Biochimica et Biophysica Acta 1847 (2015) 900–909

Contents lists available at ScienceDirect

Biochimica et Biophysica Acta

journal homepage: www.elsevier.com/locate/bbabio

Review

Photosystem II repair in plant chloroplasts — Regulation, assisting proteins and shared components with photosystem II biogenesis☆

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ARTICLE INFO

Article history:
Received 1 November 2014
Received in revised form 7 January 2015
Accepted 15 January 2015
Available online 21 January 2015

Keywords:
Chloroplast
Light
Photosystem II repair cycle
Biogenesis of PSII
Grana thylakoid
Stroma-exposed thylakoid

ABSTRACT

Photosystem (PS) II is a multisubunit thylakoid membrane pigment–protein complex responsible for light-driven oxidation of water and reduction of plastoquinone. Currently more than 40 proteins are known to associate with PSII, either stably or transiently. The inherent feature of the PSII complex is its vulnerability in light, with the damage mainly targeted to one of its core proteins, the D1 protein. The repair of the damaged D1 protein, i.e. the repair cycle of PSII, initiates in the grana stacks where the damage generally takes place, but subsequently continues in non-appressed thylakoid domains, where many steps are common for both the repair and de novo assembly of PSII. The sequence of the (re)assembly steps of genuine PSII subunits is relatively well-characterized in higher plants. A number of novel findings have shed light into the regulation mechanisms of lateral migration of PSII subcomplexes and the repair as well as the (re)assembly of the complex. Besides the utmost importance of the PSII repair cycle for the maintenance of PSII functionality, recent research has pointed out that the maintenance of PSI is closely dependent on regulation of the PSII repair cycle. This review focuses on the current knowledge of regulation of the repair cycle of PSII in higher plant chloroplasts. Particular emphasis is paid on sequential assembly steps of PSII and the function of the number of PSII auxiliary proteins involved both in the biogenesis and repair of PSII. This article is part of a Special Issue entitled: Chloroplast Biogenesis.

1. Introduction

Photosystem II (PSII) is a multi-subunit thylakoid membrane protein complex that catalyzes the light-driven electron transfer from water to plastoquinone (PQ). PSII is located mostly in the stacked areas of the thylakoid membrane, called grana membranes, and more than 40 proteins have been found associated with PSII, either stably or transiently [1,2]. Recently, the crystallographic structure of cyanobacterial (*Thermosynechococcus vulcanus*) oxygen evolving PSII complex was determined at 1.9-Å resolution [3]. The minimal reaction center complex of PSII, capable of charge separation, contains five proteins: D1, D2, α and β subunits of Cyt b_{559}, and PsbI subunit [4]. The reaction center proteins D1 and D2 bind all the redox-active cofactors, which are required for PSII electron transport. Excitation energy, captured by light harvesting complex (LHC)II and the PSII internal antenna proteins CP43 and CP47, induces charge separation in PSII, which in turn enables the oxygen-evolving complex (OEC) to oxidize water molecules and provide electrons to the electron transfer chain. Apart from the major PSII core proteins D1, D2, CP43 and CP47, the bulk of other PSII subunits are of low molecular mass and mainly involved in PSII assembly, stabilization and dimerization. Of these, chloroplast-encoded low molecular mass subunits of PSII, including PsbI, PsbJ, PsbL, PsbM and PsbTc, have been shown to be important for the assembly and/or stability of PSII in higher plants [5–9]. In turn, the nuclear-encoded PsbW is important for the accumulation of the PSII-LHCII supercomplexes [10]. In addition, a high number of auxiliary proteins assist the assembly of PSII.

2. Physiological significance of PSII damage and repair

Literature during the past 30 to 40 years has well established the susceptibility of the PSII D1 protein to damage upon exposure of plants to light in their natural environments, and similarly, the basic concept for replacement of the damaged D1 protein by a newly-synthetized copy during the repair cycle of PSII has been extensively investigated and reviewed [11–15]. Moreover, the damage of the D1 protein has been shown to be directly proportional to light intensity [16,17]. With the half-life of 2.4 h, the D1 protein was recently shown to have the fourth fastest turn-over rate of barley (*Hordeum vulgare*) proteins, when plants were growing under normal growth light intensity (500 μmol m^{-2} s^{-2}) [18]. Also, the D2, CP43 and PsbH subunits show higher degradation rate as compared to the other PSII subunits

☆ This article is part of a Special Issue entitled: Chloroplast Biogenesis.

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http://dx.doi.org/10.1016/j.bbabio.2015.01.006

[18–21]. Indeed, one of the major challenges of oxygenic photosynthetic organisms is to ensure the maintenance of PSII activity.

Originally, the vulnerability of PSII was thought to be an inherent fault of the photosynthetic machinery, but more recently it has been concluded that there is a strong physiological basis for the constant, yet highly regulated, photodamage and repair cycle of PSII. While efficient repair system is available for PSII, such a mechanism has been considered not to exist for PSI and hence the damaged PSI complexes are thought to be irreparable. The degradation of damaged PSI subunits and subsequent de novo synthesis and assembly of PSI proteins and Fe–S clusters are time consuming and energy requiring processes. Indeed, it has been observed that the amount of functional PSI in cucumber (*Cucumis sativus*) leaves is not fully recovered in six days after the photoinhibitory treatment at chilling temperatures, which causes serious damage to PSI complexes [22]. Consequently, it has been proposed that the role of PSII damage is the protection of PSI [23]. In short, the functional PSII centers are suggested to increase the redox pressure on PSI making PSI more susceptible to photoinhibition. Thus, partial inhibition of the PSII complexes is likely to protect PSI. In line with this hypothesis, a dynamic control of active PSII centers via photoinhibition-repair cycle was recently demonstrated to rescue *Arabidopsis* (*Arabidopsis thaliana*) PSI from photodamage under high light conditions, yet under normal growth temperature [24]. Taking together, the photodamage of PSII cannot any more be considered solely as an undesired consequence of the highly oxidizing chemistry of the water splitting PSII, but it is likely to function also as a PSI protection mechanism.

3. Lateral trafficking along the thylakoid membrane is essential for PSII repair cycle

In plants the thylakoid membrane network is composed of appressed grana stacks interconnected with non-appressed thylakoid domains, the stroma-exposed thylakoids. The diameter of grana is typically 300–600 nm and the extent of grana membrane stacking is dynamically regulated based on the prevailing light environment. In short, shade plants contain broader grana stacks with more membrane layers per granum as compared to sun plants [25]. Electron microcopy has revealed that in spinach (*Spinacia oleracea*) a 10 min switch to a lower light intensity increased grana size and number per chloroplast by 10–20% and returning of the leaves to the normal growth light for 10 min reversed the phenomenon [26]. The PSII complexes are most active as dimers and supercomplexes [27], which are densely packed in grana core regions of the thylakoid membrane network [28–30]. However, monomerization and migration of PSII complexes to non-appressed thylakoids are a prerequisite for the repair cycle [31]. Indeed, the FtsH and Deg proteases degrading the damaged D1 protein, ribosomes and the SecY translocon responsible for synthesis and thylakoid insertion of the newly-synthetized D1 proteins, respectively, and several auxiliary proteins assisting the PSII assembly (including PSB27, LPA1, CYP38/TLP40, LQY1 and TLP18.3) are all enriched in the non-appressed domains of the thylakoid membrane [32–35].

Several reports on molecular mechanisms related to the lateral trafficking of damaged PSII complexes from the grana domains to the stroma-exposed regions of the thylakoid membrane have been published recently. The phosphorylation of PSII core proteins is strongly linked to the destacking and lateral shrinkage of grana as well as to the mobility of PSII under high light intensity (for details, see Section 5.1). Not only the phosphorylation of PSII core proteins but also the amount of *Arabidopsis* Curvature Thylakoid 1(CURT1) proteins, which are highly enriched in the grana margins, has been shown to represent an important factor determining the number of membrane layers in grana stacks [36]. More research is, however, required to validate the role of PSII phosphorylation and CURT1 protein during high light induced changes in thylakoid architecture.

4. Sequence of PSII assembly in higher plants

During the past few years, a number of research and review articles have been published relating to the biosynthesis, assembly and repair of PSII. Not only the sequence of these pathways has emerged but also particularly the identification and characterization of PSII auxiliary proteins and regulatory mechanisms have greatly advanced our knowledge concerning the PSII assembly and repair cycle (Fig. 1) [37–41]. Distinct assembly intermediates of de novo synthesized PSII complexes and of those under repair have been characterized in detail using stepwise pulse-chase labeling experiments combined with the analysis of the composition of protein complexes by native gels. More detailed view of PSII synthesis/repair mechanisms has been gained through characterization of mutant plants deficient in a distinct structural subunit or an auxiliary protein of PSII as well as via experiments focusing on the etioplast-to-chloroplast transition. Although the assembly process of PSII in higher plants, algae and cyanobacteria resembles closely each other and several auxiliary factors assisting the assembly are evolutionarily conserved, there are also distinct differences and therefore we focus below on PSII repair occurring in plant chloroplasts.

Many aspects of PSII repair cycle and de novo biogenesis are partially overlapping. However, in the PSII repair cycle only the damaged reaction center protein D1 and occasionally also the D2, CP43 and PsbH subunits are replaced [14,18–21], while the other protein components of the complex are recycled. The repair cycle of PSII starts by monomerization of the phosphorylated dimeric PSII complex in grana stacks [31] followed by dephosphorylation of the core proteins D1, D2 and CP43, most likely on the way from grana to non-appressed stroma-exposed thylakoids. Partial disassembly of the PSII core complex (release of CP43 inner antenna protein, the OEC proteins and yet unidentified low molecular mass subunits) is followed by proteolysis of the damaged D1 protein. In turn, the biogenesis of PSII starts with the translation of α and β subunits of the Cyt b_{559}, followed by the assembly of core protein D2 [5,6,42–44]. The smallest PSII subcomplex likely to be common for both the biogenesis of PSII and the repair of PSII is composed of Cyt b_{559}, D2 and low molecular mass subunit PsbI, but lacks the D1 protein [21,42,45]. During the repair of PSII, this subcomplex is likely to contain also other low molecular mass subunits, but this remains to be elucidated.

Light modulates the initiation of *psbA* mRNA translation [46] and the chloroplast signal recognition particle cpSRP54 targets the ribosome nascent D1 chain to the thylakoid membrane [47,48], where co-translational insertion of the D1 protein into the PSII core subcomplex takes place through the cpSecY translocation channel [33,45]. Ribosome pausing on *psbA* mRNA during elongation of the newly-translated D1 protein makes it possible for nascent chains to bind cofactors, such as chlorophylls, prior to polypeptide release from the ribosomes [46,49]. D1 has five transmembrane helixes, with the N-terminus on the stromal side of the thylakoid membrane. The first two transmembrane helixes of nascent D1 chain interact loosely with the D2 protein [50,51], while strong D1–D2 interaction is reached after translation of four transmembrane helixes of the D1 protein has been completed [50]. The inner antenna protein CP47 is assembled next, followed by the assembly of low molecular mass proteins PsbH, PsbL, PsbM, $PsbT_c$, PsbR and probably also of PsbJ [21,31] upon the de novo assembly of PSII. Instead, during the repair cycle it is conceivable that a part of these proteins are not released but remain attached to the PSII core throughout the process. Low molecular mass subunits of PSII are essential for stabilization of the CP43-less PSII core complex, which is the smallest PSII repair cycle intermediate visible in silver stained gels. The C-terminal processing of the D1 protein [52–54] is likely to precede the assembly of CP43, PsbK and PsbO proteins. The assembly of PsbW and PsbZ, controlling the

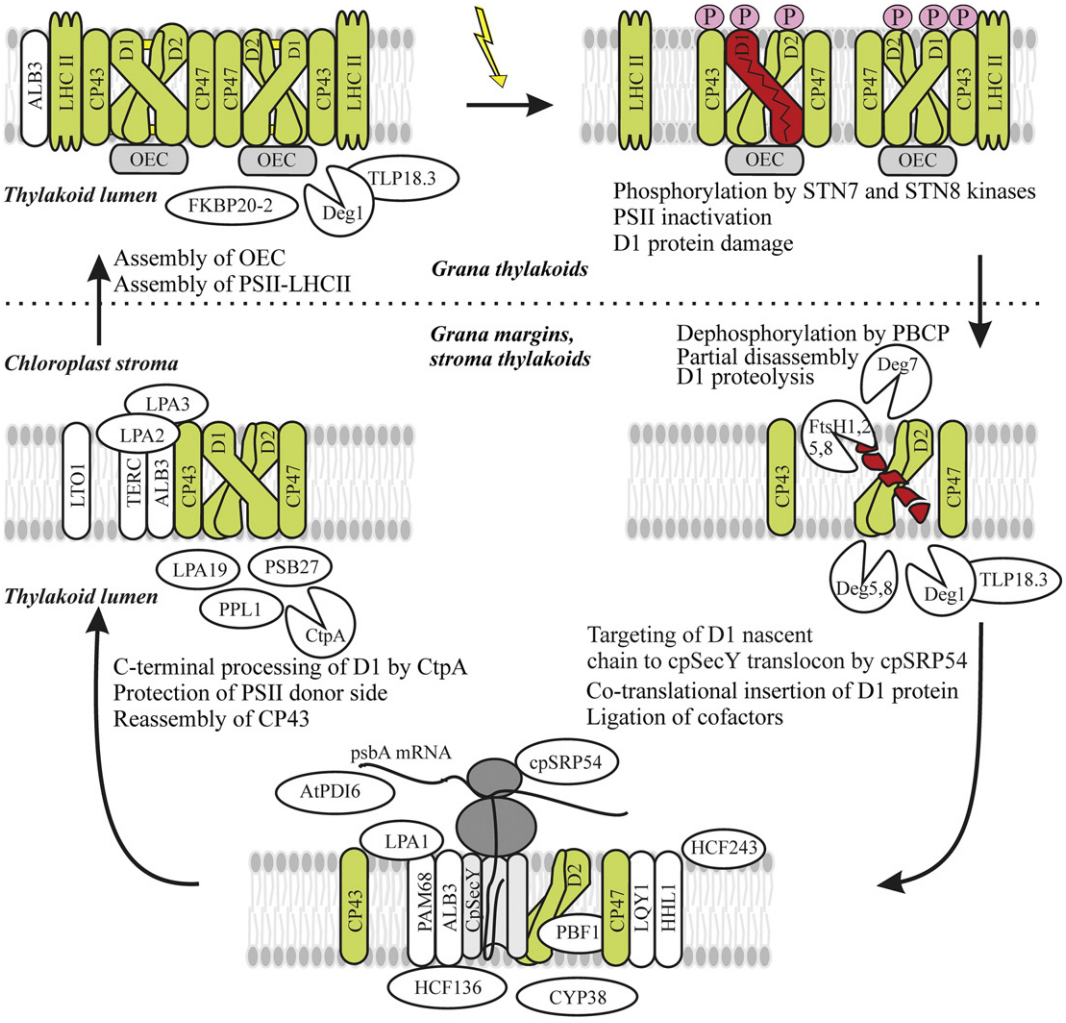

Fig. 1. Simplified model depicting the major steps of PSII photoinhibition-repair cycle and localizing the major assisting proteins of the process in higher plant chloroplasts. PSII core proteins and LHCII proteins (green), OEC proteins (gray) and assisting protein factors (white) are indicated. Light-induced damage of the D1 protein is highlighted by red color. OEC, oxygen-evolving complex, LHC, light harvesting complex, P, phosphorylation.

interaction of PSII core with LHCII [10,55], are the subsequent assembly steps, followed by PSII dimerization and formation of the PSII-LHCII supercomplexes in the appressed grana thylakoids [21].

5. Regulation of PSII repair cycle

Repair cycle of PSII, which constitutes a number of steps common with PSII assembly as discussed above, is additionally regulated by

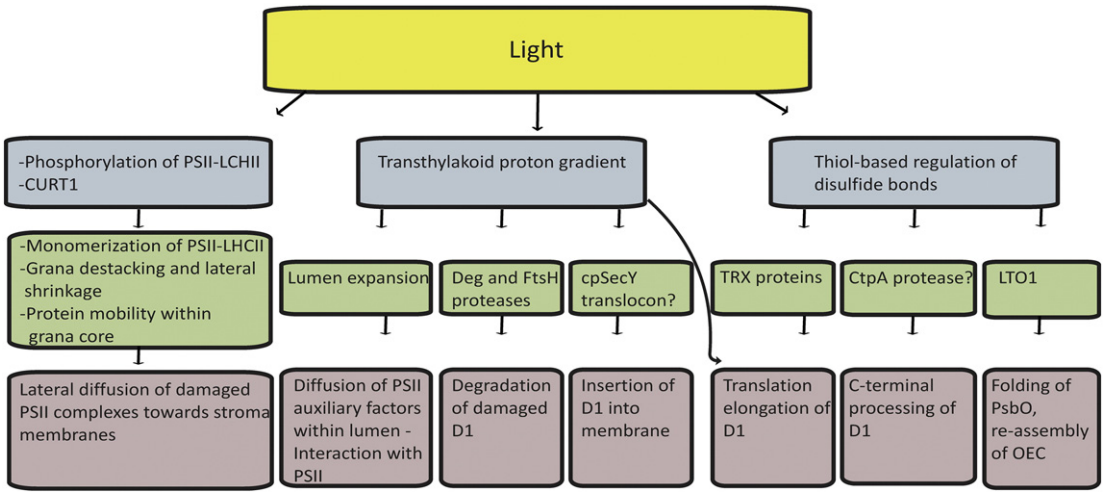

Fig. 2. Light-induced post-transcriptional regulation of the PSII repair cycle in higher plant chloroplasts.

reversible phosphorylation of PSII core proteins, transthylakoid proton gradient as well as by thioredoxin (Trx)-dependent redox regulation. Particular role is played by a number of PSII auxiliary proteins that assist and regulate the PSII repair cycle but are also often involved in the biogenesis of PSII. All these regulatory processes are shortly discussed below.

5.1. Phosphorylation of PSII core proteins facilitates the initiation of repair

Threonine residues at the very N-terminus of a group of PSII core proteins (D1, D2, CP43 and PsbH) undergo reversible phosphorylation [56,57]. Phosphorylation of the core proteins is catalyzed by STN8 and to a lesser extent at low light also by the STN7 kinase [58,59], while the Photosystem II Core Phosphatase (PBCP) is required for dephosphorylation of the PSII core proteins [60]. Phosphorylation of the PSII core proteins plays a regulatory role in the PSII repair cycle by facilitating the migration of the damaged PSII from the grana to the stroma-exposed membrane (Fig. 2). Indeed, phosphorylation of the PSII core subunits occurs before any damage of PSII but phosphorylated PSII core has been shown to facilitate the unpacking of damaged PSII complexes in the grana for subsequent repair in the stroma-exposed thylakoid membrane [59]. This is likely due to high light induced conformational changes in photosynthetic pigment–protein complexes as well as to changes in the macroscopic folding of the thylakoid membrane, such as lateral shrinkage and destacking of the grana [61,62]. Phosphorylation of PSII core proteins also increases the membrane fluidity and hence the protein complex mobility within the thylakoid membrane upon high light exposure [62,63]. Collectively, these results indicate that the phosphorylation of PSII core proteins facilitates the accessibility between the damaged PSII complexes and the repair machinery. While the migration step is assisted by PSII core protein phosphorylation, dephosphorylation is a prerequisite for efficient degradation of the D1 protein in non-appressed stroma-exposed membranes [64,65] and in line with this, the PSII core protein phosphorylation was recently shown to prevent the undesirable cleavage of damaged D1 protein [66]. It should be noted, however, that the lack of PSII core protein phosphorylation does not completely abolish the repair of PSII [59]. Instead, the PSII core phosphorylation rather facilitates and accelerates the repair cycle of PSII, which is important especially under high light conditions.

5.2. Transthylakoid proton gradient is essential for efficient D1 proteolysis and resynthesis

During vectorial photosynthetic electron transport, protons are pumped from stroma to the thylakoid lumen, thus creating a proton motive force, which is used by the ATP synthase in production of ATP. Lumen acidification has well-characterized consequences, for example the activation of non-photochemical quenching and the photosynthetic control of electron flow via the cytochrome b_6f complex as well as controlling the translocation of proteins across the thylakoid membrane. Acidification of the thylakoid lumen is also strongly linked to the repair cycle of PSII (Fig. 2). Firstly, the light-induced swelling of the lumen has been suggested to assist the lumenal auxiliary proteins to reach the PSII complexes [67]. Secondly, formation of the active Deg1 homo-hexamers, degrading the damaged D1 protein, has been shown to be a pH-sensitive process [68]. In line with this, acidification of the thylakoid lumen also stimulates oligomerization of the other D1 degrading protease FtsH [69]. Thirdly, translation elongation of the D1 protein is dependent on the formation of transthylakoid proton gradient, which might be linked to the insertion of the D1 nascent chains into the membrane via the cpSecY translocon [33,50,70].

5.3. Trx-dependent redox regulation of de novo D1 protein synthesis

Regulation of protein translocation, folding and/or activity via Trx-mediated redox control is a wide-spread phenomenon in living organisms. It has been known for a long time that the translation and C-terminal processing of the D1 protein are regulated by reducing compounds produced by PSI (Fig. 2) [50,71]. However, despite these observations the molecular mechanism behind Trx-based regulation of PSII repair cycle remained unknown. More recently, Trx affinity chromatography and reduction of the target proteins by Trx, followed by labeling of the thiol groups with a fluorescent probe or differential mass tags have revealed a high number of PSII subunits and auxiliary proteins as potential targets of Trxs, including the D1 processing protease CTPA and auxiliary proteins CYP38 and FKBP20-2 [72,73]. Moreover, the PSII subunits D1, D2, CP43, CP47 and PsbO are likely to be capable of forming redox-sensitive intra and/or intermolecular disulfide bridges connecting the redox status of the chloroplast with the assembly of PSII [74]. The discovery of protein disulfide isomerase LQY1, which interacts with CP43 and CP47 and affects the functionality of PSII [34], has shed light into the possible molecular level mechanisms behind the redox regulation of PSII assembly. Members of the Trx superfamily can also function as negative regulators of PSII biogenesis, since protein disulfide isomerases AtPDI6 have been shown to attenuate D1 synthesis [75]. It is highly likely that at least Trx m is involved in the assembly of PSII since Trx m1, Trx m2 and Trx m4 can associate with PSII assembly intermediate complexes and interact directly with PSII core subunits D1, D2 and CP47 [74]. Moreover, the triple inactivation line deficient of all three *TRX m* genes demonstrated defects both in the accumulation and activity of PSII [74]. Formation of disulfide bridges is important for the folding and stability of the PsbO protein as well, as evidenced by mutant plants lacking disulfide bond-forming catalyst LTO1 (Fig. 2) [73,76].

5.4. Auxiliary proteins assist PSII repair and assembly at multiple steps

Circa 3000 of *Arabidopsis* 28 000 genes contain a chloroplast-targeting signal but the function for only a small fraction of those proteins was known ten years ago [77,78] and even now the situation has not drastically improved. Considering the complicated nature of PSII biogenesis and repair cycle and the number of functionally unknown chloroplast proteins, it is plausible that several assembly factors, chaperones, proteases, translocation proteins, kinases, and phosphatases involved in regulation of PSII biogenesis and repair are still waiting to be discovered. As mentioned, the sequences of biogenesis and repair of PSII resemble closely each other. It is highly conceivable that many of the auxiliary proteins assigned to assist the repair cycle take part also in the biosynthesis pathway of PSII, and vice versa. Thus, the classification of auxiliary proteins separately to those involved in biogenesis and those involved in repair of PSII might not be relevant. Moreover, a comprehensive understanding of the interplay between different auxiliary proteins assisting the repair (and biogenesis) of PSII has remained elusive. In this section, the functional role of the most recently characterized auxiliary proteins of PSII is addressed in detail. Instead, detailed description of auxiliary PSII proteins of which new data is not available since [37] is omitted from this section. These include ALB3 [79], FKBP20-2 [80], HCF136 [81,82], LPA1 [83], LPA2 [84] and PPL1 [85]. Table 1 depicts all auxiliary PSII proteins that are currently known to have interaction with PSII proteins during the repair cycle, emphasizing their location in chloroplasts and functional role upon specific proteolytic and assembly steps of PSII.

5.4.1. Auxiliary proteins of PSII repair localized to chloroplast stroma

Only few proteins, which are mainly located in the chloroplast stroma and only transiently attached to the thylakoid membrane, have been shown to assist the PSII repair as auxiliary proteins. Functional roles of the stromal auxiliary proteins vary from degradation of the damaged D1 protein to assembly of the PSII core complex.

Table 1
Summary of the characterized PSII auxiliary proteins. Thylakoid membrane = TM, lumenal = L, stromal = S.

Auxiliary protein	Gene code	Location	Proposed function(s) during PSII biogenesis/repair cycle	References
FtsH1	*AT1G50250,*	TM	Degradation of D1 protein	[66,101–104,106,107]
FthH2	*AT2G30950,*			
FtsH5	*AT5G42270,*			
FtsH8	*AT1G06430*			
Deg1	*AT3G27925*	L	Degradation of D1 protein; assembly of PSII	[68,125,127,129]
Deg5	*AT4G18370,*	L	Degradation of D1 protein	[126]
Deg8	*AT5G39830*			
Deg7	*AT3G03380*	S/TM	Degradation of D1 protein	[86,87]
TLP18.3	*AT1G54780*	L	Degradation of D1 protein and dimerization of PSII	[130,131]
AtPDI6	*AT1G77510*	S	Regulator of D1 synthesis	[75]
ALB3	*AT2G28800*	TM	Insertion and assembly of PSII and LHCII proteins	[79,98]
HCF136	*AT5G23120*	L	Assembly of PSII reaction center	[81,82]
CYP38, TLP40	*AT1G01480*	L	Assembly and stabilization of PSII	[140–143]
PsbN/PBF1	*ATCG00700*	TM	Assembly of PSII reaction center	[112,113]
HCF243	*AT3G15095*	TM	Biogenesis and assembly of D1 protein	[110]
LPA1	*AT1G02910*	TM	Biogenesis and assembly of D1 protein	[83]
PAM68	*AT4G19100*	TM	Assembly of PSII	[115]
LQY1	*AT1G75690*	TM	Assembly of PSII core and OEC	[34]
HHL1	*AT1G67700*	TM	Tolerance against light stress	[123]
LPA2	*AT5G51545*	TM	Biogenesis and assembly of CP43 protein	[84]
LPA3	*AT1G73060*	S/TM	Assembly of CP43 protein	[94]
TERC	*AT5G12130*	TM	Assembly of CP43 protein	[116,117]
PSB27,	*AT1G03600,*	L	Regulator of pD1 processing	[136,137]
LPA19	*AT1G05385*			
CTPA	*AT4G17740*	L	Processing of pD1 protein	[134]
LTO1	*AT4G35760*	TM	Assembly of PsbO protein	[76,118,120]
PPL1	*AT3G55330*	L	Assembly of PSII	[85]
FKBP20-2	*AT3G60370*	L	Assembly of PSII-LHCII	[80]
PSB33	*AT1G71500*	TM	Maintenance of PSII-LHCII	[114]
ROC4/CYP20-3	*AT3G62030*	S	Tolerance against light stress	[97]
PSB29/THF1	*AT2G20890*	S/TM	Tolerance against light stress	[88–91]

ATP-independent serine endopeptidase **Deg7** is peripherally associated to the stromal side of the thylakoid membrane and involved in primary cleavage of the photodamaged D1 protein [86]. Indeed, Deg7 has been shown to interact with the PSII complex, but not with the other thylakoid membrane protein complexes. Deg7 protease is crucial for efficient PSII repair in *Arabidopsis*, as evidenced by sensitivity of the *deg7* mutants to high light. Instead, no apparent difference between wild type and *deg7* plants was observed under normal growth conditions. Deg7 contains two protease domains (one active and one degenerated) and forms homotrimeric complexes, trimerization being based on the interactions between degenerated protease domains [87].

PSB29/Thylakoid Formation 1 (TFH1)/Non-Yellow Coloring1 (NYC4)/Altered Coronative Response (ALC1) protein has been localized both to the soluble stroma and to the thylakoid membrane [88]. *Arabidopsis psb29/thf1* plants show stunted growth and variegated leaves, which have been linked both to the thylakoid biogenesis and generation of reactive oxygen species (ROS) due to uncoupled proximal antenna of PSII [88,89]. More recently, PSB29/THF1/NYC4Y/ALC1 has been shown to regulate the PSII-LHCII supramolecular dynamics under high intensity light and during leaf senescence [90,91]. Indeed, *psb29/thf1/nyc4/alc1* mutant plants were shown to retain green during senescence, which derived from the highly stable components of both PSII and LHCII. The phenotype of *psb29/thf1/nyc4/alc1* might be attributed to unstable FtsH complex in the absence of PSB29/THF1/NYC4Y/ALC1 [92]. Moreover, the PSB29/THF1/NYC4Y/ALC1 protein has been identified based on its reduced gene expression in response to coronative, a non-host-specific phytotoxin [93]. When the effect of coronative on *Arabidopsis psb29/thf1* mutants was investigated, mutant plants displayed severe growth defects and accumulated more anthocyanin as compared to the wild type plants.

It was recently shown that a **Protein Disulfide Isomerase** (**AtPDI6**) catalyzing the reduction and oxidation of disulfide bonds of the client proteins, is localized to chloroplast stroma and regulates D1 protein synthesis [75]. Even though the *psbA* transcript levels in the AtPDI6 knockdown plants were similar to those of wild type both under growth light and high light, the *pdi6* plants showed increased rates of D1 synthesis, and consequently, less PSII photoinhibition when subjected to high light stress. In addition, the amount of AtPDI6 transcripts increased upon exposure of wild type plants to high light stress. Therefore, it was concluded that AtPDI6 is an attenuator of D1 synthesis. The results provide interesting insights into regulation of PSII photoinhibition-repair cycle, and suggest that deliberate photoinhibition of PSII might be one photoprotective mechanism that could be used e.g. to protect PSI from photoinhibition.

Low PSII Accumulation3 (LPA3) is a chloroplast protein present both in the soluble stroma and the thylakoid membrane [94]. The *lpa3* plants show reduced growth rate, pale green coloration and low PSII activity, which are due to defects in the PSII assembly process. The synthesis rates of D1, D2 and CP47 in the *lpa3* mutants are not affected, but the mutant demonstrates dramatically changed synthesis and degradation rates of CP43, being lower and higher, respectively, in the *lpa3* mutants as compared to wild type plants. LPA3 transiently interacts with PSII subunit CP43 and the LPA2 protein, which was previously shown to assist the assembly of CP43 into the PSII complex. Double mutation of LPA2 and LPA3 results in a seedling-lethal phenotype, indicating that LPA3 and LPA2 have overlapping functions as assembly factors of CP43. In addition, both LPA2 and LPA3 interact with ALB3 protein indicating co-operation between these proteins.

ROC4/CYP20-3 is the only cyclophilin located in the chloroplast stroma [95,96]. ROC4 is not required for the biogenesis of PSII but the *roc4* plants show increased sensitivity to high light [97]. Instead, ROC4 functions in the repair of photodamaged PSII as evidenced by the PSII activity measurements in the presence and absence of the chloroplast translation inhibitor lincomycin.

5.4.2. Thylakoid membrane-bound auxiliary proteins of PSII repair

Majority of the auxiliary proteins assisting the PSII repair cycle are either integral membrane proteins or peripherally attached to the thylakoid membrane. Membrane-bound PSII auxiliary proteins show a variety of functions ranging from the degradation of the damaged D1

protein to assembly of the PSII core complex and OEC. Discussion on three PSII auxiliary proteins, ALB3 [79,98,99], LPA1 [83] and LPA2 [84], which play important roles in three different processes i.e. insertion of PSII core and LHCII proteins into thylakoid membrane, assembly of D1 and assembly of CP43, respectively, is omitted from this section since no recent data after [37] is available from these proteins.

FtsH proteins compose a family of ATP-dependent membrane-bound metalloprotease, which are known to have a crucial role in housekeeping proteolysis of membrane-embedded proteins [100]. In *Arabidopsis*, FtsH1, FtsH2, FtsH5 and FtsH8 have been shown to reside in the thylakoid membrane [100] and have a well-characterized role in degradation of the damaged PSII core protein D1 [101]. Of thylakoid FtsH proteins, the highly homologous FtsH1 and FtsH5 are referred to as type A and FtsH2 and FtsH8 as type B subunits. In its active form, the chloroplastic FtsHs form a ring-shaped hetero-hexameric complex, which is most likely comprised two type A subunits and four type B subunits [102–104]. FtsH-mediated degradation of the D1 protein takes place in co-operation with the lumenal Deg proteases [105]. Mutants deficient of FtsH5 and FtsH2 show variegated phenotype [106], and attenuated D1 degradation under photoinhibitory conditions [107]. Leaf variegation phenotype of the *var* mutants and high amounts of FtsH proteins in the etioplast proteome collectively indicate that the FtsH proteases not only function in the repair of photodamaged PSII but have specific functions also during thylakoid biogenesis [44,108,109].

High Chlorophyll Fluorescence 243 (HCF243) is an intrinsic thylakoid membrane protein associated to PSII assembly [110]. The mutant plants lacking HCF243 are clearly smaller than the wild type plants and show dramatically reduced PSII activity. In the absence of HCF243, the amount of chloroplast-encoded PSII subunits is greatly reduced, while the amount of the nuclear-encoded PSII proteins is not affected. The impaired accumulation of chloroplast-encoded PSII subunits in *hcf243* is likely to be caused by severely reduced synthesis rate of D1, and to a lesser extent of D2. Furthermore, enhanced D1 degradation and hampered PSII assembly were demonstrated for *hcf243* as compared to wild type. In line with these results, HCF243 has been shown to interact with the D1 protein in vivo. It is therefore evident that HCF243 is crucial for proper accumulation of the D1 protein.

Recently, two independent studies showed that a small chloroplast-encoded thylakoid membrane protein ORF43, originally assigned as **PsbN** [111], is not a PSII subunit, but instead has a role as an assembly factor of PSII [112,113]. Therefore, to prevent further misinterpretations concerning the protein, another group proposed a new name, **Photosystem Biogenesis Factor 1 (PBF1)**, for the *orf43* gene product [112]. The tobacco Δ*psbN*/Δ*pbf1* mutants are prone to photoinhibition and show drastic defects in PSII amount and function [112,113]. In vivo pulse labeling experiments revealed a malfunction in the PSII assembly in the absence of PBF1. Mutants are capable of forming PSII assembly intermediates of low molecular mass, i.e. pre-D2 and pre-D1 complexes, but already as early assembly step as the formation of the PSII reaction center complex is hampered [113]. It was thus concluded that PsbN/PBF1 is an essential assembly factor, which mediates the formation of PSII reaction center. Based on lowered amount of PSI subunits as well, it was suggested that PBF1 bears a role also in PSI biogenesis [112].

A previously uncharacterized thylakoid membrane protein AT1G71500 was recently shown to be associated with PSII [114]. The protein, named as **PSB33**, has a light harvesting complex binding motif in its transmembrane domain, and based on co-immunoprecipitations and cross-linking experiments, it was demonstrated to reside in close vicinity to CP43 and LHCII. The *psb33* mutant plants show lower oxygen-evolving activity and reduced amount of the PSII-LHCII supercomplexes as compared to wild type. Furthermore, the *psb33* mutant plants accumulate ROS under high light treatment and show higher rate of PSII damage and attenuated capacity for PSII repair. Thus, it is concluded that PSB33 ensures proper maintenance of the PSII-LHCII supercomplexes.

The Photosynthesis Affected Mutant 68 (PAM68) is yet another integral thylakoid membrane protein that functions as a PSII auxiliary protein. The mutant plants lacking PAM68 show severely reduced growth rate and pale green leaves compared to the wild type plants [115]. PAM68 co-migrates with a low molecular mass protein complex composed of LPA1, D1, and D2 proteins, which might correspond to PSII reaction center. PAM68 interacts with several PSII core subunits like D1, D2, CP43, CP47, PsbH and PsbI, but, interestingly, not with PsbE or PsbF, which are part of the inner core of PSII and are assembled among the first PSII subunits [21]. PAM68 also interacts with other PSII auxiliary proteins HCF136, LPA1, LPA2 and ALB3. The *pam68* mutant shows severely reduced amounts of chloroplast-encoded PSII core subunits D1, D2, CP43, CP47 and PsbE. In line with this, the PSII activity is dramatically reduced in the *pam68* plants. Furthermore, *pam68* suffers from decreased stability and maturation of the precursor form of D1 (pD1). The amount of the PSII reaction center complex in *pam68* is significantly elevated as compared to WT, while the amounts of PSII dimers and supercomplexes are lower. Deduced from these data, it is conceivable that PAM68 is essential for the conversion of the PSII reaction center complexes into larger PSII assembly intermediates.

Tellurite Resistance C (TERC), is an integral thylakoid membrane protein with eight predicted transmembrane helixes [116]. TERC has been shown to interact with a PSII assembly factor ALB3 [117]. The knockout *terc* plants are seedling lethal, and plants grown on sucrose show malformed thylakoid membrane network and significantly down-regulated amounts of thylakoid membrane proteins [116]. Analyses of the *terc* knockdown lines revealed defects in the amount and function of PSII, in particular the synthesis of the PSII core subunits D1, D2, CP43 and CP47 was reduced [117]. It seems conceivable that TERC functions together with ALB3 at the co-translational insertion step of thylakoid membrane proteins, most likely assisting the assembly of CP43 to PSII reaction center.

The Low Quantum Yield of Photosystem II (LQY1) is a small zinc finger thylakoid protein, which is enriched in unstacked regions of the thylakoid membrane [34]. LQY1 shows protein disulfide isomerase activity, which is predicted to locate at the lumen side of thylakoid membrane. LQY1 co-migrates with PSII monomer and CP43-less core monomer, indicating a role during biogenesis and repair of PSII. The *lqy1* mutant plants show lower quantum yield of PSII photochemistry, reduced PSII electron transport rate and accumulate less PSII supercomplexes after the high light treatment as compared to wild type. Interestingly, the absence of LQY1 has been shown to accelerate the synthesis and degradation rates of the D1 protein. Thus it seems likely that LQY1 assists the breakage and formation of disulfide bonds of the PSII core subunits and/or OEC proteins during the assembly of PSII.

Thylakoid membrane proteins have been shown to assist also the stable assembly of the lumenal OEC complex. **Lumen Thiol Oxidoreductase1 (LTO1)** is a membrane-embedded disulfide bond-forming catalyst, which was first shown to be required for the assembly of PSII. The *lto1* mutant plants show a stunted growth phenotype and impaired accumulation and function of PSII [76]. Trx-like domain in LTO1 is facing towards the lumen [118] and was shown to be required for formation of a disulfide bond in the PsbO subunit of OEC [76], thus protecting the protein from degradation [73,119]. LTO1 might have a broad range of substrates in lumen, since it is also capable of promoting the formation of disulfide bond in FKBP13 [120]. LTO1 is also involved, either directly or indirectly, in degradation of the D1 protein, in xanthophyll cycle, and in abscisic acid-mediated response to osmotic stress in *Arabidopsis* [121,122].

Stroma-exposed thylakoid membranes accumulate the **Hypersensitive to High Light1 (HHL1)** protein [123]. *Arabidopsis hhl1* mutants show markedly reduced PSII activity, high non-photochemical quenching and increased accumulation of ROS following the high light exposure. HHL1 interacts with the LQY1 protein and the *hhl1 lqy1* double mutant plants show increased photosensitivity as compared to the

single mutants. Therefore, HHL1 is suggested to protect the PSII complexes against photodamage.

5.4.3. Lumenal auxiliary proteins regulating PSII repair

Lumenal PSII auxiliary proteins bear functions linked to degradation of the damaged D1 protein, processing of the pD1 protein and assembly of the PSII core and PSII-LHCII supercomplexes. Lumen localized PSII auxiliary proteins HCF136 [81,82] and FKBP20-2 [80], which regulate the accumulation of PSII reaction center and PSII-LHCII, respectively, are not further discussed below in the absence of new information since [37]. Description of PPL1 [85] is likewise omitted.

All three lumenal serine proteases, **Deg1, Deg5 and Deg8**, [78,124] are now known to operate in cleavage of the lumen-exposed loops of the D1 protein at specific sites during PSII repair cycle [125,126]. Deg5 and Deg8 interact to form an active protease complex under high light [126]. The activation of Deg1 takes place via transformation of the Deg1 monomers into a proteolytically active hexamer at acidic pH upon protonation of a histidine amino acid residue [68]. Moreover, a novel role of Deg1 as a chaperone/assembly factor of PSII was recently suggested [127]. Similarly to its homolog in *Escherichia coli* [128], Deg1 shows chaperone activity located at its protease domain [127]. Deg1 interacts with the PSII core protein D2 in PSII-LHCII supercomplexes, PSII dimers and monomers, but not in CP43-less monomers [127]. Lower level of newly-synthetized PSII dimers and PSII-LHCII supercomplexes in *deg1* has been taken as an indication of an importance of the Deg1 protein for the assembly of PSII dimers and supercomplexes [127]. In addition, Deg1 has been shown to possess proteolytic activity against the PsbO protein in vitro [129].

Thylakoid Lumen Protein of 18.3 kDa, TLP18.3, was identified as an auxiliary protein assisting the turnover of damaged D1 protein and the dimerization of PSII complexes [130]. Later, TLP18.3 has been shown to be an acidic phosphatase, thus being the first phosphatase found in the thylakoid lumen [131]. TLP18.3 is not a tightly bound subunit of any thylakoid membrane protein complex according to analysis by 2-D BN/SDS-PAGE, although weak interactions are possible [130]. In line with defects in the degradation and dimerization steps of D1 protein, TLP18.3 has been shown to interact with lumenal Deg1 protease [132].

The Carboxyl Terminal Peptidase (CTPA) cleaves the C-terminal extension from the pD1 in thylakoid lumen, thus completing the maturation of the D1 protein upon PSII assembly [53]. Experiments with cyanobacteria mutants first demonstrated that the disruption of CTPA causes loss of PSII activity, and in particular, prevents proper processing of the pD1 protein that is an essential step for the assembly of OEC to PSII [133]. *Arabidopsis* genome has three homologous genes to cyanobacterial CTPA. Of these, the gene product of *AT4G17740* was recently shown to be responsible for C-terminal processing of pD1 [134]. A knockout line of the CTPA was devoid of mature D1 and suffered from non-functional PSII, whereas a knockdown line, capable of processing part of the pD1, showed only moderately hampered PSII activity. Furthermore, the knockout line was unable to accumulate PSII-LHCII supercomplexes, while the knockdown line exhibited a supercomplex composition comparable to the wild type [134]. Nevertheless, the knockout line contained highly reduced amounts of PSII core and OEC proteins, whereas the knockdown line did not show defects in the amounts of these proteins. Thus, the diminished amount of the PSII-LHCII supercomplexes in the knockout line might as well be due to decreased amount of the OEC proteins, which have been shown to be needed for proper assembly of the PSII-LHCII supercomplexes [135]. Both AT4G17740 and the putative C-terminal processing protease AT5G46390 have been suggested to be Trx target proteins [73], however, there is so far no experimental evidence indicating that Trx would regulate CTPA.

PSB27 and **Low PSII Accumulation 19** (**LPA19**) are homologous thylakoid lumen proteins, which are likely to facilitate the correct timing of the C-terminal processing of D1 in higher plants during the repair cycle and biogenesis of PSII, respectively [136,137]. These proteins thus make an interesting pair that has analogous functions but, indeed, two different proteins are needed to complete the function upon de novo assembly of PSII and upon PSII repair. In *psb27* mutant plants the growth rate as well as the amount and activity of PSII remain largely unaffected as compared to wild type [136]. Instead, formation of the PSII-LHCII supercomplexes is abolished in the *psb27* mutant [138]. Under high light intensity, the PSII activity and the amount D1 decrease more significantly in the *psb27* mutant than in wild type plants. Moreover, recovery of PSII activity after photoinhibitory treatment of plants is delayed in the *psb27* mutant, suggesting that PSB27 is required for efficient recovery of the photodamaged PSII complex. On the other hand, the *lpa19* plants show pale-green phenotype, reduced growth rate and lower photochemical efficiency of PSII as compared to wild type plants [137]. In line with this, the *lpa19* plants accumulate low levels of PSII core subunits. LPA19 has been observed to transiently interact with both the pD1 and mature form of the D1 protein, and in the absence of LPA19, processing of pD1 is impaired.

Thylakoid lumen proteome of *Arabidopsis* contains five cyclophilins, of which **CYP38** has been linked to both PSII biogenesis and repair cycle. Crystal structure of *Arabidopsis* CYP38 revealed two distinct functional domains: an N-terminal helical bundle and a C-terminal cyclophilin β-barrel, which are connected by an acidic loop [139]. The N-terminal helical domain is closely packed together with the cyclophilin domain, which might prevent the access of the cyclophilin domain to other proteins and thus functions as autoinhibitory mechanism of CYP38. *Arabidopsis cyp38* plants are hypersensitive to light and show dramatically stunted growth compared to the wild type [140,141]. Moreover, *cyp38* plants accumulate reduced amounts of both the PSI and PSII proteins. When young seedlings, instead of mature rosette leaves, were investigated, it turned out that the biogenesis of PSII was defective in *cyp38* and down-regulation of PSI is only a secondary effect [141]. In mature plants, reduced amount of the PSII complexes is due to a shorter half-life of PSII, which highly likely originates from defects in the assembly of PSII. The dual role of CYP38 both during the biogenesis and repair cycle of PSII is in line with observation that the cyclophilin domain of CYP38 interact with the E-loop of the inner antenna protein of PSII, CP47 [139], which is assembled after the D1 protein. The spinach homolog of CYP38, thylakoid lumen protein of 40 kDa, **TLP40**, is known to interact with a PP2A-like phosphatase at the thylakoid membrane, where it is involved in maintenance and repair of PSII complexes through regulating dephosphorylation and subsequent replacement of damaged PSII core proteins [142,143]. Also in *Arabidopsis*, CYP38 has been shown to modulate the phosphorylation level of PSII core and LHCII proteins [141].

6. Concluding remarks

The repair cycle of PSII as such has an indispensable role in sustenance of photosynthesis and life of plants. When considering the natural growth environments of plants, short- and long-term fluctuations in light availability challenge the functionality of various regulatory mechanisms, including those involved in the PSII repair cycle. To that end, instead of using the static laboratory growth conditions, the photochemical properties and regulation of PSII should be investigated under naturally fluctuating light conditions, or even better, in field conditions challenging the whole physiology of plants. Indeed, for example the *tlp18.3* plants, which do not show any phenotype when grown under constant low, medium or high intensity light, revealed stunted phenotype as compared to the wild type when grown under fluctuating light conditions [130]. The present knowledge concerning the interplay between auxiliary proteins assisting the repair cycle and biogenesis processes of PSII is emerging but more efforts are needed to reach a detailed overview. It can be predicted that interactomes of several auxiliary proteins, the known ones and those

still to be identified, are involved in guiding the assembly and repair of PSII. Thus, in-depth analyses of the interactions of PSII auxiliary proteins with each other and with different subdomains of PSII upon the assembly and repair processes are likely to be in focus during forthcoming years.

Transparency Document

The Transparency document associated with this article can be found, in the online version.

Acknowledgments

Dr. P. Mulo is thanked for the critical reading of the manuscript. This work was supported by the Academy of Finland projects 271832 and 273870.

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