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Review

Thylakoid protein phosphorylation in dynamic regulation of photosystem II in higher plants ☆

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ABSTRACT

In higher plants, the photosystem (PS) II core and its several light harvesting antenna (LHCII) proteins undergo reversible phosphorylation cycles according to the light intensity. High light intensity induces strong phosphorylation of the PSII core proteins and suppresses the phosphorylation level of the LHCII proteins. Decrease in light intensity, in turn, suppresses the phosphorylation of PSII core, but strongly induces the phosphorylation of LHCII. Reversible and differential phosphorylation of the PSII-LHCII proteins is dependent on the interplay between the STN7 and STN8 kinases, and the respective phosphatases. The STN7 kinase phosphorylates the LHCII proteins and to a lesser extent also the PSII core proteins D1, D2 and CP43. The STN8 kinase, on the contrary, is rather specific for the PSII core proteins. Mechanistically, the PSII-LHCII protein phosphorylation is required for optimal mobility of the PSII-LHCII protein complexes along the thylakoid membrane. Physiologically, the phosphorylation of LHCII is a prerequisite for sufficient excitation of PSI, enabling the excitation and redox balance between PSII and PSI under low irradiance, when excitation energy transfer from the LHCII antenna to the two photosystems is efficient and thermal dissipation of excitation energy (NPQ) is minimised. The importance of PSII core protein phosphorylation is manifested under highlight when the photodamage of PSII is rapid and phosphorylation is required to facilitate the migration of damaged PSII from grana stacks to stroma lamellae for repair. The importance of thylakoid protein phosphorylation is highlighted under fluctuating intensity of light where the STN7 kinase dependent balancing of electron transfer is a prerequisite for optimal growth and development of the plant. This article is part of a Special Issue entitled: Photosystem II.

1. Introduction

Plant productivity is a result of many processes that modulate the photosynthetic machinery in order to maintain functional equilibrium despite changes in environmental conditions and the metabolic state of the plant [1]. Changes in light quantity take place on many different time scales; the movements of leaves and clouds may cause rapid changes, the diurnal availability of light energy peaks in the middle of the day and the availability of light is very different on cloudy days as compared to sunny days. Nevertheless, it is not only the availability of light but also the metabolic and developmental states of the plant that set the requirements for the function of the photosynthetic machinery, particularly that of PSII. Regulation of the function of the photosystems needs to be very flexible in order to enhance light energy conversion when photosynthesis limits the metabolism, and conversely, to down-regulate the efficiency of solar energy conversion when excess light hits the leaf. Despite the dynamic regulation of light harvesting, PSII inevitably becomes damaged always when light energy is absorbed and the photosynthetic reactions are taking place [2,3], with the rate of damage increasing with increasing light intensity. In order to maintain the photosynthetic activity, the damaged PSII complexes must be replaced with newly synthesised functional ones in a highly co-ordinated manner (for a review see [4–6]). Indeed, as long as the repair of PSII can keep pace with the damage, no visible symptoms of photoinhibition are developed.

In higher plant thylakoid membrane, there are two major and distinct mechanisms that sense and respond to changes in the energetic balance of chloroplasts. Firstly, several PSII-LHCII proteins become strongly and reversibly phosphorylated depending on the redox state of the intersystem electron transfer chain (ETC) [7–9] and the stromal redox compounds beyond PSI [10–12]. Secondly, the protonation of the thylakoid lumen induces the PsbS- and xantophyll cycle-dependent thermal dissipation of excitation energy (NPQ) (For reviews see [13–15]). Both of these major regulatory mechanisms lead to migrations and reorganisations of the PSII-LHCII complexes along the thylakoid membrane [16–21]. Interestingly, it was recently shown that also the regulation of the xanthophyll cycle has a redox-

Abbreviations: HL, High light (relative to growth light); LHCII, Light harvesting complex II that distributes energy to PSII and PSI according to its phosphorylation and to the amount of thermal dissipation of energy; LL, Low light (relative to growth light); NPQ, Non-photochemical quenching of excess excitation energy; PSI, Photosystem I; PSII, Photosystem II

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dependent component [22]. It is highly conceivable that in natural environmental conditions, the phosphorylation and NPQ mechanisms function in great synchrony and together maintain the energetic balance of the electron transfer reactions and prevent excess photodamage of PSII [23,24].

In this review, we focus on short-term mechanisms underlying the regulation of the photosynthetic light reactions by PSII-LHCII protein phosphorylation, and on the role of such regulatory processes in the maintenance of optimal physiological performance of plants. Long-term acclimation, possibly also influenced by thylakoid protein phosphorylation, or at least by the same kinases [25–27], is not considered here since in light of recent investigations, the phosphorylation related long-term acclimation is a direct consequence from short-term regulation mechanisms. We address the cooperation between the STN7/STN8 kinase pathways and the NPQ in terms of allowing equal distribution of excitation energy to both photosystems despite fluctuations in light intensity. We want to emphasise the need to consider PSII-LHCII protein phosphorylation when resolving the regulatory mechanisms governing the structural and functional modifications of the plant thylakoid membrane. Likewise, the search for other, still unknown mechanisms that function concomitantly with thylakoid protein phosphorylation, is likely to be crucial to eventually lead to proper understanding of the regulation of the photosynthetic light reactions according to innate and environmental cues.

2. Phosphorylation of PSII-LHCII proteins

The major phosphoproteins of the PSII core complex are D1, D2, and CP43 [28], yet the phosphorylation-specific threonine residues of the PsbH and TSP9 proteins are also reversibly phosphorylated [29]. In the LHCII trimer, the N-terminal threonine residues of the Lhcb1 and Lhcb2 proteins are reversibly phosphorylated [30] whereas Lhcb3 is not a phosphoprotein. Three minor chl-binding proteins—Lhcb4 (CP29), Lhcb5 (CP26), and Lhcb6 (CP24)—are monomeric, and of these proteins only Lhcb4 is a phosphoprotein in higher plants [31,32].

The phosphorylation of PSII-LHCII proteins is regulated by the redox state of the electron transfer chain and that of the chloroplast stroma (for a review see [33]) via the STN7 and STN8 kinase-dependent phosphorylation pathways [34,35]. The reversible phosphorylation of PSII core proteins is mainly under the control of the STN8 kinase and unknown phosphatase(s), but the STN7 kinase also has a minor role in PSII core protein phosphorylation [36], particularly at low light [23,37]. The phosphorylation kinetics of the different PSII core proteins varies [23], indicating that there is protein-dependent specificity in the function of the STN7 and STN8 kinases. Even so, the reason for such specificity remains totally unknown. In contrast, the N-terminal threonine residues of the major LHCII phosphoproteins, Lhcb1 and Lhcb2, are phosphorylated solely by the STN7 kinase [34] and dephosphorylated by the constitutively active phosphatase called TAP38 [38] or PPH1 [39]. Here, the role of the Lhcb4 protein phosphorylation is not discussed due to the lack of proper information in regulation of higher plant thylakoid function.

3. Comparison of the PSII-LHCII protein phosphorylation upon state transition and in different light intensities —a classical mechanistic viewpoint

According to the classical state transition theory, the phosphorylated LHCII trimer migrates from PSII in the appressed grana membranes to serve as a light harvesting antenna for PSI in the stroma-exposed membranes, as a response to a change in light quality. Conversely, when LHCII becomes dephosphorylated, the excitation of PSII is again favoured by the movement of LHCII back to PSII [18,40,41]. It is well known that LHCII phosphorylation is a prerequisite for dynamic regulation of relative PSI/PSII excitation under artificially induced state transitions with different qualities of light [23,34,35,42,43]. However, there is no unambiguous evidence to support the movement of P-LHCII to stroma-exposed membranes, as a mechanism behind increased PSI excitation, when LHCII is phosphorylated under low light intensity. Moreover, the theories of P-LHCII movement are based on experiments performed under unnatural light conditions that cannot be found outside the laboratory. Indeed, the 77 K chlorophyll fluorescence emission spectra demonstrate only a minor or no transition to “state 2” upon a shift of leaves from darkness to light or from high light (HL) to low light (LL) [19,23], both shifts inducing a phosphorylation of the LHCII proteins [44]. When the thylakoid membrane was subfractioned in dephosphorylated state (dark incubated spinach leaves) and phosphorylated state (LL treated spinach leaves) of the LHCII proteins, it became evident that from all thylakoid subfractions, only the grana margins behaved according to the traditional view of LHCII phosphorylation-induced state transition [19]. It is important to emphasise that upon LL-induced LHCII protein phosphorylation no “state-1” to “state-2” transition could be detected in the stroma lamellae, as was previously postulated [18,40,41], indicating that the P-LHCII proteins found in the stroma lamellae as a consequence of LHCII phosphorylation do not function there to collect light energy for PSI. On the contrary, rather a small “state-2” to “state-1” transition was observed in the stroma lamellae upon LHCII phosphorylation.

Results described above on the analyses of different thylakoid subfractions [19] challenge the movement of P-LHCII to stroma membranes as a sole mechanism for dynamic redistribution of excitation energy between PSII and PSI, despite the fact that LHCII phosphorylation, or at least an active STN7 kinase, is obligatory for such energy balance to occur [35,42]. Likewise, the assumption that the induction of LHCII phosphorylation occurs in order to change the balance of excitation energy between PSII and PSI might be misleading. Indeed, it is difficult to find any physiological relevance for a need to unbalance the redox poise upon changes in light intensity—fluctuations in the light intensity change the energy supply to the entire photosynthetic machinery but do not, as such, change the need for relative excitation of PSII and PSI. We strongly believe that the reason for the need to regulate the distribution of excitation energy to PSI by LHCII protein phosphorylation/dephosphorylation cascades should be searched from the function of NPQ. Drastic and rapid changes in NPQ as a response to changes in light intensity might strongly influence the excitation of PSII. In order to maintain the redox poise of ETC, also the excitation of PSI needs regulation. Protein phosphorylation of the LHCII trimers likely connects the NPQ to cover the regulation PSI light harvesting as well, thus providing balanced turnovers of both the PSII and PSI reaction centres under changing light intensities [24].

4. The role of LHCII protein phosphorylation and NPQ

White light of different intensities has a strong influence on the extent of PSII-LHCII protein phosphorylation [44], yet in intact leaves the intensity of light has only a minor effect on relative excitation of the PSII and PSI reaction centres [19,23]. Nevertheless, the lack of LHCII phosphorylation in the *stn7* mutant strongly disturbs the redox balance between PSII and PSI, especially under low light when NPQ is relaxed and the light harvesting machinery functions in its full capacity [23,35,43]. Indeed, it seems that the STN7 kinase-dependent phosphorylation of the LHCII proteins is required to provide sufficient energy for PSI under periods of low light intensity. Subsequent increase in light intensity protonates the PsbS protein and turns LHCII to dissipative state [45], thus favouring thermal dissipation of absorbed light energy instead of efficient light harvesting to PSII.

Due to high light intensity and thermal dissipation of most of the excitation energy absorbed by the LHCII system, the LHCII protein phosphorylation becomes dispensable for excitation balance of the two photosystems. It has been unambigously shown that the LHCII

phosphorylation strongly declines when plants are shifted to high light, apparently due to inhibition of the STN7 kinase [10,23,37]. When the light intensity again decreases, LHCII restores its function in efficient light harvesting to PSII. This, in turn, leads to higher excitation of PSII as compared to PSI, and as a consequence reduces the plastoquinone pool, which again activates the STN7 kinase. If, however, the STN7 kinase is missing (the *stn7* mutant), there is no LHCII phosphorylation-dependent enhancement of PSI excitation under the LL period [23]. Such deficiency in balancing the excitation energy between PSII and PSI in the *stn7* kinase mutant leads to constantly changing chloroplast redox state according to the changes in light intensity upon the growth of plants under fluctuating light intensities (Grieco et al., manuscript in preparation).

Even when grown under stable low light condition, which tends to keep the PSII over-excited as compared to PSI, the Arabidopsis *stn7* mutants can, however, compensate for the insufficient excitation of PSI via a still unknown long-term acclimation mechanism and generally no growth penalty or visual phenotype exists [23,35] (Grieco et al., manuscript in preparation). Severe growth phenotype, on the contrary, is typical to the *stn7* mutant grown under fluctuating light where relative over excitation of PSII under LL phase and balanced excitation of PSII and PSI under HL phase are constantly taking turns [23]. The *stn7* mutant cannot keep the redox state of the ETC in balance under fluctuating light conditions and thus the signalling and development of the long term compensation mechanisms are likely to be disturbed with frequent changes in the redox state of the photosynthetic electron carriers [23]. It is thus conceivable that the STN7 kinase and LHCII phosphorylation form a regulatory network together with the NPQ system to optimise the function of the thylakoid membrane despite strong fluctuations in the light intensity. We conclude that the STN7 kinase provides the plants with the means to alleviate strong changes in the redox balance in chloroplasts and thereby allows long term acclimation even under strongly fluctuating light conditions.

5. Model for the role of phosphorylation and NPQ in the dynamics of the PSII-LHCII complexes

The exact nature of the physical forces behind the lateral movements of the PSII-LHCII proteins in the thylakoid membrane still remains unknown and will not be discussed here. However, the phosphorylation of both the PSII core proteins [46,47] and the LHCII proteins [48] as well as the protonation of the PsbS protein [17,20,21] has been shown to have a role in facilitating the migration of the PSII-LHCII proteins along the thylakoid membrane under changing light intensity. Mechanistically, the co-operation between PSII-LHCII protein phosphorylation and NPQ is not yet understood. In Fig. 1, we present a schematic and still hypothetical model based on our own investigations on the *stn7* and *stn8* kinase mutants as well as the NPQ mutants [23,37] and on recent reports from other laboratories focusing on the role of PSII-LHCII protein phosphorylation in thylakoid membrane dynamics [46,49]. Likewise, the recent achievements in understanding the role of the PsbS protein in controlling the migration of the PSII-LHCII protein complexes along the thylakoid membrane upon induction of thermal dissipation of excess energy [17,20,21,50] are taken into consideration in the model (Fig. 1). It should be noted that these events take place inside the highly organised lipid membrane environment. Indeed, the delicate three dimensional structure of the thylakoid membrane [51–55], the lipid composition of the thylakoid membrane [56] as well as their dynamics according to environmental cues are likely to participate in the regulation of excitation energy distribution and redox balance. However, due to the lack of integrative studies, these factors are not introduced into the model.

LHCII protein phosphorylation-based regulation of excitation energy distribution to PSII and PSI is of greatest importance under periods of moderately low light intensity (for details, see the model in Fig. 1 and details in the figure legend). Subsequent increase in light intensity enhances the thermal dissipation of excitation energy by NPQ and speeds up the damage of the D1 protein of PSII. All this occurs together with dephosphorylation of LHCII and phosphorylation of the PSII core proteins [44]. LHCII dephosphorylation prevents the energy flow from LHCII to PSI, and PSII core protein phosphorylation weakens the attractive forces between the subunits of PSII-LHCII supercomplexes thus facilitating the movement of damaged PSII core from grana to stroma membrane for repair (see succeeding section).

6. PSII core protein phosphorylation regulates the PSII D1 protein turn-over

The monomeric PSII complex is composed of more than 20 subunits [57–59]. In plant thylakoid membrane, PSII is mostly present as PSII-LHCII supercomplexes, each composed of a dimeric core complex of PSII, which is associated with two copies of each minor light-harvesting protein, two strongly bound LHCII trimers, and one or more less tightly bound trimer(s) [60–62] PSII-LHCII supercomplex is functionally the most efficient form of PSII [63]. These supercomplexes are organised in arrays and mainly located in the appressed grana membranes [62,64]. However, the assembly of new PSII-LHCII supercomplexes and the repair of photodamaged PSII core occur through the monomeric form of PSII [65]. Thus, the dynamic and reversible oligomerization, monomerization, and reoligomerization of PSII are required for biogenesis of PSII and for the maintenance of PSII activity via the repair cycle. The monomerization process of the PSII-LHCII supercomplex requires lateral migration of different subcomplexes of PSII-LHCII along the thylakoid membrane system. PSII-LHCII complexes form highly organised arrays of supercomplexes in the grana core, and the structure is gradually monomerized via the dimeric state of PSII towards the stroma lamellae [63].

PSII core proteins are maximally phosphorylated under strong light [66–69]. This behaviour of the PSII core protein phosphorylation, together with the notion that phosphorylated D1 is a poor substrate for proteolytic degradation [70–72], initially led to the suggestion that the PSII core protein phosphorylation is required for successful acclimation of plants to high light. The light-induced phosphorylation of the PSII core proteins, especially that of the D1 protein, was then connected to the regulation of PSII protein turnover upon photodamage and the repair of the damaged PSII proteins [4,47,71–74]. The D1 protein is always prone to photodamage when the photosynthetic light reactions are running [2,75]. Rapid dynamic degradation of the damaged D1 protein and *de novo* synthesis and insertion of the new D1 protein into PSII are prerequisites for survival of the water-splitting photosynthetic organisms in a wide range of light intensities (for a review, see[65]). Initially, it was hypothesised that the damaged D1 is marked by phosphorylation, which then functions as a signal for migration of the damaged PSII from the grana to the stroma lamellae, where D1 is dephosphorylated, degraded and the newly synthesised D1 is inserted into PSII [4]. These early theories were based on indirect experimental evidence, due to the lack of access to the kinase mutants at that time.

When the kinase mutants became available, direct evidence was produced for the role of thylakoid protein phosphorylation in regulation of PSII turnover and repair. The first report on the *stn8* and *stn7 stn8* mutants, however, demonstrated that PSII core protein phosphorylation does not significantly affect the photoinhibition of PSII or turnover of the D1 protein [34], challenging the earlier indirect results assigning a specific role for PSII core protein phosphorylation in regulation of PSII D1 protein turnover [47,70]. Due to a discrepancy between the old model and the new results based on the *stn8* mutant, it was agreed that we attempt to reinvestigate the effect of PSII-LHCII protein phosphorylation on D1 protein degradation and turnover by using the *stn7, stn8,* and *stn7 stn8* mutants [37]. First, the range of light intensities in inducing the damage to PSII in control WT plants was

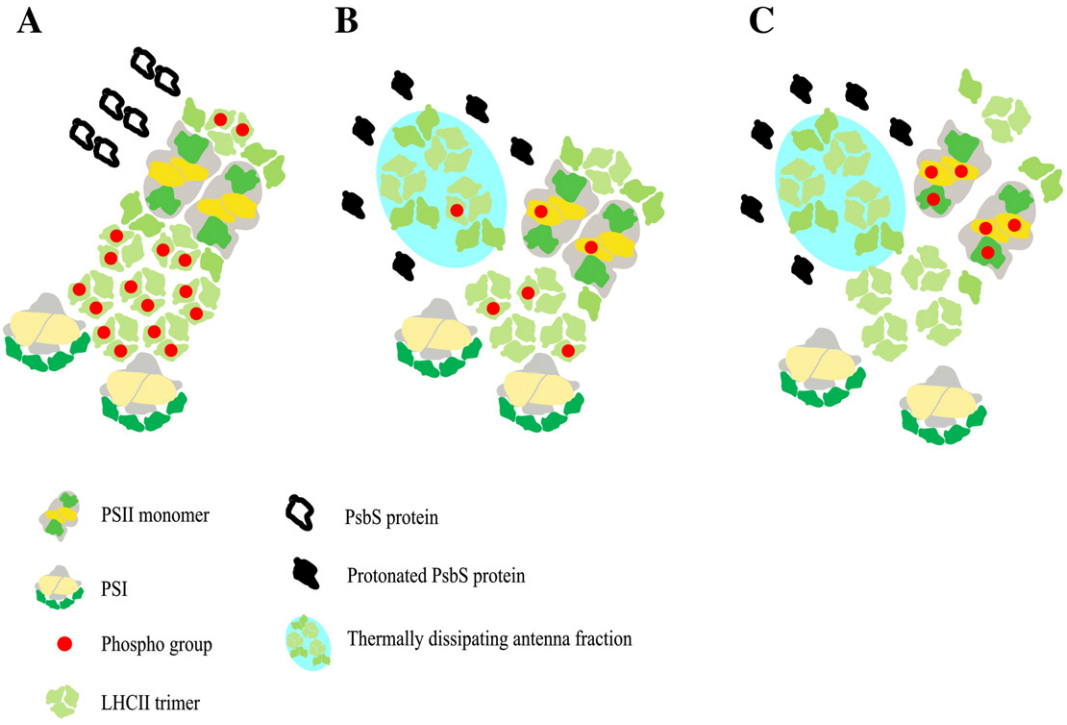

Fig. 1. Interplay between LHCII protein phosphorylation, NPQ and organisation of the chlorophyll protein complexes in the thylakoid membrane, particularly in grana margins, upon changing light intensities. A. Under low light, the Lhcb1 and Lhcb2 proteins in the LHCII trimers are strongly phosphorylated, the PSII core proteins CP43, D1 and D2 are dephosphorylated and the PsbS protein is deprotonated. In this condition the thermal dissipation of excitation energy is minimal and the efficiency of excitation energy transfer from LHCII to the photosystems is maximal. The STN7-kinase-dependent LHCII protein phosphorylation is required for sufficient energy transfer to PSI thus enabling excitation and redox balance between PSII and PSI. B. Upon increase in light intensity the PsbS protein becomes protonated with concomitant rearrangements in the antenna system, which changes the antenna into dissipative state, favouring thermal dissipation of excitation energy over energy transfer to the reaction centres. Gradually the STN7 kinase becomes inhibited and the STN8 kinase becomes more activated leading to dephosphorylation of LHCII and strong phosphorylation of the PSII core proteins. In this quenched state, the PSII-LHCII phosphorylation has no effect on the distribution of excitation energy between the two photosystems. C. Further increase in light intensity speeds up the rate of damage to the PSII complex, which in turn requires an enhancement of the repair cycle of PSII. Under this condition, a strong phosphorylation of the PSII core proteins loosens the attractive forces between the subunits of the PSII-LHCII complexes, allowing fluent migration of damaged PSII to stroma thylakoids for repair. Dephosphorylation of LHCII at high light may help this process to occur. (Model is an adaptation from the models presented in [21,50].

tested, and it turned out that far too high intensities were used by Bonardi et al. [34] to be able to evaluate the difference in the susceptibility of PSII to high light damage between the WT and the *stn* mutant plants. Accordingly, the photoinhibition and D1 protein degradation experiments of the novel kinase mutants were repeated in feasible high light conditions in order to be able to evaluate the role of the STN kinases in the turnover of the D1 protein in PSII.

These experiments clearly demonstrated that during the course of illumination, the degradation of the damaged D1 protein is retarded in the *stn8* mutant and in the *stn7 stn8* double mutant as compared to the WT or the *stn7* mutant [37]. Further investigation revealed that the lack of PSII core protein phosphorylation hampers the disassembly of the damaged PSII supercomplexes in the grana membrane, which is the first step of the PSII repair cycle [37]. As a consequence, the *stn8* and the *stn7 stn8* double mutant accumulate more damaged PSII complexes and generate more oxidative stress under prolonged high light stress than the WT.

The mechanism facilitating the repair of PSII in the WT as compared to the *stn8* and the *stn7 stn8* mutant was found to reside in reversible dynamics of PSII oligomerization, enhanced by PSII core protein phosphorylation, and not directly in the regulation of D1 protein degradation. Indeed, it was shown that the PSII core protein phosphorylation allows fluent migration of photodamaged PSII to stroma-exposed membranes for repair and subsequent detachment of the damaged D1 from the core complex [37]. It has thus become clear that PSII core protein phosphorylation has a role in regulation of PSII turnover (Fig. 2).

More recent experiments have confirmed that PSII core protein phosphorylation increases the fluidity of the thylakoid membrane and thus also improves the mobility of the protein complexes along the thylakoid membrane [46]. Moreover, it was recently shown that the lack of PSII core protein phosphorylation in the *stn8* mutant changes the thylakoid ultrastructure [76], which possibly also hinders the capacity for fluent turn-over of PSII upon photodamage [77] Additionally, it has been demonstrated that the FtsH protease may be located in close proximity to the PSII reaction centres inside the appressed grana stacks [76,78,79]. This clearly indicates that further experimentation is required to understand the still hypothetical changes in the structure of the thylakoid membrane upon high light illumination (Fig. 2). Indeed, many new data are emerging on the structure of the grana, distribution of PSII complexes in the grana, membrane crowdedness, and stress-induced structural changes of thylakoids [51,77,80].

It is worth noting that the *stn8* mutation in rice has now also been analysed (a congress poster [81] and personal communication Choon-Hwan Lee and Krishna Nath). Rice *stn8* mutant revealed a stunted growth phenotype and demonstrated severe problems in the degradation of damaged D1 protein. Retarded D1 degradation and enhanced susceptibility of PSII to high light stress, as compared to WT, were in rice *stn8* mutant even more conspicuously than those recorded for the Arabidopsis *stn8 mutant.*

7. Model for the physiological role of PSII core protein phosphorylation

Physiologically, the phosphorylation of PSII core proteins plays an important role under prolonged high light stress when fluent access of damaged PSII complexes to the repair machinery is required to maintain

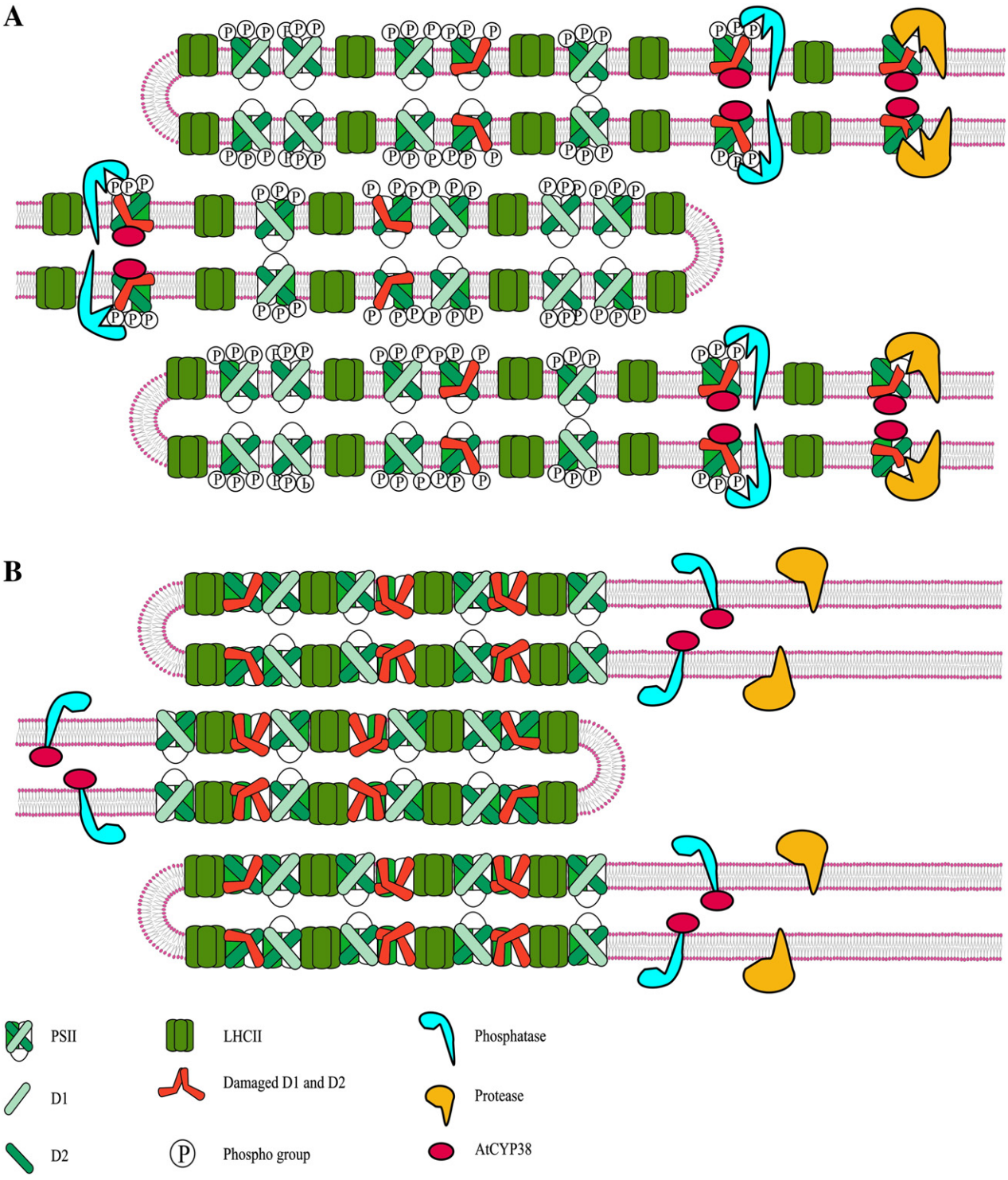

Fig. 2. Role of PSII core protein phosphorylation in repair of photodamaged PSII. A. Photosyntetically active PSII complexes are located in the appressed grana membranes in a tightly packed and highly organised manner. In contrast, the PSII repair machinery, consisting of the D1 phosphatase, the D1 protease and many regulatory and helper proteins, is located in the non-appressed stroma membrane. The repair of damaged D1 protein requires unpacking of the PSII-LHCII structure in the grana and migration of the PSII monomer from grana to stroma membrane for access to the repair machinery. In the presence of PSII core protein phosphorylation the damaged PSII complexes can fluently migrate from grana to stroma membranes for repair (see text for details). B. In the absence of PSII core protein phosphorylation (*stn8* mutants) the PSII core complexes get stuck in the grana and efficient repair is prevented. The accumulation of damaged PSII reaction centres in the grana leads to enhanced photoinhibition and eventually to generation of oxidative stress and uncontrolled oxidative damage to the photosynthetic machinery.

sufficient photosynthesis and to prevent further photo-oxidative damage to the photosynthetic apparatus. In Fig. 2, we present a model explaining our present understanding of the regulation of PSII repair cycle by core protein phosphorylation. The key observation was that the photodamaged PSII complexes accumulate in the grana appressions in the *stn8* kinase mutants. The STN8 kinase-dependent phosphorylation of the PSII core proteins thus facilitates the unpacking of PSII complexes, allowing photodamaged PSII monomers to migrate from grana to grana margins and the stroma lamellae. Upon arrival to these thylakoid regions, the damaged D1 protein is first dephosphorylated by a still

unknown phosphatase and then degraded by the FtsH protease, possibly in co-operation with Deg proteases [82]. It is hypothesised that the damaged PSII monomers, upon arrival in non-appressed thylakoid regions, come into contact with a phosphatase, AtCYP38 immunophilin complex, and this contact is assumed to activate the phosphatase by dismantling the connexion with AtCYP38 [83–85]. In this scheme, the released and activated phosphatase dephosphorylates the damaged D1 protein and makes it susceptible to proteolytic degradation. AtCYP38 immunophilin was recently shown to be a crucial chaperone for co-translational reassembly of the new D1 protein into PSII [84,85] and it is hypothesised that after release from the phosphatase, AtCYP38 directly binds to the PSII complex under repair and guides the co-translational insertion of the *de novo* synthesised D1 protein into the PSII complex. Final proof for the dual function of AtCYP38 (phosphatase inhibitor and immunophilin) in the repair of PSII needs a characterisation of plants with differential mutation of the phosphatase binding domain and the immunophilin domain of AtCYP38.

8. Conclusions

We have presented above the recent developments in unravelling the role of the LHCII and PSII core protein phosphorylation in the dynamic function of plant thylakoid membrane upon changing environmental cues, particularly the light intensity. It can be summarised that the strictly light intensity controlled functions of the STN7 and STN8 kinases enable the energetic balance in the thylakoid membrane under periods of low light intensity and the fluent turn-over of the PSII core protein D1 under high light induce photodamage. Moreover, the STN kinase dependent functions are fully synchronised with the PsbS and xanthophyll-cycle dependent NPQ. Thus the reversible phosphorylation of the PSII-LHCII proteins, together with NPQ, provides the plant with the means to cope with ever changing light environment as well as with other stress factors and limitations of the plant metabolism.

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