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Review Article

Roles of Light-Harvesting Complex Stress-Related Proteins in the Stress Responses of *Chlamydomonas*

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Abstract: Light is very important for photosynthesis in plants. However, excess light can result in photodamage to the photosynthetic apparatus. Via nonphotochemical quenching (NPQ), the oxidative stress caused by excess light energy can be counteracted by photoprotective mechanisms that evolve photosynthetic/oxygenic organisms. Energy-dependent quenching (qE), as the major NPQ component, relies on the accumulation of specific proteins that are termed light-harvesting complex stress-related (LHCSR) proteins in microalgae and mosses. LHCSRs have been reported to participate in adaptation to diverse environmental stresses, including excess light. In this review, we discuss the identification of LHCSRs in *Chlamydomonas* and the basic biochemical properties and functions of LHCSRs in acclimation to environmental stresses such as excess light and salt stress. We further review the potential interactive factors and upstream regulators of LHCSRs in *Chlamydomonas*, aiming to explore the underlying mechanism of *LHCSRs* in adaptation to multiple environmental stresses. We also discuss the evolution of LHCSRs in green algae and mosses and tentatively speculate about their participation in the adaptation to environmental change of the Earth. Work on *Chlamydomonas* LHCSR could provide clues to analyze the roles of LHCSR in both green algae and mosses. Thus, we offer an overview of current knowledge on the characteristics and functions of *Chlamydomonas* LHCSRs, which could shed new light on their detailed studies in both green algae and moss in the future.

Keywords: *Chlamydomonas,* LHCSR, Adaptation, Environmental Stress, Evolution

1. Introduction

Algae, unicellular organisms with no roots and other nonphotosynthetic organs, are very efficient in converting solar energy into biomass. Although higher plants and microalgae share the same basic functions of photosynthesis [1], microalgae do not need arable soils for their growth and have no competitive relation with food crops. Microalgae are highly rich in lipids, and over 50% of their dry biomass is lipids [2]. However, the use of microalgae to produce biomass remains limited, reducing cost-effectiveness.

Microalgae have low light utilization efficiency due to energy dissipation at high solar energy. Although the maximal theoretical efficiency of photosynthetically active radiation (PAR, 400-700 nm) solar energy conversion to biomass is approximately 27%, the actual efficiency of solar energy conversion to biomass in laboratory-scale growth tests is less than 6% [3]. One of the most important reasons is the uneven distribution of light in algal culture. Insufficient light into cells in the deeper layers results in respiratory loss, while photoinhibition occurs in surface cells exposed to strong light [4]. Excess light induces photoprotective mechanisms via

nonphotochemical quenching (NPQ), which relies on light-harvesting complex stress-related (LHCSR) proteins in microalgae. To provide a whole picture of LHCSRs in acclimation to environmental stress, we will review the recent progress on their biochemical characteristics, functions and underlying regulatory mechanisms in *Chlamydomonas*.

LHCSRs are widely distributed in most algae and mosses but absent in higher plants [5]. Here, we also analyze the distribution and evolutionary relationship of LHCSRs in green algae and moss, aiming to trace their conserved functions.

Based on these results, we present the future prospects of studies on acclimation to environmental stress fulfilled by LHCSRs, which may be applied in food crop resistance in the future.

2. Photoprotective Systems in Microalgae

Light is essential for photosynthesis. Previous studies indicate that a considerable amount of light-harvesting complex (LHC) proteins play important roles in light harvesting [6]. Three helices contained in LHC proteins are typically thylakoid transmembrane and bind lipid molecules, chlorophyll, and carotenoids [7]. Although photosynthesis depends on light, excess light could cause overexcitation of the photosynthetic apparatus and then produce highly active molecules or byproducts that could damage microalgae [8].

Fortunately, a variety of photoprotective mechanisms have been developed in microalgae to relieve photodamage, among which NPQ of chlorophyll (Chl) *a* fluorescence plays the most important role. In contrast to the inherent NPQ in higher plants, NPQ is manifested as a result of acclimation in *Chlamydomonas* under high light conditions [9]. The transcript of the *LI818 gene,* which encodes LHCSR proteins, is a mRNA induced by light [10]. The expression pattern of *LI818* is different from those of most of any other genes encoding LHCs [11], which suggests its functions in addition to light harvesting. Since then, a series of studies have demonstrated that LHCSRs mainly function in the photoprotection process in microalgae exposed to excess light [12]. In contrast to photosystem II subunit S (PsbS), which is employed to activate NPQ in higher plants, LHCSRs are adopted in the same process by microalgae [5, 13]. Although PsbS has also been identified in microalgae, it is not as important for the protection of the photosynthetic apparatus as in higher plants but important for the transient response to high light [14].

In *Chlamydomonas*, the family of LHCSRs is composed of LHCSR1 and LHCSR3, which are encoded by *LHCSR1* (Cre08.g365900) and *LHCSR3.1* (Cre08.g367500) \ *LHCSR3.2* (Cre08.g367400), respectively. The *Chlamydomonas npq4* mutant was screened out using chlorophyll fluorescence. LHCSR3.1 and LHCSR3.2 were initially identified to be responsible for the energy-dependent quenching (qE)-deficient phenotype [9]. With the mutation of a homologous gene, *LHCSR1,* in the *npq4* background, qE was completely inactivated in the *Chlamydomonas* triple mutant [5]. This evidence supports the possibility of the functions of *LHCSR1, LHCSR3.1* and *LHCSR3.2* in photoprotection.

Further studies indicate that the transcripts of *LHCSRs* accumulate in cells under photooxidative stress, including excess light [15], CO_2 [16], sulfur [17] or iron [18] deprivation, suggesting their roles in acclimation to environmental stress. Detailed studies have revealed that the polypeptide sequences of *LHCSR3.1* and *LHCSR3.2* are identical, but they have different promoter sequences and 3'UTR regions, demonstrating their different regulatory mechanisms to adapt to stress [19]. More interestingly, although the 3D structures of LHCSR1 and LHCSR3 are very similar (Figures 2A, B, C), LHCSR1 is reported to have a weaker quenching ability than LHCSR3, possibly because of their different binding sites with L2 carotenoids [20], suggesting their different biochemical activities.

3. Nonphotochemical Quenching and Its Components

Plants are exposed to different amounts of light in different environments. Algae live in water with a lack of ultraviolet, red and an abundance of blue wavelengths. Algae grown in aquatic environments are usually exposed to low light conditions, and fluctuating light may bring photoinhibition. [21].

Excess light triggers many quenching reactions of chlorophyll fluorescence, and more reactions could be uncovered with the development of capture and detection technology in the future. NPQ, as the major and fastest form of chlorophyll fluorescence-quenching reactions, mainly occurs within the antenna system of photosystem II. LHCSRs are the most important parts of microalgae (Figure 1A). NPQ is comprised of several components, and they are reported to have different degrees of importance depending on the microalgae species and physiological states. There are several steps in NPQ, including qE (fast phase), qZ and qT (middle phase), and qI (slow phase) [22]. Among them, qE is the most unique component of NPQ [23], can be activated reversibly within seconds along with light intensity and has the highest quenching ability.

In higher plants, qE is combined with a quenching component that is zeaxanthin dependent and triggered by low lumenal pH [24]. Zeaxanthin is the enhancer of qE, which can also be triggered by low lumenal pH. The initial synthesis of zeaxanthin originates from violaxanthin de-epoxidase activation, which is located in the lumen. The V1 site and L2 site of LHCII have the ability to bind newly synthesized zeaxanthin [25-29]. After exposure to low light, zeaxanthin is catalyzed back to violaxanthin by zeaxanthin epoxidase [30]. Then, qE is triggered by the specific amino acid protonation of acidified photosystem II (PSII) proteins that are exposed to the thylakoid lumen.

Photosynthetic organisms contain several xanthophyll cycles, including the lutein epoxide cycle, diadinoxanthin cycle and violaxanthin cycle. The lutein epoxide cycle only exists in some higher plant families. The diadinoxanthin cycle mainly exists in

species such as Bacillariophyceae, Xanthophyceae, Haptophyceae, and Dinophyceae. The violaxanthin cycle of brown algae and green algae is the same as that of higher plants [31]. However, the relationship between the violaxanthin cycle and NPQ in algae varies with species. For example, the NPQ of *Chlorella vulgaris*, *Chlorella saccharophila* and *Bracteacoccus minor* was dependent on zeaxanthin, while the violaxanthin de-epoxidation of *Pedinomonas minor*, *Tetracystis aeria* and *Chlamydomonas* was not closely associated with NPQ [32, 33].

The activation mechanisms of qE are not fully conserved among species, which makes species-specific responsiveness dependent on both the protonation of responsive PSII proteins and the involvement of the xanthophyll cycle [5, 13]. PsbS was reported to be responsible for qE activation in vascular plants two decades ago [13], and LHCSRs confer the same function in microalgae [5]. Interestingly, both LHCSRs and PsbS are active in qE in *Physcomitrella patens*, which diverged early from the green algae to the higher plant [34].

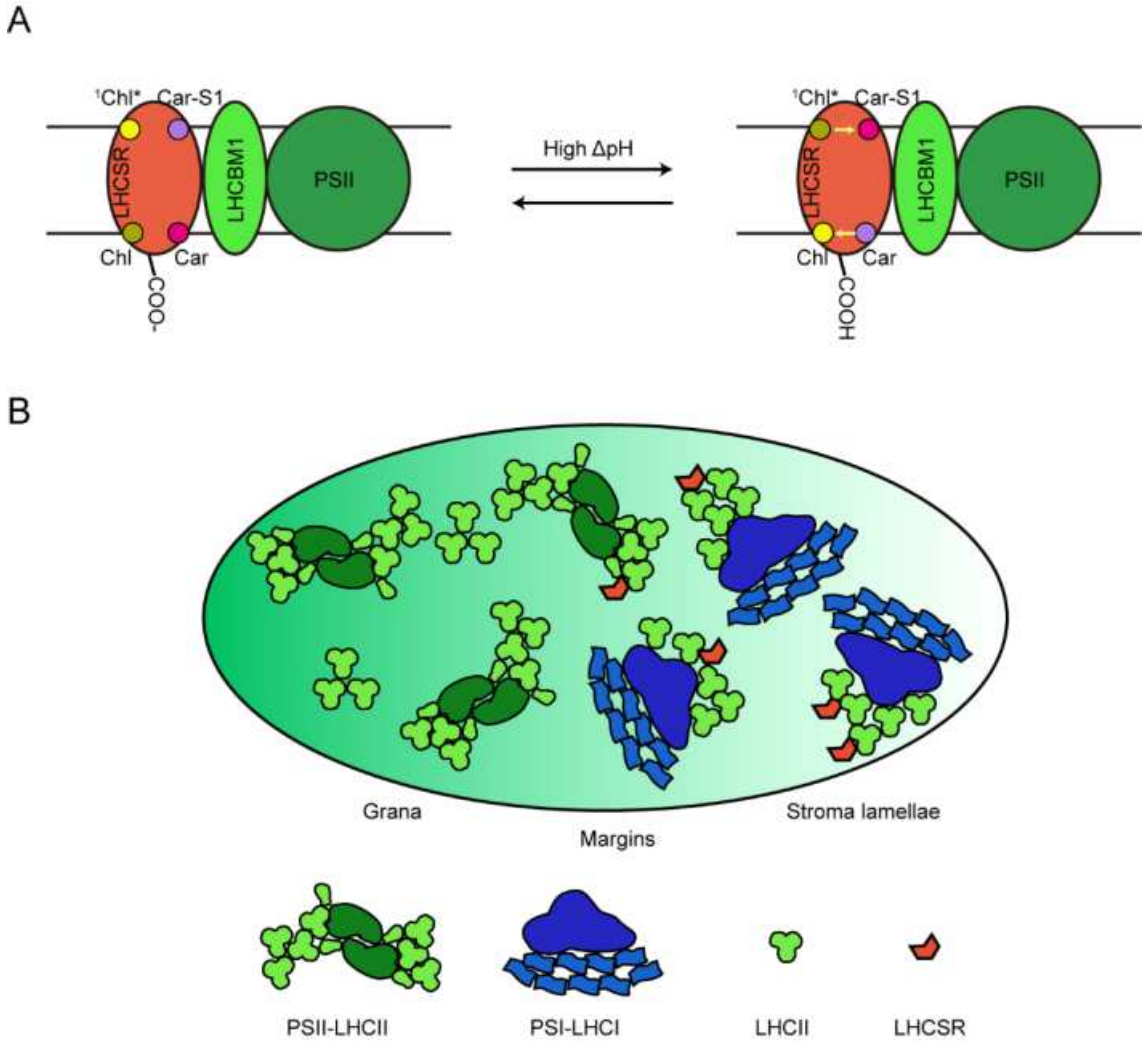

Figure 1. *Model for qE in microalgae. Schematic of LHCSR-dependent NPQ in Chlamydomonas reinhardtii. Energy transfer to the S1 state of carotenoids and electron transfer from carotenoids to chlorophyll are enhanced under high light conditions, and epoxidation reactions are reversed under limited light. LHCBM1 is proposed to be an LHCSR docking site. This picture is modified from Niyogi and Truong (2013) [35]. (B) Microalgae model of PSI and PSII fluorescence quenching dependent on LHCSR. No clear boundary among grana, margins, and stromal thylakoids is observed in algal chloroplasts. LHCSRs participate in the dissipation of excess energy in both PSII and PSI by quenching the associated LHCII antenna. This picture is modified from Pinnola (2019) [36].*

4. LHCSRs and Their Biochemical Characteristics in Microalgae

LHCSRs participate in the initial activation of qE in microalgae [5, 9]. However, it is difficult to isolate grana domains of unicellular algae, so the LHCSR location is still unclear [37]. It is speculated that LHCSRs independently catalyze the quenching of PSI-LHCI-LHCII complexes (in stroma-exposed domains) and PSII-LHCII (in grana membranes). This mechanism maintained the plastoquinone redox poise and controlled both PSI and PSII antenna system excitation in the short of far-red absorption forms of PSI (Figure 1B).

The binding of LHCSR3 to PSI or PSII is modulated by light conditions in *Chlamydomonas* [37, 38]. Energy transfer to the S1 state of carotenoids and electron transfer from carotenoids to chlorophyll are enhanced under low pH conditions (Figure 1A) [39]. LHCSR3 may act as an energy

quencher considering that it could bind pigments and has a state transition with a short fluorescence lifetime [9, 40]. The dissipative states are reversibly activated at low pH, which enables organisms to adapt to stepwise changes in solar irradiance. Moreover, LHCSR3 loss causes an increase in the Chl *a/Chl b* ratio, indicating that some reconfiguration occurs in the antenna [5].

The LHCSR1 conformation that controls the transition changes rapidly between dissipative states. [41]. The LHCSR1 lifespan of the quenched conformations is 80 ps, and the unquenched conformation is 3.7 ns, indicating that unquenched LHCSR1 only acts as a regular light-catching antenna but competes with excitation trapping in the quenching state [42].

Thus, this evidence supports that multiple biochemical characteristics of LHCSRs make them respond to possible environmental stress in microalgae.

5. LHCSRs Play Roles in Acclimation to Environmental Stresses

LHCSRs were first identified in mutant screening in which strains were exposed to high light [5], indicating that LHCSRs could be induced by such stress. Joliot and Finazzi further demonstrated the possible mechanism: the Calvin cycle is saturated, resulting in the lack of both ADP and PI under high light conditions. Thus, the activity of ATPase is progressively inhibited, leading to the accumulation of H^+ and a decreased pH value in the stroma, and low pH further induces qE [43].

Intensive studies provide clues on the characteristics of LHCSRs in response to excess light. Similar to known LHC proteins, LHCSRs are embedded into the thylakoid membrane [20]. Unlike PsbS, LHCSRs form complexes with Chl *a*, Chl *b*, lutein and Vio/Zea [9].

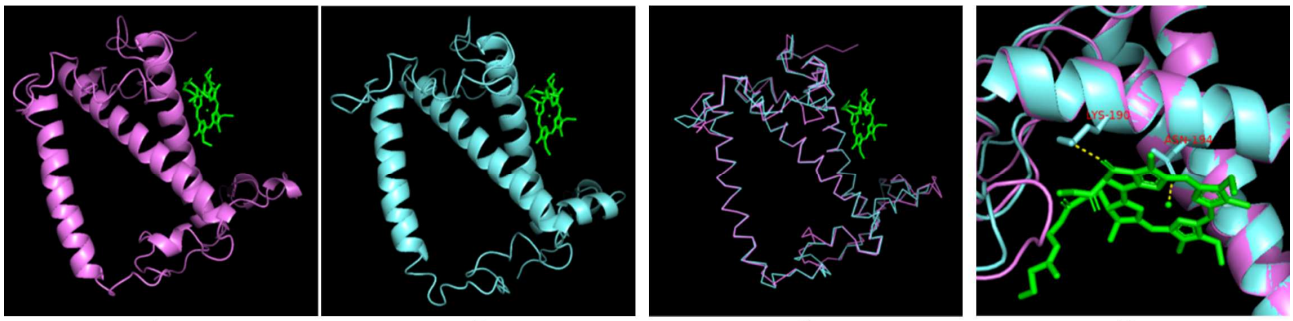

Figure 2. *Model structure of LHCSR1 and LHCSR3. Structural model of LHCSR3 obtained by sequence alignment with Chl a/b binding protein (PDB 6zzx.1.T). (B) Structural model of LHCSR1 obtained by sequence alignment with Chl a/b binding protein (PDB 6zzy.1.S). (C) Structural alignment of LHCSR3 (violet) and LHCSR1 (cyan). (D) Enlargement of the Chl a binding site region with the LYS-190 and ASN-194 residues predicted in LHCSRs to be in close contact with the Chl a molecule. Molecular graphics was performed with PyMOL software.*

3D model prediction of LHCSR proteins confirms that they can bind to Chl *a*, and the binding sites are LYS-190 and ASN-194. Unlike most LHC proteins, the site selectivity of LHCSRs is less strict, and other pigment-specific binding sites of LHCSRs could not be confirmed (Figure 2D).

LHCSRs respond to low pH depending on the exposure of their glutamate/aspartate residues to the cavity [9, 44]. Biochemical and spectral analysis showed that as pH-sensitive proteins, ^{1} Chl*-binding LHCSRs could be catalytically decomposed and quenched under acidic conditions, and the excitation energy could be quenched by the charge transfer mechanism of photosynthetic pigments [20].

Unlike most of the LHC proteins, the lifetime of chlorophyll fluorescence of LHCSRs is unusually not very long and even shorter under low pH conditions, indicating that the protein adopts active energy-dissipation mechanisms that make it suitable for the transfer between proton sensing and quenching reactions. The active energy dissipation mechanism of LHCSR allows quenching to be active even under limited illumination conditions, and the quenching activity may be enhanced by acidic residue protonation of the protein. Therefore, the expression of LHCSR is usually kept at a low level under limited light conditions to avoid unnecessary quenching reactions [9, 45, 46]. Based on the quenching characteristics of the LHCSR3 subunit mentioned above, scientists have proposed a variety of underlying mechanisms, including energy transference of carotenoids, formation of carotenoid radical cations, carotenoid-chlorophyll charge transfer, and the induction of chlorophyll-chlorophyll charge transfer [39].

In addition to the quenching effect of the PSII antenna, LHCSRs also catalyze the quenching of PSI by affecting PSI-related LHCII complexes in mosses and algae [46, 47]. In obtaining excitation energy from a light trapping system [9, 44, 48], LHCSRs interact with other antenna system components, such as CP26 [49] or LHCBM1 [40, 50].

In addition to excess light, LHCSR3 was also observed to accumulate when the cells were exposed to carbon dioxide deprivation [19, 51] or high salinity or nitrogen starvation conditions [14]. Under the stress conditions mentioned above, reactive oxygen species are generated to an abundant level, which may activate the expression of LHCSRs (Figure 3). Without LHCSRs, the release of singlet oxygen is increased, which results in damage to PSII [52]. Therefore, the present evidence supports that there is a close relationship between LHCSRs and reactive oxygen species, and the fine-tuning mechanism still remains to be explored.

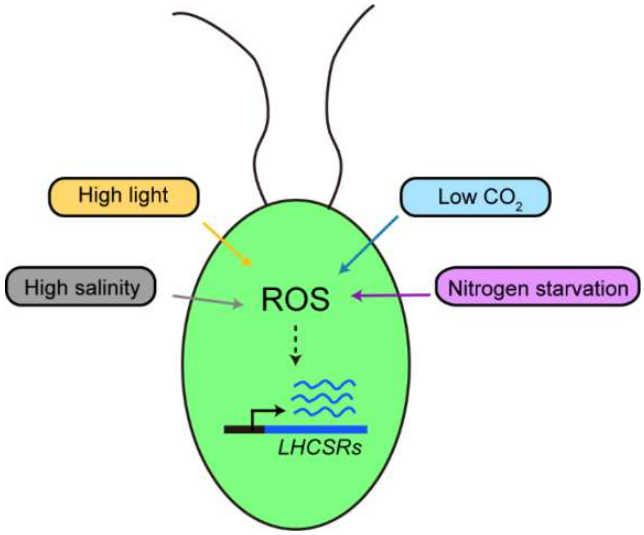

Figure 3. *Schematic diagram showing the expression of LHCSRs activated by stress conditions. Under stress conditions, reactive oxygen species are generated at an abundant level, which may activate the expression of LHCSRs.*

6. The Underlying Regulation Mechanism of LHCSRs

LHCSRs accumulate at various levels under high light stress, indicating that they are differentially regulated by the underlying mechanism. In particular, the transcripts of LHCSR3.1 and LHCSR3.2 both accumulate at much higher levels than that of LHCSR1, suggesting that LHCSR3 may play a more important role and be greatly upregulated under high light [53]. However, LHCSR1 can partially compensate for the phenotype of the *Chlamydomonas npq4* mutant [5], demonstrating its role in NPQ, although it is not as important as LHCSR3. This evidence reveals that the expression of *LHCSRs* could be precisely regulated by the fine-tuning system.

Light signals and light utilization can be interlinked by LHCSRs and photoreceptors, which is supported by the evidence that both *LHCSR*s and qE are induced by ultraviolet light [54] and blue light [55], uncovering the relationship between light signals and energy utilization. Photoreceptors, such as UVR8 [54, 56] and phototropin (PHOT) [55], are required to induce the expression of *LHCSRs* and the occurrence of qE. Interestingly, the expression of *LHCSR3* is modulated by the photoreceptor PHOT and photosynthesis [19, 55, 57], while the expression of *LHCSR1* is controlled by UVR8 and is independent of photosynthesis [19]. Since ultraviolet light can damage both the PSI and PSII systems, LHCSR1, as the UVR8 responser, may participate in the quenching of both photosystems *in vivo* [46, 58, 59].

Once the light signal is captured by the photoreceptors, the downstream regulation of the *LHCSRs* is activated via the conserved E3 ubiquitin ligase complex through CrCO, which is homologous to the plant transcription factor (CONSTANS, CO) [60]. CrCO can be targeted by both the COP1-SPA1 and DET1-DDB1 pathways (Figure 4).

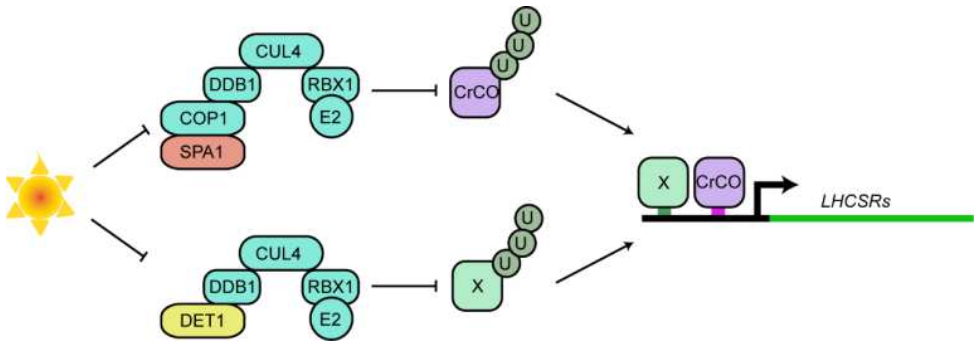

Figure 4. *Schematic model of the expression of LHCSRs regulated by the ubiquitin ligase complex in Chlamydomonas. Under high light conditions, the activity of both the COP1-SPA1 E3 ubiquitin ligase and DDB1-DET1 E3 ubiquitin ligase is inhibited. This enables the accumulation of trans-acting factors and their binding to cis-acting elements to activate the transcription of LHCSR, but the specific factor X remains to be identified. CUL4: a cullin-based ring-type ubiquitin E3 ligase complex type 4; RBX1: a small ring finger protein.*

6.1. COP1-SPA1 Pathway

The expression of LHCSRs can be regulated in acclimation to various light conditions through the COP1-SPA1 pathway. Under low light conditions, CrCO can be ubiquitinated and degraded by the COP1-SPA1 E3 ubiquitin ligase complex in *Chlamydomonas*, which results in reduced expression of *LHCSRs* (*LHCSR1*, *LHCSR3.1* and *LHCSR3.2*). When algae are transferred to high light conditions, the COP1-SPA1 E3 ubiquitin ligase is inhibited by photoreceptor-mediated signaling through an unclear mechanism, which allows CrCO accumulation and binding with a CO-like binding motif, resulting in the activation of *LHCSRs* [61].

6.2. DET1-DDB1 Pathway

DDB1 (Cre10. g432000) and DET1 (Cre13. g571560) are two subunits of the CUL4-DDB1 DET1 E3 ligase complex, and their defects lead to the accumulated transcription of *LHCSR1* and *LHCSR3* in the dark, which is similar to the lack of CRL4 $^{COP1\ SPA1}$ [62]. The results from vascular plants and mammals indicate the possible mechanism of the CUL4-DDB1 DET1 system [63]. DDB1 and DET1 are thought to be part of the cullin-based ring E3 ubiquitin ligase complex 4 (CRL4). As a central scaffold subunit, the carboxyl termini of the Cullin protein bind to RBX1 (a small RING finger protein), and aminoa termini bind to the substrate

receptor module (e.g., DET1). The substrate receptor motif and the central scaffold subunit were connected by DDB1. The CRL4-DET1 complex ($CRL4^{DET1}$) is an E3 ligase in *Arabidopsis* that negatively regulates the critical developmental stages in different organisms [64] or works in conjunction with another E3 ligase in regulating plant photomorphogenesis [65].

Both of the above E3 ligase complexes may target CrCO, or they may have different substrates to coregulate the expression of qE-related genes [62]. Thus, the expression of LHCSRs is precisely regulated starting from the initial photoreception of the cells and throughout transcription, even though translation or posttranslation still remains to be identified.

7. Distribution and Evolution of LHCSRs in Algae

LHC proteins with three transmembrane helices are assumed to evolve from two-helix progenitors [66]. These LHC proteins may appear after the differentiation of green and red algae because there are no such proteins in glaucophytes [67]. LHCSRs have three transmembrane helices that are typical characteristics of the LHC superfamily, and they may follow the same evolutionary history.

LHCSRs are widely distributed in mosses, green algae, haptophytes, photosynthetic apicomplexan (*Chromera velia*) and photosynthetic stramenopiles [68] but absent in dinoflagellates [69], red algae, cryptophytes [70] and vascular plants [5]. Some reasons are suggested to explain the distribution pattern of LHCSRs mentioned above. Analysis of ancient origin suggests that LHCSRs are lost in red algae [71], but genes may transfer horizontally between green algae and secondary red algae, which is related to red plastid acquisition [72]. However, phylogenetic analysis indicates an opposite direction of gene transference, from red plastids containing lineages to green algae and plants [70]. This result is also supported by other studies [73, 74]. Although the timing seems questionable at first glance, Dittami et al. suggest that the window of opportunity occurred 200 million years ago [70] after secondary red algae and before green lineage diversification [36]. Therefore, the evolution of *LHCSRs* remains to be explored to explain their distribution in algae species.

LHCSR proteins have high efficiency in catalytic quenching reactions [5, 9]. However, in the course of evolution, they were replaced by PsbS. There are many differences between PsbS and LHCSR, such as quenching response mechanisms, thylakoid membrane localization, and interactions with other proteins. LHCSR has both quenching and pH sensing functions, while a single PsbS sensor can promote the quenching of multiple different LHC proteins [35]. PsbS is located in grana in mosses and vascular plants, [47, 75], concordant with its PSII quenching. However, LHCSR is limited to the stromal membrane [47], and the PSII supercomplex is quenched directly by LHCSR only if it is exposed at the stromal margins. In algae and mosses, the overall proportion of PSII protected by LHCSR quenching is approximately 30%, and most of the PSII is unprotected [76]. Therefore, the localization of LHCSR in PSII-rich stromal membranes is not particularly suitable for protecting PSII under excessive light conditions.

In the stroma, PSI is quenched by LHCSR through LHCII [46, 47]. Although PSII reaction centers are limited to grana in mosses, LHCII is evenly distributed between stromal membranes and grana [47], and LHCII in stromal membranes helps regulate the size of the PSI antenna to offset the overreduction and charge recombination of PSII [77]. In green algae, LHCSR also transfers excitation energy to PSI through multiple LHCII complexes [78]. In summary, the main target of algal NPQ is PSI-associated LHCII of the stroma, which is colocalized with LHCSR.

Early land plants were directly exposed to sunlight [21], while with the emergence of vascular plants, the sunlight exposure of different plants depended on the degree of shading. Ultraviolet and blue light under shadows are consumed, and far-red and green light are enriched [79]. As the intensity and angle of sunlight change, plants may be exposed to suddenly increased light and produce photoinhibition effects [80]. The absorption efficiency of redshifted LHCI is much higher than that of LHCII, which reduces the overexcitation of PSI in high light, resulting in redundant LHCII and LHCSR in stromal membranes. PsbS can replace algae LHCSR and induce NPQ in vascular plants because both PsbS and PSII are located in the grana, where PsbS can enhance the photoprotective energy dissipation and repair rate of PSII [81]. In conclusion, during plant evolution, LHCSR was gradually replaced by PsbS, which is more suitable for terrestrial environments.

8. Future Research Prospects

Plant energy dissipation mechanisms limit growth but enhance photoprotection to avoid photoinhibition. Photosynthetic organisms have evolved the ability to dissipate excess solar energy as heat under high light conditions. A study has shown that under limited light, the biomass accumulation rates of *Arabidopsis thaliana* with insufficient or excessive energy dissipation increased or decreased, respectively, indicating an inverse relationship between energy dissipation and growth [82]. Additionally, increasing the NPQ relaxation rate of tobacco reduces its overall heat dissipation and significantly increases biomass yield under field conditions [83]. However, no consistent results have been obtained in studies of the energy dissipation of algae. Perozeni *et al* found that *Chlamydomonas npq4* mutants grew faster than the wild type in photobioreactors [84], while other experiments showed the opposite results [53, 85].

In single-celled algae, energy dissipation is catalyzed by pigment-bound LHCSR proteins [9]. qE is induced by pigment-free PsbS proteins in higher plants [86-88]. LHCSR

and PsbS are located in different domains in the thylakoid membrane. PsbS and PSII are located together in the granum membrane [89-91], while LHCSR is located in the stromal membrane and can act on PSI and PSII [46, 47].

LHCSRs are the major components of NPQ in *Chlamydomonas* [5], and studies have been conducted to infer their physical and chemical properties [37, 42], stress response [20, 52], and gene regulatory mechanism [61, 62]. However, there are still many questions to be answered in understanding the action mechanism of LHCSRs. The expression of *LHCSRs* has been shown to be regulated from the initial photoreception to the transcription level, but the downstream regulatory network after transcription remains to be clarified. In particular, there is no information about the interacting proteins of LHCSRs at the translation level and transportation to the thylakoid membrane. Whether LHCSR could be transported into the thylakoid membrane in the liquid phase remains to be identified. How LHCSRs are degraded when cells are transferred from high light to low light needs to be explored.

With the development of synthetic biology, advanced technology, including artificial design, could be used to build an LHCSR scaffold with high catalytic activity efficiency. Meanwhile, synthetic promoters are also helpful for the precise regulation of LHCSR expression in response to environmental stress. Thus, specific strains could be developed with the LHCSR module being assembled to fulfil the requirements of practical application.

9. Conclusion

Excessive light is harmful to plants. This paper mainly introduces the involvement of *Chlamydomonas* LHCSRs in NPQ to consume excessive light energy to protect cells. Meanwhile, LHCSRs are also involved in other types of environmental stress. Therefore, this paper further reviewed the potential interaction factors and upstream regulatory factors of LHCSRs in *Chlamydomonas* and explored the potential mechanisms of LHCSRs in adaptation to various environmental stresses. By discussing the evolution of LHCSRs in green algae and mosses, we found that LHCSRs play an important role in plant adaptation to environmental changes on Earth. Future studies on LHCSRs could focus on the corresponding and regulatory mechanism of LHCSRs to other kinds of stress and apply it to synthetic biology to improve the resilience of food crops.

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