessarily photoprotective (although as discussed in Section 1.3, there is the notion that photoinactivated D1 may be photoprotective). To differentiate qI from photoprotective sustained antenna quenching, we termed the latter quenching qH; in answering the question posed in the title, qH is thus a process that is both photoinhibitory and photoprotective. The plastid lipocalin LCNP is required for qH to occur, and the suppressor of quenching1 SOQ1 prevents qH from occurring under non-stress conditions. Future work will explore the physico-chemical mechanism of qH, its exact location in the peripheral antenna of PSII, its regulation and synergy with other quenching components, and possible occurrence in evergreens. Manipulation of light harvesting and its regulation by photoprotection might be key to increase bioenergy and food crop yields.

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