Rapid Communication in Photoscience(2013), vol. 2, no. 1, pp.18-23 ISSN 2234-8182
http://dx.doi.org/10.5857/RCP.2013.2.1.018

Review article

Mobilization of Photosystem II-Light Harvesting Complex II Supercomplexes during High Light Illumination and State Transitions

Krishna Nath[1], John Elizabeth[1], Roshan Sharma Poudyal[1], Su Yeon Ko, Woon Ki Lim, and Choon-Hwan Lee*

Department of Molecular Biology, Pusan National University, Busan 609-735, Korea

ABSTRACT: The photosystem II (PSII) light harvesting complex (LHC) consists of a variety of pigment protein complexes which are involved in structural organization and regulation of photosynthetic unit. These LHC proteins encoded by a group of Lhcb genes are essential for the structural integrity of PSII supercomplex, the channeling the excitation energy to the reaction center of PSII and its redistribution to photosystem I by state transitions. Numerous studies with the help of recent technological advancements have enabled a significant progress in our understanding on the structure of PSII-LHCII supercomplexes and their mobilization under various light conditions. Here, we present a mini-review on the latest concepts and models depicting the structure of PSII-LHCII supercomplexes and the role of Lhcb proteins in their supra-molecular organization. Also we will review on the current understandings and remaining problems involved in the mobilization of the supercomplexes during state transitions and during high light illumination for controlling light energy distribution between the two photosystems.

Light harvesting during photosynthesis in green plants is carried out in photosystem II (PSII) and photosystem I (PSI) located in the thylakoid membrane inside leaf chloroplasts. In both photosystems, the excitation energy is harvested by light harvesting complexes, LHCII and LHCI and migrates to reaction center, P680 and P700 for PSII and PSI, respectively. The LHCII is associated with PSII as a large supra-molecular structure known as the PSII-LHCII supercomplex[1], and LHCI is associated with PSI similarly as PSI-LHCI supercomplex[2].

Since the absorbance spectra of PSI and PSII differ, the optimum light harvesting is determined by balancing the absorbed light between PSI and PSII depending on the available light intensity. This short term physiological adaptation is achieved by shuttling of the mobile pool of LHCII trimer between PSI and PSII. Some LHCII bound to PSII in State 1 can be mobilized to a place nearby PSI during state transitions to State 2 as a result of preferential excitation of PSII. The state transition is controlled by reversible phosphorylation of Lhcb1 and Lhcb2 by the thylakoid kinase Stn7/Stt7[1,3,4,5].

In view of the fact that LHCII trimers along with other minor Lhcb antennae proteins are involved in optimizing the light harvesting capacity of plants, this review will focus on the recent progress and understanding on the structural and functional role of these Lhcb proteins in the PSII-LHCII supercomplex and also their roles during state transitions and high light illumination.

[1]**These authors contributed equally to this work**
***To whom correspondence should be addressed.**
E-mail: chlee@pusan.ac.kr

Structure of PSII-LHCII supercomplex

The structure of a typical photosynthetic supercomplex PSII-LHCII is depicted as Figure 1 and includes a dimeric core protein (C_2) complex surrounded by peripheral major and minor antenna complexes[1]. Each PSII core is composed of proteins D1and D2, which harbor the reaction center P680 and the cofactors of the electron transport chain, and proteins such as CP43 and CP47, which act as the inner antennas for the light harvesting process. In the peripheral antenna LHCIIs consisting of the major antenna complexes exist as trimeric protein complexes that are composed of products of the *Lhcb1, Lhcb2* and *Lhcb3* genes, and the usual occurrence of these gene products are 8:3:1[6]. LHCIIs consisting of the minor antenna complexes include gene products of *Lhcb4* (CP29), *Lhcb5* (CP26) and *Lhcb6* (CP24).

Depending on the composition of Lhcb proteins, LHCII trimers vary in their functional association with PSII in the PSII-LHCII supercomplex. They can be distinguished into three forms namely strong (S), moderate (M) and loose (L) depending on the strength of binding to PSII complex. The LHCII trimer S is mostly composed of Lhcb1 and 2, whereas M has Lhcb1 and high content of Lhcb3[1]. The composition of Lhcb isoforms in trimer L is probably different from other trimers and not well-known, because trimer L is difficult to purify[3].

The level of organization of PSII-LHCII supercomplex is varied and the characterization of each of these supercomplexes is highly depended on the ability to natively isolate these conformations. Although it is emphasized that the most natively existing supercomplex is composed of $C_2S_2M_2L_2$[7], the difficulty in isolation and characterization of such a structure limits our understanding of this supercomplex. The next to native structure which can be easily isolated is the $C_2S_2M_2$ supercomplex composed of a dimeric PSII core each of which is associated with two LHCII trimers, S and M. Each LHCII-S trimer interacts with both core complexes through CP26 and CP29, whereas each LHCII-M interacts with only one core complex via CP24 and CP29 as summarized in Figure 1. The interaction of a LHCII trimer with a minor complex is predicted based on the studies using antisense or knock-down mutants for each minor complex. CP29 is essential for anchoring both LHCII proteins to the core complex and indicates a structurally significant role in the organization of the dimeric supercomplex. This was evidenced in Arabidopsis expressing antisense CP29 which failed to form any PSII-LHCII supercomplex[8,9]. Moreover, CP29 antisense mutation also causes decrease in the amount of CP24[10,11]. However, LHCII-S may still interact with a single core complex via CP26 and was successfully isolated recently. The CP26 knockout failed to form such a complex suggesting that CP26 is the primary anchoring protein of LHCII-S to the core complex and that CP29 just provides an additional docking point. Similarly, CP24 knockouts may form C_2S_2 supercomplex but fail to form $C_2S_2M_2$ supercomplex suggesting that it is the main protein involved in docking LHCII-M trimer. However Lhcb3 knockout failed to incorporate CP24 into the supercomplex suggesting a more mutualistic interaction of CP24 and LHCII-M[1].

Role of LHC proteins in Energy transfer

The energy transfer from the light harvesting antennae to core in the PSII-LHCII supercomplex is carried out by the proteins rich in low energy chlorophyll (Chl) a molecules. This indicates that the monomeric Lhcb proteins which are vital for the structural integrity of the supercomplex may not directly affect the energy transfer as long as the low energy Chl a molecules are in contact. For example, LHCII trimer S requires CP26 for anchoring to the core, but the Chl a molecule of trimer S transfers the excitation energy directly to the Chl479 of CP43 located in the core (Figure 2). CP26 also transfers energy to the PS core through the Chl611/612 which transfers the energy to Chl486 of CP43. However in the case of LHCII trimer M, CP29 mediates the energy transfer to the RC. CP24 also cannot transfer energy directly to the PS core as no Chl a molecules of CP24 is facing the Chl a molecule of CP47 in the core. Instead CP24 transfers energy to CP29. The Chl603 of CP29 transfers the excitation energy to Chl511 of CP47. CP47 and CP43 therefore act as inner antennae of the core complex and mediate the energy transfer from the peripheral antennae to the reaction center.

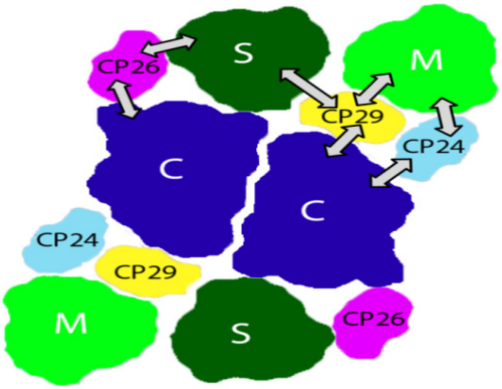

Figure 1. The structural organization of a cynobacterial $C_2S_2M_2$ supercomplex. LHCII trimers S and M are shown in dark green and light green, respectively. The core complex is represented in dark blue. The monomeric Lhcb proteins CP24, CP26 and CP29 are represented in light blue, magenta and yellow, respectively. The double headed arrows indicate the interaction between the protein complexes. This model is drawn based on Caffarri et al.[1] and Kouril et al.[12].

These energy transfer pathways are depicted in Figure 2 based on the atomic model of PSII-LHCII supercomplex of cyanobacteria[1,12]. In higher plants, the organization of the PSII-LHCII supercomplex is pretty much similar to cyanobacteria[13] and hence the energy transfer mechanism in higher plants may be expected to be similar to that of cyanobacteria. However, the

genetic variation in the proteins of the supercomplex must be taken into account, considering the fact that these plants are highly adapted to a particular environment and lighting schedule[14].

In the dimeric $C_2S_2M_2$ supercomplex of higher plants the excitation energy can be transferred to the nearest available energy quencher when the reaction center of the default path is 'closed' due to saturation of electrons. The dimeric state of the supercomplex thus ensures that there is ample amount of low energy Chl molecules surrounding the excited antennae enabling the electrons to by-pass the usual path and reach the next available electron acceptor. However, there are two connections which cannot be compromised. These are the path from CP43 to reaction center and CP29 to CP47. Removing these connections lead to a significant increase in the average excited state lifetime[15].

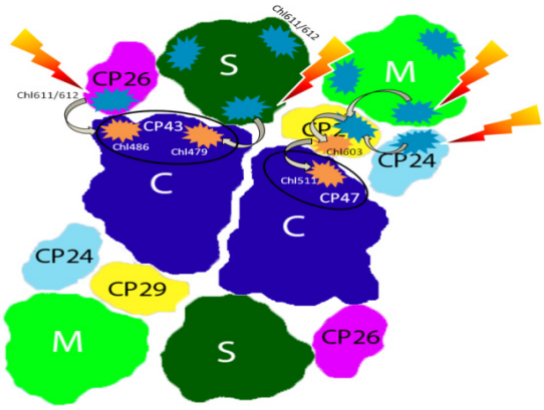

Figure 2. Excitation energy transfer pathway in a $C_2S_2M_2$ supercomplex. Chl a of LHCII trimers are represented in blue and those within the core is represented in orange. Chl603 of CP29 is also represented in orange. The circled area of the core represents the location of CP43 and CP47. The arrows indicate the path of energy transfer through the low energy Chl a molecules within the peripheral antennae and the core upon excitation. This model is drawn based on Caffarri et al.[1] and Kouril et al.[12].

Role of LHC proteins in state transition

Over the years, there have been arguments regarding which Lhcb proteins are involved in state transitions and a couple of theories were put forward[16,17,18]. One theory suggests the role of LHCII trimer in state transitions and the other holds CP29 responsible. However these concepts were put forward using two different model organisms: *C. reinhardtii* and *A. thaliana* suggesting the existence of two completely different modes of state transitions.

LHCII-L is responsible for state transitions: Of the three LHCII trimers, S, M and L, the trimer which is involved in state transitions has been a mystery until recently. Initially it was thought that LHCII-M might be involved in state transitions as it is the trimer which could be easily dislocated from the supercomplex[19]. However, the failure to detect Lhcb3 in the stroma lamella discarded this hypothesis as it is the main protein in LHCII-M. Also trimer S is very strongly bound to PSII core and therefore its involvement in state transitions is quite unlikely. The next suitable candidate for state transitions is LHCII-L. However, the inability to isolate a stable LHCII-PSI supercomplex hampered the confirmation of this theory until Galka et al.[3] who reported the isolation and characterization of PSI-LHCII supercomplex from *Arabidopsis thaliana* and *Zea mays*. They showed that the Lhcb protein composition of PSI-LHCII is different from that of LHCII-S, LHCII-M and CP29, although they couldn't directly prove that it is composed of LHCII-L as stable isolation of LHCII-L has not been successfully accomplished.

CP29 induced total molecular remodeling: There is also another school of opinion which suggests that state transitions involve total molecular remodeling of PSII-LHCII supercomplex leaving behind only the PSII core in State 2 as there are evidences that all the Lhcb proteins are phosphorylated and detached from the PSII core[20]. Further proof from *C. reinhardtii* suggests that CP29 has strong affinity to PSI during State 2 resulting in the dissociation of the PSII-LHCII supercomplex[2,21]. Although total molecular remodeling is reported in Arabidopsis, there is little evidence of CP29 binding to PSI in higher plants and only the LHCII trimers are known for this role.

Role of Lhcb3, CP43, Psb27 and PsbW during state transitions: Although Lhcb3 is found only in LHCII-M, the failure to detect Lhcb3 in the stroma lamella makes the possible involvement of the trimer M during state transitions questionable. However, based on the studies on the Lhcb3 knock-out mutants, Damkjær et al.[16] suggest that the main function of Lhcb3 is to modulate the rate of state transitions.

State transition to state 2 is initiated by phosphorylation of the PSII core subunit, CP43[22], but CP43 phosphorylation is independent of state transitions, because wild type-like supercomplex mobilization is observed in an Arabidopsis STN7 knockout mutant[23].

The role of the luminal protein Psb27 is suggested to generate and/or stabilize molecular bridges between

neighboring PSII-LHCII supercomplexes[23]. This link will allow higher order supercomplex arrays with regular pattern and may hinder the start of state transitions, which can explain the acceleration of sate transitions in Arabidopsis Psb27 knockout mutants[23].

Like Psb27, the loss of PsbW destabilizes the supra-molecular organization of PSII and accelerates state transitions[24]. PsbW is a 6.1 kD protein with one trans membrane helix, and it is probably the docking site of Psb27, which is required for the generation of the supercomplexes.

Mobilization of PSII-LHCII supercomplex under high light illumination

PSII-LHCII supercomplexes are the functional form of PSII which are organized in the appressed region of grana membranes. When plants are illuminated with strong light, the mobilization of PSII-LHCII supercomplexes from grana to stroma lamella is observed probably as a photoprotective mechanism. Although the exact mechanisms of this high light induce mobilization of PSII-LHCII supercomplexes is still unclear, several recent reports have suggested that the mobilization of PSII-LHCII supercomplexes is controlled by the phosphorylation of PSII core proteins, especially the phosphorylation of PSII core antenna protein CP43 by the activity of STN8 kinase and the phosphorylation of LHCII proteins by STN7 kinase[23,25].

We can divide this process into two steps: an initial step until CP43 phosphorylation and the next step for actual dissociation and movement of the supercomplex to granal margins or to stroma lamella. Then the damaged PSII core by high light illumination will be dephosphorylated and degraded for PSII repair[25].

Recently the mobilization of CP29 from PSII-LHCII supercomplexes to PSII monomers and dimers is reported after its phosphorylation during high light illumination, and CP29 phosphorylation may induce the disassembly of the PSII supercomplexes in *A. thaliana*[26]. The phosphorylation of CP29 is also proposed to be essential for state transitions in *C. reinhardtii*[18]. Therefore, it has been postulated that the phosphorylation of PSII proteins accelerate its mobilization during light illumination. Apart from this the protonation of the PsbS protein also facilitates the migration of the PSII-LHCII proteins along the thylakoid membrane for non-photochemical quenching during light illumination[27].

Problem to be clarified in the regulation of PSII supercomplex mobilization

State transitions can be monitored by the measurement of qT during fluorescence induction process as shown in Figure 3A. During state transitions 77K fluorescence spectra of photosystems are also monitored, and the change in the ratio of F733/F685 is often used as the signal. When plants were illuminated with PSII light Lhcb1 and Lhcb2 are phosphorylated, and they are dephosphorylated under PSI light, therefore state transitions can be monitored by immunoblotting as reported using *A. thaliana* by Tikkanen et al.[28]. In this experiment, the occurrence of state transitions were proved by showing changes in 77K fluorescence spectra that are characteristic to state transitions similar to that shown in Figure 3B.

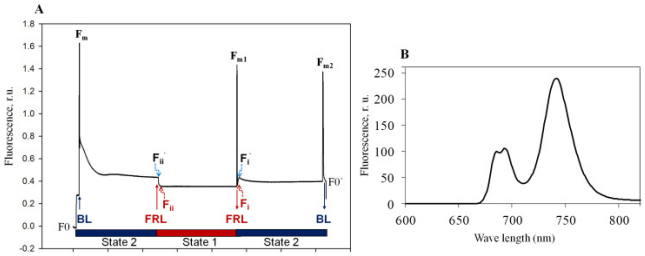

Figure 3. Measurements of state transitions and 77 K fluorescence emission spectra of mature rice leaf segments. **A**, Chl fluorescence transients used to measure state transitions between State 1 and State 2. Upward arrows and downward arrows represent turn on and turn off the PSII favored blue light (BL) and PSI favored far-red light (FRL), respectively. Elbow arrows are indicating the points where values of Fi, Fii, Fi` and Fii` were taken. The maximum fluorescence (Fm) yield was determined by exposing leaf segment with an 800 ms flash of saturating white light pulse (SL). The FRL was turned on to create State 1, and after 15 min the maximum fluorescence (Fm1) yield in this state was determined by giving SL for 2 sec. The FR light was then switched off to create State 2, and after 15 min the maximum fluorescence yield (Fm2) in this state was determined by giving SL for 2 sec. The relative changes in state transitions can be estimated either by the changes in maximal fluorescence (Fm1 − Fm2)/Fm1[29] or by ST2 = [(Fi` - Fi) - (Fii`-Fii)]/(Fi` - Fi), where Fi and Fii designate fluorescence in the presence of PSI light in State 1 and State 2, respectively, while Fi` and Fii` designate fluorescence in the absence of PSI light in State 1 and State 2, respectively[30]. **B,** *In vivo* 77 K fluorescence emission spectra of mature rice leaf segment. The fluorescence emission of detached leaf segment was excited at 440 nm wavelength and recorded between 620 and 820 nm wavelengths with a fluorescence spectrophotometer (F4500, Hitachi, Japan).

Most of these signals used for state transitions are observed during high light illumination for PSII supercomplex mobilization. When studying PSII repair cycle this mobilization process is also known to be involved[25]. Similarly non-photochemical quenching is known to be induced under high light illumination, which involves migration of PSII supercomplex[27]. Therefore, a question arose is whether the first step of high light induced mobilization is the same as the first step of PSII light induced state transition, or not. The mobilization of PSII-LHCII supercomplex is observed before the start of PSII repair during high light illumination, which involve the phosphorylation of PSII core proteins including CP43, and CP43 phosphorylation is required for the initiation of state transitions as mentioned earlier. In the second step of the state transition to State 2 the actual mobilization of PSII supercomplex is initiated by the phosphorylation of LHCII by STN7, which decouples LHCII from the supercomplex to be migrated to PSI. However, LHCII is known to be phosphorylated and dephosphorylated very rapidly in the beginning of high light illumination. Interestingly, phosphorylation and dephosphorylation kinetics of CP43 is different from other PSII core proteins regulated by STN8: unlike the phosphorylation of D1 and D2, CP43 phosphorylation was up-regulated in the night compared with the level under the light condition in mid-day[31]. Thus, it should not be ignored that the dissociation of this core antenna during high light illumination to balance the excitation energy between PSI and PSII during state transitions. As we have mentioned a stable PSI-LHCII supercomplex is purified from *A. thaliana* and *Zea mays* plants after transition to State 2[3]. LHCIIs loosely bound to PSII are involved in state transitions and become strongly bound to PSI in State 2 to form the PSI-LHCII complex. In the agreement of Galka et al.[3], LHCII serves as an antenna of both PSI and PSII in most natural light conditions to achieve excitation balance between the two photosystems[32]. Therefore acclimation to different light intensities is possible simply by regulating the expression of Lhcb genes only. Through the time-resolved fluorescence measurements on the photosynthetic thylakoid membranes they could show that LHCII even became more efficient light harvester when it is associated with PSI than with PSII[32].

In *C. reinhardtii* PSI-LHCI supercomplex strongly associated with CP29 is isolated and this complex help binding with LHCII in State 2[2, 18,21]. Phosphorylation of Lhcb proteins is important for the regulation of state transitions[4,5], and CP29 is known to be strongly phosphorylated during high light illumination and under stress conditions including chilling in the light[33,34]. Now the question is why mobilization of CP29 during state transitions is observed only in *C. reinhardtii*. Therefore it would be interesting to know the involvement of CP29 for mobilization of trimer L during state transitions. Previously, the involvement of trimer M in state transitions was in question. Even further a fundamental question is raised by Kouril et al.[12] that the existence of State 2 is aimed at decreasing the antennae surface of PSII rather than increasing the light harvesting by PSI.

Another question is why there is few report on CP29 phosphorylation in dicots. Tikkanen et al. [25] and Chen et al.[33] reported CP29 phosphorylation in Arabidopsis when illuminated with PSII light (not by high light). Fristedt and Vener[26] reported CP29 phosphorylation in Arabidopsis after high light illumination by mass spectrometry, not by Western blotting. Therefore, the role of CP29 phosphorylation is also a topic to be studied, and it would be interesting to understand the role of CP29 phosphorylation in supercomplex mobilization during state transitions in higher plants and to know whether there are differences in its role between dicots and monocots.

ACKNOWLEDGEMENTS

This work was supported by a grant from the National Research Foundation of Korea (NRF), funded by the Korea government, Ministry of Education Science and Technology (MEST) (No. 2010-0011395, 2011-0017947, 2012-0004968).

KEYWORDS: PSII-LHCII supercomplex mobilization, high light, phosphorylation, PSII core proteins, state transitions.

Received April 21, 2013; Accepted May 21, 2013.

REFERENCES AND NOTES

1. Caffarri, S.; Kouřil, R.; Kereiche, S.; Boekema, E. J.; Croce, R. *EMBO J.* **2009,** *28*, 3052-3063.
2. Kargul, J.; Turkina, M. V.; Nield, J.; Benson, S.; Vener, A. V.; Barber, J. *FEBS J.* **2005,** *272*, 4797-4806.
3. Galka, P.; Santabarbara, S.; Khuong, T. T. H.; Degand, H.; Morsomme, P.; Jennings, R. C.; Boekema, E. J.; Caffarri, S. *Plant Cell* **2012,** *24*, 2963-2978.
4. Depège, N.; Bellafiore, S.; Rochaix, J. D. *Science* **2003,** *299,* 1572-1575.

5. Pesaresi, P.; Hertle, A.; Pribil, M.; Kleine, T.; Wagner, R.; Strissel, H.; Ihnatowicz, A.; Bonardi, V.; Scharfenberg, M.; Schneider, A. *Plant Cell* **2009,** *21,* 2402-2423.
6. Jansson, S. *Bioenergetics* **1994,** *1184,* 1-19.
7. Boekema, E. J.; van Roon, H,; Calkoen, F.; Bassi, R.; Dekker, J. P. *Biochemistry* **1999,** *38,* 2233-2239.
8. de Bianchi, S.; Betterle, N.; Kouril, R.; Cazzaniga, S.; Boekema, E.; Bassi, R.; Dell'Osto, L. *Plant Cell* **2011,** *23,* 2659-2679.
9. Yakushevska, A. E.; Keegstra, W.; Boekema, E. J.; Dekker, J. P.; Andersson, J.; Jansson, S.; Ruban, A. V.; Horton, P. *Biochemistry* **2003,** *42,* 608-613.
10. Andersson, J.; Walters, R. G.; Horton, P.; Jansson, S. *Plant Cell* **2001,** *13,* 1193-1204.
11. Mioslavina, Y.; de Bianchi, S.; Dell'Osto, L.; Bassi, R.; Holzwarth, A. R. *J. Biol. Chem.***2011,** *286,* 36830-36840.
12. Kouril, R.; Dekker, J. P.; Boekema, E. J. *Biochim. Biophys. Acta* **2012,** *1817,* 2-12.
13. Buchel, C.; Kuhlbrandt, W. *Photosynth. Res.* **2005,** *85,* 3-13.
14. Croce, R.; van Amerongen, H. *J. Photochem. Photobiol. B: Biol.* **2011,** *104,* 143-153.
15. Caffarri, S.; Broess, K.; Croce, R.; van Amerongen, H. *Biophys. J.* **2011,** *100*, 2094-2103.
16. Damkjær, J. T., Kereïche, S.; Johnson, M. P.; Kovacs, L.; Kiss, A. Z.; Boekema, E. J.; Ruban, A. V.; Horton, P.; Jansson, S. *Plant Cell* **2009,** *21*, 3245-3256.
17. Kouril, R.; Zygadlo, A.; Arteni, A. A.; de Wit, C. D.; Dekker, J. P.; Jensen, P. E.; Scheller, H. V.; Boekema, E. J. *Biochem.* **2005,** *44*, 10935-10940.
18. Tokutsu, R.; Iwai, M.; Minagawa, J. *J. Biol. Chem.* **2009,** *284*, 7777-7782.
19. Allen, J. F. *Biochim. Biophys. Acta* **1992,** *1098*, 275-335.
20. Minagawa, J. *Biochim. Biophys. Acta* **2011,** *1807*, 897-905.
21. Kargul, J.; Barber, J. *FEBS J.* **2008,** *275*, 1056-1068.
22. Minagawa, J. *Biochim Biophys. Acta* **2011,** *1807*, 897-905.
23. Dietzel, L.; Bräutigam, K.; Steiner, S.; Schüffler, K.; Lepetit, B.; Grimm, B.; Schttler, M. A.; Pfannschmidt, T. Plant Cell **2011,** *23*, 2964-2977.
24. García-Cerdán,; J. G.; Kovács, L.; Tóth, T.; Kereïche, S.; Aseeva, E.; Boekema, E. J.; Mamedov, F.; Funk, C.; Schröder, W. P. *Plant J.* **2011,** *65*, 368-381.
25. Tikkanen, M.; Nurmi, M.; Kangasjärvi, S; Aro, E.-M. *Biochim. Biophys. Acta* **2008,** 1777, 1432-1437.
26. Fristedt, R.; Vener, A.V. *PLoS ONE*, **2011,** *6(9)*: e24565. doi:10.1371/journal.pone.0024565.
27. Kereïche, S.; Kiss, A. Z.; Kouřil, R.; Boekema, E. J.; Horton, P. *FEBS Lett.* **2010,** *584*, 759-764.
28. Tikkanen, M.; Piippo, M.; Suorsa, M.; Sirpio, S.; Mulo P.; Vainonen J.; Vener, A. V.; AllahverdiyevaY.; Aro E.-M. *Plant Mol. Biol.* **2006,** *62*, 779-793.
29. Jensen, P. E.; Gilpin, M.; Knoetzel, J.; Scheller, B. *J. Biol. Chem.* **2000**, *275*, 24701-24708.
30. Lunde, C.; Jensen, P. E.; Haldrup, A.; Knoetzel, J.; Scheller, H. V. *Nature.* **2000,** *408*, 613-615.
31. Fristedt, R.; Granath, P.; Vener, A. V. *PLoS ONE* **2010,** 5(6):e10963. doi:10.1371/journal.pone.0010963.
32. Wientjes, E.; Amerongen, H. V.; Croce, R. *Biochim. Biophys. Acta* **2013,** *1827*, 420-426.
33. Chen, Y.-E.; Zhao, Z.-Y.; Zhang, H.-Y.; Zeng, X.-Y.; Yuan S. *J. Exp. Bot.* **2013,** *64*, 1167-1178.
34. Hwang, H.-J.; Xu, C. C.; Moon, B.-Y.; Lee, C.-H. *J. Plant Biol.* **2003,** *46*, 122-129.