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Review

Strategies for *psbA* gene expression in cyanobacteria, green algae and higher plants: From transcription to PSII repair☆

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ABSTRACT

The Photosystem (PS) II of cyanobacteria, green algae and higher plants is prone to light-induced inactivation, the D1 protein being the primary target of such damage. As a consequence, the D1 protein, encoded by the *psbA* gene, is degraded and re-synthesized in a multistep process called PSII repair cycle. In cyanobacteria, a small gene family codes for the various, functionally distinct D1 isoforms. In these organisms, the regulation of the *psbA* gene expression occurs mainly at the level of transcription, but the expression is fine-tuned by regulation of translation elongation. In plants and green algae, the D1 protein is encoded by a single *psbA* gene located in the chloroplast genome. In chloroplasts of *Chlamydomonas reinhardtii* the *psbA* gene expression is strongly regulated by mRNA processing, and particularly at the level of translation initiation. In chloroplasts of higher plants, translation elongation is the prevalent mechanism for regulation of the *psbA* gene expression. The pre-existing pool of *psbA* transcripts forms translation initiation complexes in plant chloroplasts even in darkness, while the D1 synthesis can be completed only in the light. Replacement of damaged D1 protein requires also the assistance by a number of auxiliary proteins, which are encoded by the nuclear genome in green algae and higher plants. Nevertheless, many of these chaperones are conserved between prokaryotes and eukaryotes. Here, we describe the specific features and fundamental differences of the *psbA* gene expression and the regeneration of the PSII reaction center protein D1 in cyanobacteria, green algae and higher plants. This article is part of a Special Issue entitled Photosystem II.

1. Introduction

Higher plants, algae and cyanobacteria all perform oxygenic photosynthesis, and the basic structure of their photosynthetic machinery is highly conserved. The Photosystem (PS) II is composed of the core proteins, D1 and D2, which bind all the redox-active components involved in electron transfer of PSII. In addition to the D1 and D2 proteins, PSII contains the inner chlorophyll-binding antenna proteins CP43 and CP47, and the Cyt b_{559} proteins PsbE and PsbF. Moreover, several low molecular mass proteins are required for proper function and assembly of the PSII dimer. Although the ultimate function of the oxygen evolving complex (OEC) of PSII is very similar in the eukaryotic organisms and the prokaryotic cyanobacteria, the detailed characteristics of the individual OEC proteins differ between these organisms [1,2]. Another difference in PSII between the eukaryotic and prokaryotic organisms concerns the light harvesting machinery. In higher plants, light is harvested by the membrane embedded light harvesting complex, whereas in cyanobacteria light energy is captured by the soluble phycobilisome antenna. Naturally also the compartmentalization of the cell differs dramatically between these organisms. In higher plants and algae photosynthesis takes place in chloroplasts. Although the key proteins of photosynthesis (e.g., D1 and D2) are encoded by the chloroplast genome, the vast majority of the chloroplast proteins are encoded by the nuclear genome, translated on the cytosolic ribosomes and translocated to the chloroplast. In cyanobacteria, no such elaborate signalling and trafficking of the proteins between the distinct organelles are needed, although more and more evidence is accumulating about the early steps of PSII biogenesis apparently taking place at the plasma membrane, which necessitates extensive membrane transfer processes [3–6]. Another distinct difference is the structural organization of the thylakoid membrane. In chloroplasts of higher plants, the thylakoid membrane is laterally segregated into distinct granal stacks connected by the stroma lamellae, in green algae such heterogeneity is less strict while in cyanobacteria the thylakoid membrane is peripherally organized in distinct layers around the cells.

The PSII of all organisms is prone to light-induced oxidative damage due to the highly oxidative chemistry of water splitting [7,8]. The D1 protein is the primary target of the damage, and it is sacrificed in order to avoid complete inactivation and disassembly of PSII. Therefore, under normal photosynthetic growth conditions the D1

Abbreviations: OEC, oxygen evolving complex; PEP, plastid encoded RNA polymerase; PS, photosystem

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protein, encoded by the *psbA* gene, is constantly degraded and re-synthesized in the multistep process called PSII repair cycle [9–11]. Although the D1 protein in higher plants and green algae is encoded in the chloroplast genome, the repair process requires assistance by a multitude of nuclear encoded proteins and therefore the regulation of these two genetic systems must be well coordinated. Thylakoid heterogeneity in chloroplasts provides further complexity to the repair process. Here, we describe the specific features and fundamental differences of the *psbA* gene expression in cyanobacteria and chloroplasts of green algae and higher plants, which have adopted distinct differences in the regeneration of the PSII reaction center protein D1. In the chloroplasts of higher plants and green algae the prevalent mechanism for the regulation of *psbA* gene expression, as well as many other chloroplast genes, seems to be the control of translation, whereas the main regulatory step in cyanobacteria is transcription (Fig. 1). Specific for plants, on the other hand, is the regulation of *psbA* gene expression by D1 protein phosphorylation.

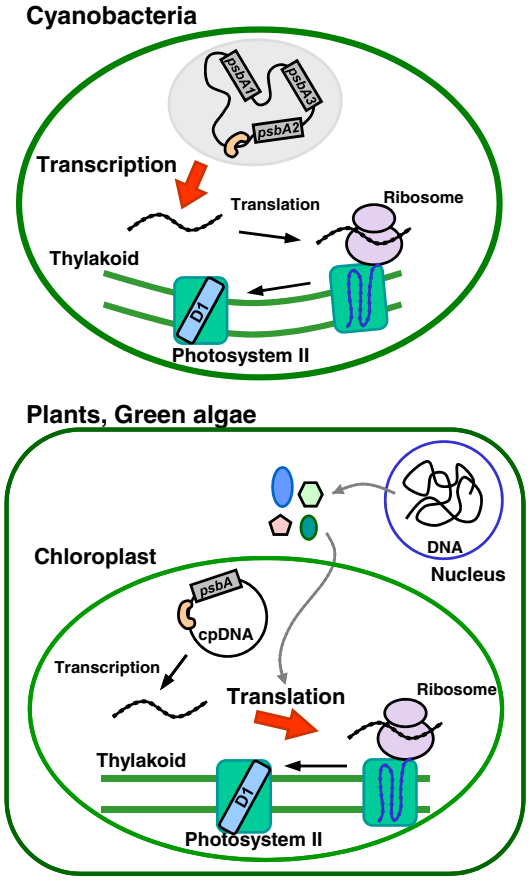


Fig. 1. General scheme depicting the major regulatory steps of *psbA* gene regulation in cyanobacteria and chloroplasts of green algae and higher plants. In cyanobacteria, a single genome consisting of a number of circular DNA molecules encode all the structural and regulatory proteins that constitute the cyanobacterial cell. Photosynthetic pigment–protein complexes are embedded in the concentrically layered thylakoid membranes. Transcription is the main regulatory step in the control of cyanobacterial gene expression, depicted as a thick red arrow, and only few proteins are so far known to regulate the activity of transcription. The photosynthetic machinery of green algae and higher plants is encoded by both the nuclear and chloroplast (cp) genomes. The production of photosynthetic proteins is strictly co-regulated in both compartments in order to guarantee optimal assembly and function of the chloroplasts. The main regulatory step in chloroplast gene expression is the translation of proteins from a stable, pre-existing pool of transcripts (depicted as thick red arrows). The stability of mRNA and efficiency of translation are regulated *via* binding of various nuclear-encoded proteins to the 5′ and 3′ UTRs of the genes. Major regulatory steps in each organism are indicated by the thickness of the arrows.

2. *psbA* gene expression in cyanobacteria

In all cyanobacteria studied so far, a small gene family with two to six members codes for the D1 protein [12]. In many cyanobacteria, one of the D1 isoforms dominates under standard growth conditions, while upon shift of the cells to adverse conditions another, stress-induced isoform of the D1 protein is expressed (for a review, see [12]). The functional properties of the different isoforms are known to differ from each other, for example in respect to light tolerance [13–18]. Two cyanobacterial species, *Synechococcus* 7942 and *Synechocystis* sp. PCC 6803, are the most popular cyanobacterial model organisms for studies of PSII function and assembly. The *psbA* gene expression in these species possesses a very different mechanistic principle. In *Synechococcus* 7942, the D1:1 protein is encoded by the *psbAI* gene, and the stress-induced D1:2 by the *psbAII* and *psbAIII* genes [14,17,19–22], while for a long period of time only one type of D1 ($D1_m$) protein, encoded by the *psbA2* and *psbA3* genes, was detected in *Synechocystis* sp. [12,23]. However, it has been recently shown that actually two forms of D1 protein exist also in *Synechocystis* sp., as the "silent" *psbA1* gene has been proven to be induced under microaerobic conditions [24,25]. Recent studies using other cyanobacterial species, such as *Thermosynechococcus elongatus* [18,26–28], *Gloeobacter violaceus* [29], *Anabaena* 7120 and *Synechococcus* WH 7803 [30] have provided evidence that the principle dogma of cyanobacterial *psbA* gene expression (one "standard" D1 isoform expressed under normal conditions and a functionally different stress-induced form dominating upon adverse conditions) is valid also in other species.

2.1. Regulation of psbA gene expression in cyanobacteria at the level of transcription

Regulation of the *psbA* gene expression has been investigated for decades, and considerable amount of knowledge about the details in transcriptional regulation has emerged. Yet, the ultimate mechanisms behind the transcriptional regulation remain poorly characterized. Under normal growth conditions (ca. 50 μmol photons m^{-2} s^{-1}) the *Synechocystis psbA2* gene produces ca. 90% of the *psbA* transcripts, while the *psbA3* gene produces only 3–10% [23,31]. Intense illumination, as well as exposure of the cells to UVB radiation, increases the transcription of *psbA2*, but especially that of the *psbA3* gene, which is followed by enhanced translation of the *psbA* transcripts [32–35]. An increase in D1 synthesis rate is required to balance the light intensity-dependent damage and degradation of the D1 protein, and actually not only the light intensity but also the rate of D1 synthesis regulates the *psbA* gene transcription in *Synechocystis* 6803 [36–40]. In *Synechococcus* 7942, a majority (>80%) of the total *psbA* transcript pool originates from *psbAI* under low light conditions (125 μmol photons $m^{-2}s^{-1}$) [41,42]. Shift of the cells to intense illumination (750 μmol photons $m^{-2}s^{-1}$) results in a decrease of *psbAI* transcription, while the transcription of *psbAII* and *psbAIII* increases [41–44]. Transcriptional changes are directly reflected at translational level, and the interchange of the D1:1 form by D1:2 is required for proper acclimation of cells to changing environmental cues [13,15,20–22,45].

All the *psbA* genes expressed under standard or high light conditions produce transcripts of 1.2 kb with the 5′ ends comprising 49 to 88 bases upstream from the coding region [19,23], while the transcription start site of the *psbA1* gene in *Synechocystis* sp. PCC 6803 has not been characterized yet. In *Synechocystis* 6803 and *Synechococcus* 7942, the conserved −35 and −10 elements are present in the upstream regions of the *psbA2*/*psbAII* and *psbA3*/*psbAIII* genes, respectively [23,44]. The promoter regions of *psbA1* and *psbAI* genes differ from those of other *psbA* genes and contain atypical fingerprints [19]. Although the principal sigma factor (Group 1) has been shown to recognize the hexameric −35 and −10 regions located in the promoter region of the *psbA* genes [46–48], the light responsive expression requires also the presence of SigB, SigD and SigE [49–52].

In addition to the promoter of the *psbA* gene, the tertiary structure of DNA (AT tracts) in the upstream region of the *psbA* genes [53,54] as well as the regulatory elements upstream from the promoter [44] and within the transcribed region is known to enhance gene expression and mediate responses to light.

In *Synechococcus* 7942 various regulatory proteins bind to the upstream region of the *psbAI* gene [55,56]. Gel mobility shift experiments have demonstrated binding of a *de novo* synthesized protein factor, essential for the transcriptional activation, to the 5′ end of the *psbAI* coding region [57], while at least one of the regulatory factors is known to be shared with *psbAI* and *psbAII* [56]. The PsfR protein is one of the few identified regulatory factors, as the overexpression of *psfR* has been shown to enhance the expression of *psbAI* without an effect on *psbAII* and *psbAIII*. However, knocking out of the *psfR* gene does not stall the expression of the *psbAI* gene, and therefore it has been suggested that PsfR does not bind directly to the *psbAI* promoter but might regulate gene expression *via* protein–protein interactions. Additionally, the degradation products of the D1:1 protein have been shown to bind the upstream region of the *psbAI* gene, implying that the turn-over rate of the D1 protein might have an impact on its own synthesis [58]. The CmpR protein, which is involved in cyanobacterial carbon concentration mechanisms [59], binds to the enhancer elements of the *psbAII* and *psbAIII* genes and regulates the *psbA* gene expression *via* an uncharacterized mechanism. Moreover, the AT-rich region between the −10 basal promoter and the Shine-Dalgarno sequence of the *psbAII* gene functions as a negative element, which might serve as a binding site for regulatory factors and/or ribosomes controlling the accumulation of *psbAII* transcripts [60].

In *Synechocystis* sp. PCC 6803, much less is known about the trans-acting factors regulating the *psbA* gene expression, and no proteins enhancing the expression of the *psbA* genes have been characterized. In contrast, it has been shown that putative repressor proteins bind to the upstream region of the *psbA2* and *psbA3* genes in darkness, which is in line with the fact that light is required for the accumulation of *psbA* transcripts [61,62]. Although transcription of the *psbA* genes seems to require *de novo* synthesized protein factors [63], the transcription of the *psbA* genes during the recovery process after photoinhibitory treatment of *Synechocystis* 6714 is not prevented by inhibition of translation [33].

A great number of studies have implicated that the redox state of *Synechococcus* cells regulates the *psbA* gene expression, while there is no such consensus yet concerning the redox-regulation of *Synechocystis* 6803 *psbA* gene expression. In *Synechococcus* 7942, conditions resulting in accumulation of thiol reductants, such as direct addition of thiol reductant DTT to cell culture [64], or exposure of cells to high light intensity [15,65], UVB radiation [16], anoxia [66] or shift of the cells to low temperature [65] induce the expression of the *psbAII* and *psbAIII* genes. Addition of an electron transfer inhibitor DCMU or DBMIB, in contrast, does not cause any changes in *psbA* gene expression upon low light conditions, but both inhibitors dramatically reduce the expression of the *psbAII and psbAIII* genes when added upon a high-light shift. These results have been taken as an indication that the thiol redox state, and not the redox state of the plastoquinone pool, regulates the *psbA* gene expression in *Synechococcus* 7942 [64]. Besides redox regulation, also other factors may control the *psbA* gene expression in cyanobacteria. In *Synechococcus* 7942, the expression of the *psbA* genes has been suggested to be controlled *via* a blue light photoreceptor [67–69], possibly by the function of NblS, which is a putative histidine kinase [70].

The *psbA* gene expression in *Synechocystis* sp. is not under the control of the thiol redox state of the cell (unpublished results from our laboratory). Instead, lots of contrasting data concerning the involvement of the intersystem redox status in the regulation of *psbA* gene expression in *Synechocystis* have been published during the past 10 years. Several studies have suggested that the reduction of Q_A, plastoquinone pool, or the Cyt b_6f complex has an impact on accumulation of *psbA* transcripts [40,71–73]. RppA, which acts as a response regulator of a two-component system, is a putative candidate to mediate the changes in the redox poise to the expression of photosynthetic genes, including *psbA* [40]. However, other studies implicate that the redox state of the electron transfer chain is an unlikely candidate to carry information for regulation of *psbA* expression, since the action spectrum of *psbA* transcription resembles rather the action spectrum of photoinhibition than that of PSII activity or photosynthesis [74]. The major determinants of the cyanobacterial *psbA* gene expression are presented schematically in Fig. 2a.

2.2. Post-transcriptional regulation of the psbA gene expression in cyanobacteria

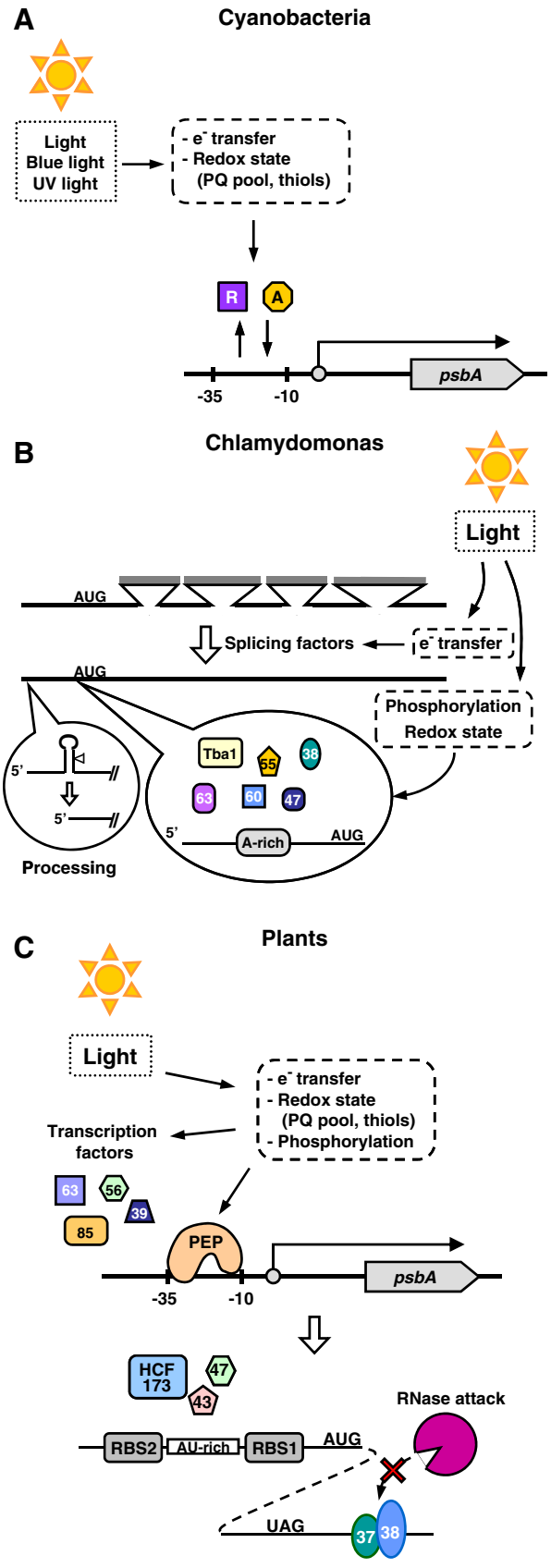
As typical for the transcripts in most prokaryotes, the half-life of the *psbA2* and *psbA3* mRNA in *Synechocystis* 6803 is rather short: under illumination the half-life is around 10–20 min, and independent of both the light intensity and the rate of PSII electron transfer [32,33,37,71,72,75]. It is also important to note that although the translation of the *psbA* messages ceases in darkness, the stability of the *psbA* transcripts increases remarkably [23,32,62,76]. The stabilization is not dependent on light *per se*, but rather on the cessation of photosynthetic electron transfer [32,35,62,72]. Other factors, such as polyamines, have been suggested to affect the stability of *psbA* transcripts as well [76]. In *Synechococcus* 7942, the untranslated leader regions of the *psbA* genes as well as parts of the coding region determine the stability of the *psbA* mRNA [42,77]. Especially the region encoding the first membrane span of the D1 protein is of utmost importance for *psbA* mRNA turnover, probably due to the fact that pausing ribosomes protect the mRNAs from degradation [77]. *psbAI* and *psbAIII* transcripts are destabilized upon exposure of *Synechococcus* cells to high light intensity ($T_{1/2}$ = 10–12 min), whereas the *psbAII* transcripts are long-lived and apparently not subject to post-transcriptional regulation [42]. Neither do *Synechococcus psbA* transcripts show any dark-stabilization, which is in contrast to *Synechocystis* sp. PCC 6803. In both species, however, the *psbA* messages are stabilized upon prolonged photoinhibitory treatment [13,33], but the changes in the transcript amounts under these extreme conditions are not followed by corresponding changes in translation [13].

It has been shown that the existing *psbA* messages in *Synechococcus* 7942 are always associated with ribosomes [78]. This suggests that the membrane targeting of nascent D1 protein ribosome complexes might be an important determinant for D1 protein synthesis, while apparently the initiation of translation is not strictly regulated [78]. Elongation of translation is an important regulatory step also in *Synechocystis* 6803. The *psbA* transcripts are attached to ribosomes even in the dark, and the translation of the D1 protein continues up to a distinct pausing site. The newly formed ribosome-nascent D1 chain complexes are targeted to the thylakoid membrane only upon illumination, and therefore the synthesis of the D1 protein can be completed only in light [79]. This indicates that in line with *Synechococcus*, also in *Synechocystis* 6803 the translational elongation is an important regulatory step in expression of the *psbA* genes [79]. Moreover, singlet oxygen generated during photosynthetic light reactions has been shown to arrest the translational elongation process of the D1 protein, while the lack of chlorophyll affects the initiation of *psbA* translation [80].

3. *psbA* gene expression in eukaryotes

The chloroplasts of higher plants and green algae contain a single *psbA* gene, which produces a very stable pool of transcripts. Translation of a number of chloroplast transcripts, including *psbA*, is induced by light, and it is nowadays evident that in contrast to

cyanobacteria, the mRNA processing, and especially the control at the level of translation are the major steps in the regulatory network of the *psbA* gene expression in chloroplasts. The unifying concept might be that in all investigated species the D1 protein is made available according to the needs of the cell, yet using a variety of strategies depending on the genetic and physiological context of the system. Nevertheless, distinct differences have been reported in *psbA* gene expression in chloroplasts of green algae and higher plants, which are described and discussed below.



3.1. Highlights of psbA gene expression in Chlamydomonas reinhardtii

Chlamydomonas reinhardtii (hereafter referred as Chlamydomonas) is a suitable model organism for photosynthesis research, since this unicellular eukaryotic green alga contains only a single giant chloroplast. Routine transformation methods have been developed for Chlamydomonas, and it can survive on acetate-containing media even if the photosynthetic machinery has been inactivated due to mutations. Indeed, an impressive amount of knowledge has accumulated concerning the regulation of *psbA* gene expression in the chloroplasts of Chlamydomonas.

The *psbA* gene of Chlamydomonas is located within the inverted repeat region of the chloroplast genome, and therefore it is present in two identical copies [81]. Although in Chlamydomonas transcription of the *psbA* gene increases slightly upon illumination, it is apparently not under strict control, and instead it has been reported that the *psbA* gene is constitutively expressed [82]. The *psbA* mRNA exists in two forms in the chloroplasts of Chlamydomonas: a larger form with a 5′ UTR of 91 nucleotides and the predominant shorter form with a leader of 36 nucleotides [81,83,84]. The larger form has been hypothesized to be a precursor of the shorter mRNA *via* 5′ processing [85]. Whether the several stem–loop structures predicted to be formed in the 5′ UTR of the *psbA* mRNA are involved in the processing remains to be resolved [81]. The 3′ UTR of *psbA* mRNA contains an inverted repeat sequence, which is able to fold into a stem–loop structure. Although the loop resembles bacterial transcription terminators, it has been shown both *in vitro* and *in vivo* that these elements rather function as 3′ end processing signals than terminators [86]. The *psbA* gene is interrupted by four Group I introns [81], and three of these introns have been shown to self-splice under non-physiological conditions *in*

Fig. 2. Major regulatory steps of the *psbA* gene expression in prokaryotes and eukaryotic chloroplasts. (A) Regulation of *psbA* gene expression in cyanobacteria occurs mainly at the level of transcription. Changes in environmental conditions (e.g., light quality and quantity) affect the redox state of the cell and the rate of electron transfer, which regulate the binding of various trans-acting regulatory factors (R denotes for repressors, and A for activators) to the promoter region of the *psbA* gene. In addition to the binding of repressors and activators, which determine the transcriptional activity of the *psbA* gene, also the D1 degradation fragments may act as transcriptional regulators. Also stability of the *psbA* mRNA as well as translational regulation (not shown in the figure) controls the *psbA* gene expression in cyanobacteria. (B) The main regulatory steps in *psbA* gene expression in *Chlamydomonas reinhardtii* are the mRNA processing and initiation of translation. Efficiency of splicing is determined by nuclear encoded splicing factors regulated by light and the rate of electron transfer. The stem–loop structures in the 5′ and 3′ termini of the *psbA* mRNA are probably involved in the processing of the transcript, which results in formation of two distinct *psbA* transcripts. Light quality and quantity cause changes in phosphorylation and the redox state of several RNA binding proteins (63, 60, 55, 47 and 38 kDa proteins and Tba1, see 3.1 for details), which in turn regulate the efficiency of translation initiation. The *psbA* gene is currently thought to be constantly transcribed, and so far no distinct transcription factors have been identified. (C) Regulation of the *psbA* gene expression in higher plants. Although there is generally no strict limitation of D1 synthesis at transcriptional level, the transcription of the *psbA* gene is nevertheless affected by rapid changes in the quality and quantity of light affecting the redox state of chloroplasts and the phosphorylation status of the plastid encoded RNA polymerase (PEP). Transcription activity is determined by the function of PEP and various transcription factors (39, 56, 63 and 85 kDa). As *psbA* gene is an intronless chloroplast gene, control of splicing does not play a role in regulation of *psbA* gene expression in the chloroplasts of higher plants. In the light, *psbA* mRNA translation initiation complexes are formed but the synthesis of D1 protein is mainly regulated at the level of translation elongation. A few regulatory proteins (HCF173, 47, 43, 38 and 37 kDa) identified this far bind to the 5′ UTR and the 3′ end of the *psbA* transcript and control the *psbA* gene expression *via* mechanisms that still remain largely uncharacterized (see 3.2.2 for details). The different shapes of the regulatory proteins reflect the variety of (unrelated) proteins involved in the regulation of *psbA* gene expression.

vitro [87]. *In planta*, the splicing of *psbA* mRNA in Chlamydomonas is dependent on light and photosynthetic electron transfer: unspliced transcripts accumulate in darkness, while efficient splicing of each intron occurs within the first hour of light exposure (Fig. 2b). Moreover, inhibition of electron transfer with DCMU or DBMIB results in cessation of splicing, while depletion of ATP does not have any effect [88]. The specific light-dependent splicing of the *psbA* transcript implies that apparently one or more *psbA* specific, light-activated splicing factors might be present in Chlamydomonas chloroplasts, and indeed nuclear loci with distinct effects on splicing have been identified [89].

In addition to mRNA processing, the light-induced accumulation of the D1 protein in Chlamydomonas is regulated at the level of translation initiation [90–92]. A complex of four major and several minor nuclear encoded proteins bind to the to stem–loop structure within the 5′ UTR of the *psbA* mRNA thereby activating the synthesis of the D1 protein [93] (Fig. 2b). These proteins include a chloroplast poly(A) binding protein cPAB1 (RB47) [94,95], a protein disulfide isomerase cPDI (RB60) [96,97], an RNA binding protein RB38 [98,99], and the RB55 protein [98,100]. Binding of these proteins to the *psbA* mRNA is dependent on light and protein phosphorylation [93]. In darkness, the cPDI (RB60) protein is phosphorylated *via* an ADP-dependent kinase, which in turn results in a release of the protein complex from the 5′ UTR of the *psbA* mRNA, and cessation of translation [93]. In the light, cPDI reduces the cPAB1 protein, which results in tight binding of cPAB1 to the A-rich sequences of the 5′ UTR of *psbA* mRNA, and in the initiation of translation [96,101]. The Tba1 (translational affector of *psbA*) protein, a putative stromal oxido-reductase, enables cPAB1 to bind *psbA* mRNA and the subsequent loading on ribosome, implying that Tba1 functions as another redox regulator of cPAB1 [102].

In turn, the RB38 protein interacts with the uridine-rich regions within the 5′ UTR of *psbA* mRNA [99]. Recently, it was shown that the accumulation of the *rb38* and *rb60* transcripts is regulated by red light (although the red light receptor has not been identified in Chlamydomonas yet) and calmodulin [103], while the expression of the *rb47* is rather regulated at translational level by an unknown chloroplast signal [98,103]. Moreover, the RBP63 protein (RNA binding protein of 63 kDa) has been shown to bind to the 5′ UTR of the *psbA* transcript *via* an element located between the nucleotides −36 and +1. Also a tract of seven consecutive A-residues located 14–8 nucleotides upstream of the start codon has an impact on binding [104]. Other determinants, such as secondary structures might further facilitate recognition. It is worth noting that along with the *psbA* transcripts, the RBP63 protein is associated with the thylakoids [104–106].

Yet another level of *psbA* gene regulation was demonstrated by the study showing that in the absence of assembly partners (control by epistasy of synthesis), the *de novo* D1 synthesis is hindered [107,108]. This was shown to be due to translational autoregulation mediated by the *psbA* promoter together with the 60 first nucleotides downstream of the initiation codon [92].

3.2. Regulation of psbA gene expression in higher plant chloroplasts

Various plant species, both dicots and monocots (i.e., spinach, mustard, tobacco, barley), have been used as model organisms in studies of regulation of the *psbA* gene expression. Although some differences have been reported between dicots and monocots, or between different species, we describe below the current knowledge concerning the *psbA* gene expression in general in the chloroplasts of all studied higher plants. Several lines of evidence indicate that as in cyanobacteria and chloroplasts of Chlamydomonas, the expression of the *psbA* gene in all higher plant chloroplasts is regulated *via* a complex network including transcriptional control, regulation of mRNA stability, and in particular the control at the level of translation (Fig. 2c).

The single *psbA* gene found in higher plants is located in a large single copy region of the chloroplast genome, and it does not contain introns. The promoter of the *psbA* gene has been analyzed in detail. It consists of a −10 and −35 region interspersed by a single TATA-box-like element, and the transcription is initiated in a single defined site [109–112]. In mustard, however, the *trnK* gene is read-through into the *psbA* coding region resulting in two *psbA* transcripts with different 5′ leader regions [113,114]. The *psbA* gene is predominantly transcribed by the plastid encoded RNA polymerase (PEP) [115–117], and the transcription activity is affected by the developmental stage of the plant [111,112,118]. Although the transcription of also the plant *psbA* gene is somewhat induced by light [119–121], high amounts of *psbA* mRNA are present in all green tissues of higher plants even in darkness due to a strong promoter [122] and a very stable RNA [123]. The D1 protein, however, is synthesized only in response to light [124–129].

3.2.1. Transcriptional regulation in plants

Intersystem redox poise has been shown to regulate the transcriptional activity of various photosynthetic genes. Upon conditions of reduced plastoquinone pool, the transcription of genes encoding the PSI subunits is enhanced, while upregulation of the *psbA* gene expression occurs under conditions leading to an oxidized plastoquinone pool [130–132]. It has been proposed that the redox signals originating from the photosynthetic machinery embedded in the thylakoid membrane might be transferred to the gene expression level *via* a phosphorylation cascade [133]. Phosphorelay might be transduced *via* the chloroplast sensor kinase, which has been shown to regulate the expression of the *psaA* gene [134], or *via* phosphorylation of PEP and various transcription factors [135–138], reviewed in [139]. Recently, Steiner et al. [140] showed that a set of four proteins (39, 63, 56 and 85 kDa) are specifically bound to the promoter region of the *psbA* gene, and that various other trans-acting factors are shared with *psbA* and *psaA* genes. The DNA binding proteins are apparently regulated by a synergistic action of phosphorylation and thiol redox signalling, which collectively form a complex regulatory network [140,141].

3.2.2. Translational regulation is the most prominent step in the regulation of higher plant psbA gene expression

Translation of the D1 protein takes place in membrane-bound polysomes [121,127], and the D1 protein is co-translationally inserted into the thylakoid membrane *via* the cpSecY translocation channel [142–146], reviewed in [147]. The accumulation of the D1 protein is tissue-specific and light-dependent, and moreover, affected by the developmental stage of the plant [123,125,127,129,148,149]. The accumulation is partly controlled *via* the untranslated region of the *psbA* mRNA at the level of translational initiation [129,150,151], and is known to be dependent on several cis-elements, including RBS1, RBS2 and an AU-box (UAAAUAAA) [152].

RBS1 and RBS2 have been suggested to act as a bipartite Shine-Dalgarno sequence for ribosome binding, and the AU-box located between RBS1 and RBS2 probably binds various trans-acting factors [152]. One of these factors is HCF173 (high chlorophyll fluorescent; At1g16720), which is a chloroplast-targeted, nuclear-encoded protein, that belongs to a superfamily of the short-chain dehydrogenases/reductases [153]. HCF173 is needed for the efficient translation of the *psbA* mRNA, and it probably acts during the initiation of translation [153]. Moreover, it may directly or undirectly affect also the stability of the *psbA* mRNA. Eibl et al. [151] have further described a stretch of 17 terminal nucleotides in the 5′ UTR of the *psbA* mRNA that influences translation efficiency. Also, 43 kDa and 48 kDa proteins binding to the 5′ UTR, "the central protein binding element" (−49 to −9), of the *psbA* mRNA, have been identified [154–156]. The 43 kDa protein is a chloroplast homologue of the *Escherichia coli* ribosomal protein S1 [156], and it binds specifically to the U-rich region of the

spinach 5′ UTR of the *psbA* mRNA, while it remains to be seen whether the 47 kDa protein might be an ortholog of the cPAB1 protein in Chlamydomonas [94,154,157].

In addition to proteins that bind to the 5′ UTR of the *psbA* mRNA, also proteins interacting with the 3′ UTR have been described. Two RNA binding proteins of 37 and 38 kDa have been shown to bind specifically to the 30 nucleotide region of the *psbA* mRNA immediately downstream from the translation termination codon, possibly as a heterodimer [155]. It has been suggested that the 37/38 kDa proteins might protect the *psbA* mRNA from nuclease attack, or they might modulate translational activity [155]. The ultimate function of these proteins, however, remains to be elucidated. The 5′ and 3′ UTRs may additionally interact with each other, e.g.,*via* binding of regulatory proteins resembling nuclear poly(A) binding proteins [151], such as Chlamydomonas RB47 [94,95], thereby ensuring the maximal level of translation.

Although the abundant *psbA* transcripts are associated with the polysomes even in darkness [127], the D1 protein starts to accumulate only upon illumination [127,158,159], which indicates that in addition to translation initiation, also elongation of translation is strictly regulated in the chloroplasts of higher plants. During translational elongation, the ribosomes pause at several distinct sites along the *psbA* mRNA, which probably allows binding of chlorophyll and stabilization of the D1 protein [128,159,160]. Moreover, successful and efficient elongation of the D1 protein translation requires maintenance of transthylakoid proton gradient [144,161] as well as intact electron flow through the electron transfer chain [144,162]. Similarly to Chlamydomonas, control by epistasy of synthesis [163] seems to be valid in the chloroplasts of higher plants, and the interaction of the D1 nascent chain with the D2 protein is needed for successful cotranslational insertion of the D1 protein into the thylakoid membrane [142,144,164,165].

4. The PSII repair cycle

During the *de novo* assembly of the PSII complex all the subunits are synthesized and assembled into functional dimers, while in the process of PSII repair primarily the D1 protein is degraded and resynthesized to replace the damaged D1 copy, the other PSII proteins possessing significantly slower turnover rate and being mainly recycled [9–11,164] (Fig. 3). In the PSII repair cycle of plant chloroplasts, the post-translational phosphorylation of the D1 protein

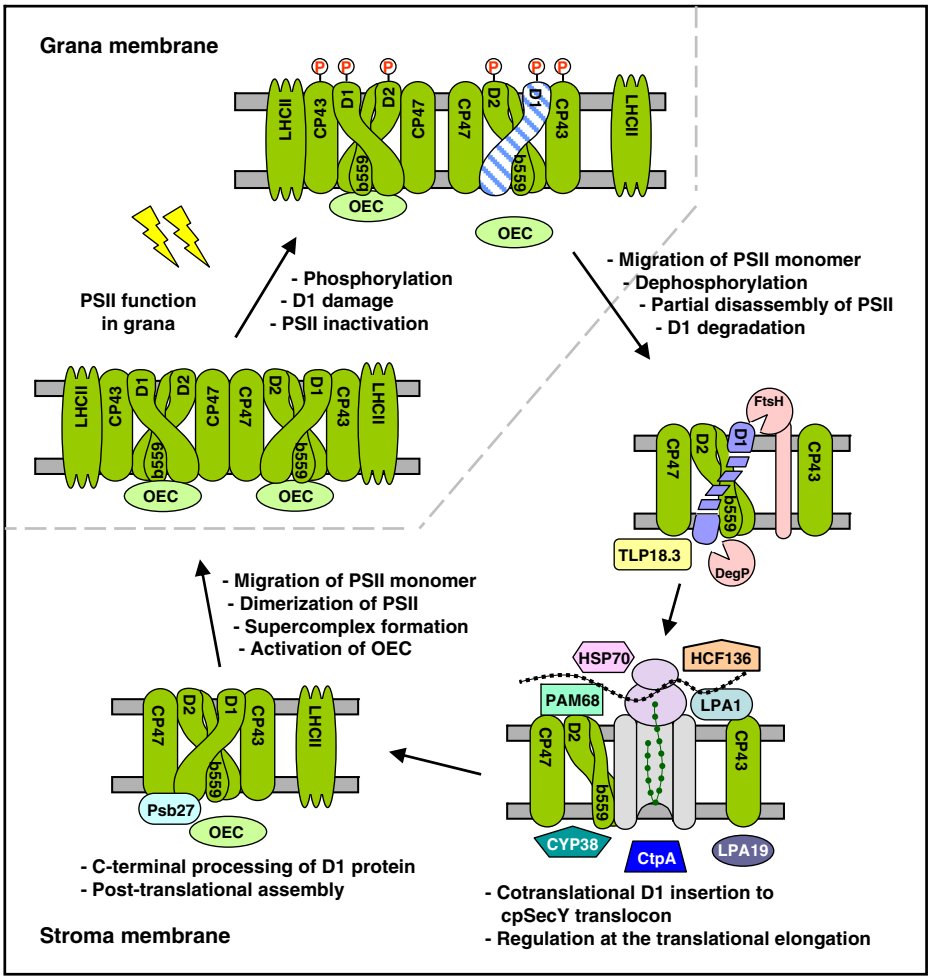


Fig. 3. Repair cycle of the PSII D1 protein. Light induces phosphorylation and damage of the D1 protein (shown as stripes). The damaged PSII monomers migrate from the grana stacks to the stroma thylakoids, where the D1 protein is dephosphorylated and subsequently degraded by the FtsH, which functions as a hexamer, and DegP proteases assisted by the TLP18.3 protein. The D1 proteolysis is followed by a partial disassembly of the PSII complex and co-translational insertion of the newly synthesized D1 into the thylakoid membrane. The LPA1 protein is required for efficient translation of the *psbA* transcripts and insertion of the new D1 copy into PSII. CYP38 assists in the proper folding of the D1 protein and thus facilitates the proper assembly of the oxygen evolving complex. PAM68, shown to interact with the various PSII assembly factors and early PSII assembly intermediates, affects the stability and maturation of the D1 protein thereby promoting the early steps in PSII biogenesis. HCF136 and HSP70 are probably also assisting in the *psbA* mRNA translation, proper folding of the D1 protein and the assembly of the PSII complex. The C-terminus of the D1 protein is processed by the CtpA protease with the aid of the LPA19 protein. Thereafter, the Psb27 protein mediates the assembly of the oxygen evolving complex to the PSII complex. When the assembly of PSII core monomers is completed, they migrate back to the granal stacks, where supercomplex formation, dimerization and activation of the complexes occur.

plays a crucial role in *psbA* expression. The phosphorylation of the damaged D1 protein is required for fluent migration of PSII complexes from the grana stacks to the nonappressed stroma lamellae, where the D1 protein is dephosphorylated and subsequently degraded [166–173]. Only after degradation, the synthesis of the D1 protein can be completed and the co-translational insertion into the thylakoid membrane takes place. It is worth noting that although the PSII repair cycle occurs at the thylakoid membrane, it has been shown that the *de novo* D1 synthesis in Chlamydomonas takes place in discrete stromal regions (T zones) near the pyrenoid [174]. It is unlikely that the PSII repair, different to the *de novo* assembly of PSII, involves any specific compartmentalization or phosphorylation of PSII core proteins in cyanobacteria.

A number of auxiliary proteins are involved in the PSII repair, many of which are conserved in chloroplasts and cyanobacteria. The lumenal DegP [169,175,176] reviewed in [177] and [178], and the stromal FtsH [179–182] proteases (see Zhang et al. this issue) are responsible for the D1 degradation, and the degradation process is assisted by the lumenal auxiliary protein TLP18.3 [183]. The D1 proteolysis is followed by a partial disassembly of the PSII complex. Thereafter, the newly synthesized D1 copy is co-translationally inserted into the thylakoid membrane [145], and concomitantly chlorophyll molecules are ligated to the D1 protein [160].

The three proteins shown to have specific impact on D1 synthesis and co-translational insertion of the newly synthesized copy into the PSII complex are LPA1 (Chlamydomonas REP27), CYP38, and PAM68 (*Synechocystis* Sll0933). LPA1 is an integral thylakoid membrane chaperone required for efficient translation of the *psbA* transcripts and insertion of the new D1 copy into PSII [184], while PAM68 affects the stability and maturation of the D1 protein [185]. PAM68 interacts with LPA1 (and other PSII assembly factors) and is associated with the early assembly intermediates of PSII suggesting that PAM68 promotes the early steps in PSII biogenesis [185]. CYP38, in turn, is a lumenal immunophilin ensuring the proper folding of D1 protein into PSII complexes, which thereby facilitates the correct assembly of the oxygen evolving complex [186–189]. AtHCF136 [190,191] and HSP70 [192–198] are probably also assisting in the *psbA* mRNA translation, proper folding of the D1 protein and the assembly of the PSII complex. Also a number of other auxiliary proteins are involved in distinct steps of PSII assembly, but they have not shown any direct interaction with the D1 protein [11,199]. The C-terminal processing of the D1 protein by the lumenal CtpA protease, aided by the Psb27 homolog LPA19, is a prerequisite for the formation of functional PSII complexes capable of oxygen evolution [200–207]. When the assembly of PSII monomers is completed, the PSII complexes migrate back to the granal stacks (in chloroplasts), where dimerization and supercomplex formation occur with the assistance of ALB3 [208–212], Psb29 [197,213,214], and FKBP20-2 [215]. In addition to various auxiliary proteins required for the optimal repair of the light-damaged PSII complex, several low molecular mass PSII subunits are indispensable for the stabilization, assembly, dimerization and supercomplex formation of PSII (reviewed in [216]).

5. Conclusions

All organisms performing oxygenic photosynthesis face an apparent paradox, as light induces damage to the D1 protein of PSII. The basic structure of PSII is very similar in prokaryotic cyanobacteria and in chloroplasts of higher plants or green algae, but during the evolution each group has been furnished with specific strategies to guarantee efficient synthesis of the D1 protein as a response to ever-changing environment. A vast number of studies concerning the *psbA* gene expression in cyanobacteria have revealed that although the transcription is the major regulatory step, also translational regulation plays a role. Still, however, there is a scarcity of detailed knowledge concerning the trans-acting factors regulating the *psbA* gene expression. In chloroplasts of Chlamydomonas the regulation of *psbA* gene expression occurs at the level of mRNA processing and translation, and the composition and function of the regulatory proteins affecting the translational efficiency have been relatively well characterized. In the chloroplasts of higher plants, a constant pool of *psbA* mRNA initiation complexes exists, but the synthesis of D1 can be completed only when a D1-less PSII complex is available to accept the integration of a new D1 protein. This suggests that translational elongation is the main determinant of the *psbA* gene expression in higher plants. Moreover, phosphorylation of the D1 protein has a major impact on regulation of the D1 degradation, which in turn is a prerequisite for efficient D1 synthesis in the chloroplasts of higher plants. Further research is required and will reveal novel aspects of *psbA* gene regulation at the levels of transcription, translation and assembly of the PSII complexes, which with no doubt is a fascinating phenomenon with great biological importance.

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