FEBS Letters 571 (2004) 26–30 FEBS 28601

Two independent light signals cooperate in the activation of the plastid *psbD* blue light-responsive promoter in *Arabidopsis*

Takateru Mochizuki[a], Yayoi Onda[a], Emi Fujiwara[a], Masamitsu Wada[b], Yoshinori Toyoshima[a,*]

[a]*Department of Bioscience and Nano-biotechnology Research Center, School of Science and Technology, Kwansei Gakuin University, Gakuen 2-1, Sanda, Hyogo 669-1337, Japan*
[b]*Department of Biological Sciences, Graduate School of Science, Tokyo Metropolitan University, Hachioji, Tokyo 192-0397, Japan*

Received 21 May 2004; revised 21 June 2004; accepted 21 June 2004

Available online 1 July 2004

Edited by Ulf-Ingo Flügge

Abstract The *psbD* blue light-responsive promoter (BLRP), whose activation has been considered to require strong blue light, is recognized only by SIG5 among six σ factors of plastid RNA polymerase in *Arabidopsis*. We found *SIG5* transcript accumulation was rapidly induced after a 30-min induction time by blue light (470 nm) with an intensity threshold of 5 μmol m^{-2} s^{-1} through cryptochromes. Besides this weak blue light, the *psbD* BLRP activation required the stronger light such as 50 μmol m^{-2} s^{-1} irrespective of blue or red light (660 nm). Thus, the two independent light signalings, the cryptochrome-mediated signaling to induce *SIG5* transcription and the stronger light-dependent signaling, cooperate to activate the *psbD* BLRP.

Keywords: Plastid-encoded plastid RNA polymerase; σ factor; SIG5; *psbD* blue light-responsive promoter; Blue light; Cryptochrome

1. Introduction

The photosystem II reaction center proteins, D1 and D2, are intensively damaged under high irradiances [1,2]. To maintain high rates of their synthesis, transcription of *psbA* and *psbD*, the chloroplast genes encoding D1 and D2, respectively, are elevated in response to light [3–5].

psbA and *psbD* are transcribed by plastid-encoded plastid RNA polymerase (PEP), a eubacterial-type multi-subunit enzyme in which the core enzyme consisting of the plastid-encoded subunits (2α, β, β′, β″) is assembled with a nuclear-encoded σ factor [6–9]. PEP has been found to be different from such a prokaryotic enzyme in being associated with further additional proteins in chloroplasts (reviewed in [10]). Transcription of *psbA*, as well as *psaA* and *psaB* encoding the photosystem I reaction center proteins, is regulated by photosynthetic light via the redox state of plastoquinone pool in mustard [11], while *psbD* transcription has been considered to be differentially regulated by high fluence of blue light in various higher plants [3,5,12–18].

Under illumination, *psbD* transcription is activated from 190, 550, and 950 nucleotides upstream from the *psbD* translation start codon in *Arabidopsis* [15]. Transcripts from −190 and −550 positions are readily accumulated by light in a similar profile to the 16S rRNA, the *rbcL* and *psbA* mRNAs, whereas transcription from −950 position is relatively delayed in time [15] and induced differentially by high fluence of blue light such as 50 μmol m^{-2} s^{-1} under the blue light-responsive promoter termed the *psbD* BLRP [12–18]. The *psbD* BLRP is a σ^{70}-type but unique promoter that lacks the functional −35 promoter element [17,18]. In *Arabidopsis*, there occur six genes for σ factors, *SIG1-6*, which contain the highly conserved sub-domains defined for eubacterial σ^{70} factors [19–21]. In addition to the above three transcripts of *psbD* gene, transcripts from −256 position has recently been found [22]. Accumulation of −256 transcripts markedly and specifically decreases in the *sig2*-deficient mutant [22], while light-activated accumulation of transcripts from the *psbD* BLRP severely decreases in the *SIG5*-deficient mutant [23], suggesting that the promoter for −256 transcription and the *psbD* BLRP are recognized by different σ factors and that SIG5 is responsible for blue light-dependent activation of the *psbD* BLRP. *SIG5* transcription is induced by blue light, but not by red light [24]. However, the strength of monochromatic blue light required for inducing *SIG5* transcript accumulation is lower than that for activating the *psbD* BLRP by one order of magnitude [5,12–18,24].

Here, we analyze the effects of light intensity and wavelength on kinetic behaviors of *SIG5* transcript accumulation and the *psbD* BLRP activation, and show that at least two independent light signals cooperate to activate the *psbD* BLRP: low fluence of blue light to induce *SIG5* transcript accumulation and high fluence of light such as 50 μmol m^{-2} s^{-1} which is not restricted to blue light. Furthermore, we determine cryptochromes rather than phototropins as photoreceptor for blue light-dependent induction of *SIG5* transcript accumulation by mutational analyses. The role of high fluence light in the *psbD* BLRP activation is discussed.

2. Materials and methods

2.1. Plant materials

Arabidopsis thaliana was grown on compound soil (Jiffy7, Sakata Seed) for 4 weeks at 22 °C under continuous white light (15 μmol m^{-2} s^{-1}). The seeds used were: the wild type (ecotype Columbia);

* Corresponding author. Fax: +81-79-565-7613.
E-mail address: ytoyoshima@ksc.kwansei.ac.jp (Y. Toyoshima).

Abbreviations: PEP, plastid-encoded plastid RNA polymerase; *psbD* BLRP, *psbD* blue light-responsive promoter; cry, cryptochrome; phot, phototropin

0014-5793/$22.00
doi:10.1016/j.febslet.2004.06.052

the phototropin double-mutant, *phot1phot2* (*phot1-5phot2-1*) (ecotype Columbia) [25]; the cryptochrome double-mutant, *cry1cry2* (*hy4-3cry2-1*) (ecotype Wassilewskija) [26]; the cryptochrome single-mutants, *cry1* (*hy4-3*) (ecotype Wassilewskija) [27] and *cry2* (*cry2-1*) [28]. The *cry* mutants were provided by Dr. Lin. Each plant was dark-adapted for 24 h, and subsequently irradiated by white light (15 $\mu mol\,m^{-2}\,s^{-1}$), blue light (470 nm, indicated fluences) or red light (660 nm, indicated fluences) for the indicated times at 22 °C using LED panels (LED-B or LED-R, EYELA), before harvested for RNA analysis. The photon-fluence rates were measured using a quantum photometer (LI-250, Li-Cor).

2.2. RT-PCR

Immediately after light treatment, rosette leaves were harvested and frozen in liquid N_2, and total RNA was isolated using RNeasy plant mini kit (Qiagen), followed by DNase I treatment (Qiagen). Total RNA served as template for cDNA synthesis using reverse-transcriptase and oligo$(dT)_{12–18}$ primer or random primer (Roche). We analyzed the resulting cDNA by quantitative PCR method using Light Cycler (Roche) with SYBR Green I (Roche) and gene specific primers for each σ factor gene, the sequence just downstream from the *psbD* BLRP or the gene coding actin2 (*ACT2*), which has been reported to expressed constitutively [29]. In order to determine the levels of their transcripts quantitatively, we also carried out PCR using each corresponding gene cloned into vector as template to produce a standard curve. The resulting values were normalized on the basis of *ACT2* as an internal standard. For *SIG*s, the primers used were as follows: for *SIG1* (AB019942), 5′-ggaagttgtgcgcttgtcta-3′ and 5′-caaacgcacattactcatagcc-3′; for *SIG2* (AB019943), 5′-atcaggtattccgtctgtgaag-3′ and 5′-acgtccactacgctctgtaag-3′; for *SIG3* (AB 019944), 5′-agctgagaatggtgatcagag-3′ and 5′-cactttcttcctccagttgtg-3′; for *SIG4* (AB021119), 5′-gtcgtttagaggaggttcagc-3′ and 5′-accaacctacggtaacaacg-3′; for *SIG5* (AB021120), 5′-tgatatagtgagcttggactgg-3′ and 5′-cttgcagctctacctatttcg-3′; for *SIG6* (AB029916), 5′-tcctcagaaaactagtgcgaag-3′ and 5′-ggaacttatccccatagcttca-3′. The primers for *ACT2* were 5′-atgaatgggatcaaagtttc and 5′-acaagtgcatcatagaaacg-3′, to amplify 96-bp fragment of its 3′ UTR. For the *psbD* BLRP, were used 5′-ggaaatccgtcgatatctct-3′ and 5′-ctctctttctctaggcaggaac-3′, as sense and anti-sense primers, respectively, designated to amplify the fragment from −887 to −801, just downstream from the *psbD* BLRP transcription initiation site. In order to exclude a possibility that the *psbD* BLRP transcripts analyzed using these primers might be contaminated with transcripts originating from promoters upstream, the reverse transcription products were subjected to PCR using the sequence upstream from the *psbD* BLRP transcription initiation site (5′-agtaagtggacctaacccatcg-3′) as sense primer without altering the anti-sense primer. With these primers, there was no significant transcript accumulation observed under blue light illumination (50 $\mu mol\,m^{-2}\,s^{-1}$). The specificity of each pair of the primers was examined using *SIG1-6* DNAs cloned into pUC18 [24]: their cross-reactivity was 0.01–0.00001% (data not shown). The fragments amplified from cDNA showed a single band with the mobility corresponding to each mature mRNA size in agarose gel electrophoresis and a sharp single melting peak identical to that from the corresponding clone (data not shown).

The resulting cDNA was also subjected to PCR (preheated at 94 °C for 10 min, then 35 cycles, each of 94 °C for 30 s, 53 °C for 30 s, and 72 °C for 1 min), using AmpliTaqGold (Applied Biosystems). The primers used were: for *SIG5*, to amplify 650-bp fragment, 5′-aggaataatgtgaagaaggaaaaac-3′ and 5′-tctttgctttatatatctccactct-3′; for *ACT2*, the same primers as described above.

3. Results and discussion

3.1. Blue light-specific induction of SIG5 transcript accumulation and its photoreceptor

We examined the effects of blue light and red light on accumulation of transcripts from each σ factor gene. Illumination of dark-adapted plants with blue light at 5 $\mu mol\,m^{-2}\,s^{-1}$ caused an extensive induction of *SIG5* transcript accumulation in comparison to that under red light of the same intensity (Fig. 1), being consistent with the previous report [24]. Such a blue light-specific induction was observed only in *SIG5* among the six σ genes: accumulation of transcripts from the other five σ factor genes was induced by red light as well as by blue light. The induction efficiency of blue light to red light on *SIG5* transcript accumulation was approximately 10 times as high as on transcript accumulation from the other σ factor genes.

To determine the photoreceptor mediating the induction of *SIG5* transcript accumulation, we compared the effects of

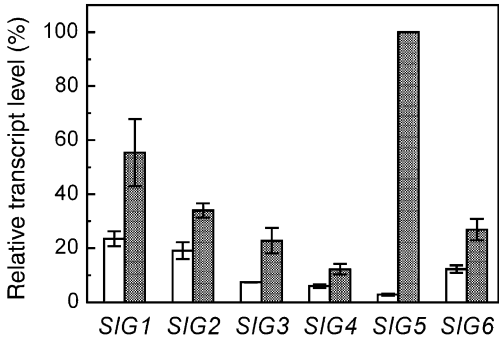

Fig. 1. Effect of light quality on the levels of σ factor gene transcripts. After dark-adaptation, the wild type plants were exposed to blue light (closed bar) or red light (open bar) at the intensity of 5 $\mu mol\,m^{-2}\,s^{-1}$ each for 90 min. The levels of *SIG1-6* transcripts were determined by quantitative PCR with the primers specific to each σ factor gene. Each value was normalized to that of *ACT2*, as described in Section 2, and converted to a percentage of the highest transcript level. The data are shown as mean values with standard deviation of three independent measurements.

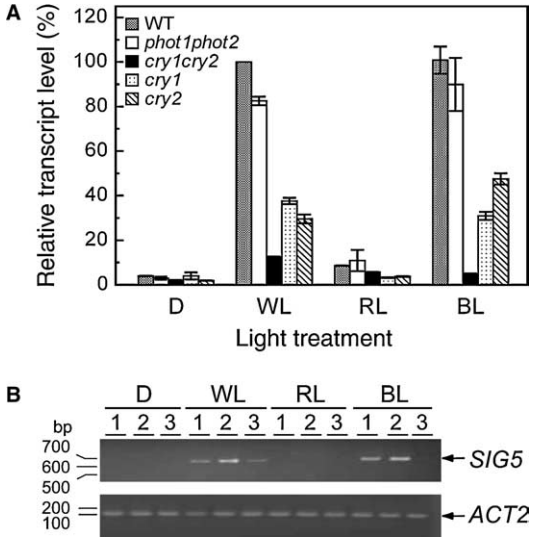

Fig. 2. *SIG5* transcript levels in illuminated leaves of the wild type, *phot1phot2*, *cry1cry2*, *cry1* and *cry2*. Each plant grown under continuous white light was exposed to darkness (D), and subsequently illuminated for 90 min with white light (WL, 15 $\mu mol\,m^{-2}\,s^{-1}$), red light (RL, 5 $\mu mol\,m^{-2}\,s^{-1}$) or blue light (BL, 5 $\mu mol\,m^{-2}\,s^{-1}$). (A) *SIG5* transcript levels were determined by quantitative PCR. Each value was converted to a percentage of that of the wild type exposed to WL. The data are shown as mean values with standard deviation of three independent experiments. (B) Semi-quantitative RT-PCR products from *SIG5* (upper panel) and *ACT2* (lower panel) were separated in 2% agarose gels and visualized by ethidium bromide staining. Lanes 1: the wild type; lanes 2: *phot1phot2*; lanes 3: *cry1cry2*.

lights on *SIG5* transcript accumulation in the wild type, the double mutants of *cry1cry2* and *phot1phot2*, lacking cryptochromes (cry) 1 and 2, and phototropins (phot) 1 and 2, respectively, and the cry single mutants. After dark adaptation of plants grown under continuous lights, *SIG5* transcript accumulation disappeared in all the plants examined (Fig. 2A and B). The quantitative PCR analyses showed that the subsequent illumination with blue light caused a sharp contrast in *SIG5* transcript accumulation between the wild type and *cry1cry2* (Fig. 2A). Illumination of the wild type with blue light (5 $\mu mol\,m^{-2}\,s^{-1}$) for 90 min dramatically increased the level of *SIG5* transcripts. Blue-light illumination of *cry1cry2*, however, hardly caused an increase in the level of *SIG5* transcripts, while that of *phot1phot2* increased the level of *SIG5* transcripts almost to the wild-type level. Blue-light illumination of the single mutants, either *cry1* or *cry2*, expressing cry2 or cry1 to the wild-type level, respectively [28,30], induced *SIG5* transcript accumulation to a nearly half of the wild-type level. These results indicate that cryptochromes rather than phototropins are photoreceptor to mediate the blue light-specific induction of *SIG5* transcript accumulation, and both cry1 and cry2 function as photoreceptor. This is consistent with the recent report that mutational deficiency of Ser/Thr protein phosphatase, PP7, which acts downstream of cryptochrome, causes a decrease in *SIG5* transcript accumulation [31]. Since transcripts from *ACT2* accumulated at the equivalent level in all the cases examined (Fig. 2B), we used it as an internal standard for normalization, as described in Section 2.

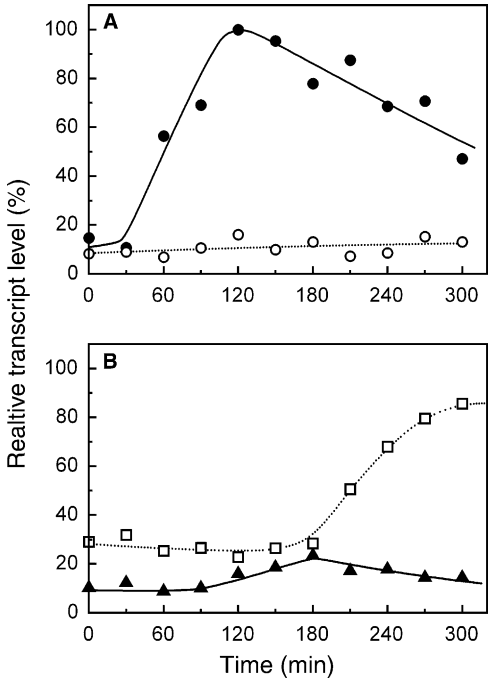

Fig. 3. Kinetics of *SIG5* transcript accumulation under illumination with blue light or red light. (A) After dark adaptation, the wild type (●, solid line) and *cry1cry2* (○, dot line) plants were exposed to illumination with blue light (5 $\mu mol\,m^{-2}\,s^{-1}$) for the indicated times. *SIG5* transcript levels were determined by quantitative PCR. (B) Dark-adapted plants of the wild type were exposed to illumination with red light (50 $\mu mol\,m^{-2}\,s^{-1}$) for the indicated times. Transcript levels were analyzed for *SIG5* (▲, solid line) and *SIG2* (□, dot line) by quantitative PCR. Each value was converted to a percentage of the highest transcript level in (A).

3.2. Kinetics of SIG5 transcript accumulation in response to blue light and red light

Kinetic analyses showed that illumination with low fluence blue light (5 $\mu mol\,m^{-2}\,s^{-1}$) rapidly increased the level of *SIG5* transcripts after an induction time of 30 min in the wild type, while such a blue light-dependent induction of *SIG5* transcript accumulation was not observable in *cry1cry2* (Fig. 3A). In contrast to *SIG5*, red light as well as blue light caused the induction of *SIG2* transcript accumulation, as shown in Fig. 1. Red-light illumination of *cry1cry2* induced *SIG2* transcript accumulation in a similar manner to that of the wild type (data not shown), suggesting that the induction deficiency of *SIG5* transcript accumulation in *cry1cry2* is not due to the ecotype difference of *cry1cry2* (Wassilewskija) from the wild type (Columbia).

Accumulation of *SIG2* transcripts, which remained to some extent after 24-h dark adaptation, was enhanced by red-light illumination, but it occurred with an induction time of 180 min (Fig. 3B). Such a red light-induction of *SIG2* transcript accumulation (Fig. 3B) was much slower than blue-light induction of *SIG5* transcript accumulation (Fig. 3A). The origin of such a difference in the kinetic features between blue-light and red-light inductions remains to be examined.

We next examined blue light intensity dependency of *SIG5* transcript accumulation. The blue light-induced accumulation of *SIG5* transcripts required blue light with a fluence threshold of about 5 $\mu mol\,m^{-2}\,s^{-1}$ (Fig. 4), being consistent with the fluence threshold for the cryptochrome activation by phosphorylation [32,33]. The level of *SIG5* transcripts increased with the exposure time from 30 to 120 min, and then decreased after prolonged blue light treatment. An increase in intensity up to 10 $\mu mol\,m^{-2}\,s^{-1}$ did not make a difference in the kinetics of *SIG5* transcript accumulation. In *cry1cry2*, however, such a blue light-induced *SIG5* transcript accumulation was hardly observed at either 5 or 10 $\mu mol\,m^{-2}\,s^{-1}$ up to 240 min.

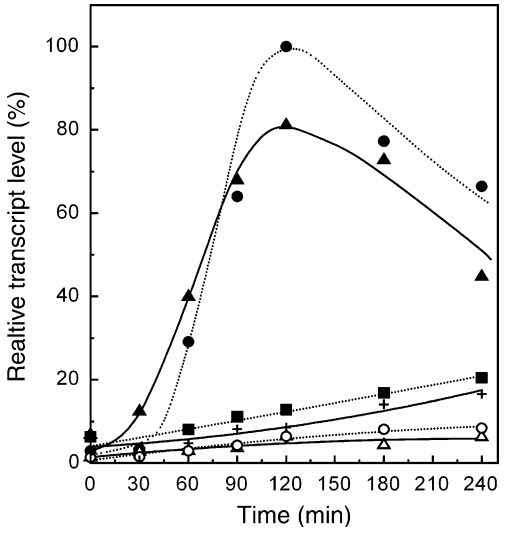

Fig. 4. Effect of blue light intensity on kinetics of *SIG5* transcript accumulation. Dark-adapted plants of the wild type (closed symbols) and *cry1cry2* (open symbols) were illuminated with blue light at the intensities of 1 (+, solid line), 3 (■, dot line), 5 (▲, solid lines) and 10 (●, dot lines) $\mu mol\,m^{-2}\,s^{-1}$ for the indicated times. *SIG5* transcript levels were determined by quantitative PCR. Each value was converted to a percentage of the highest transcript level.

3.3. Cooperative effects of blue light and high fluence light on the process of the psbD BLRP activation

The present results suggest that the blue light requirement for the *psbD* BLRP activation is presumably due to that for *SIG5* transcription. In contrast to *SIG5*, however, the accumulation of transcripts from the *psbD* BLRP was hardly induced by illumination with low fluence (5 $\mu mol\,m^{-2}\,s^{-1}$) of blue light for 180 min (Fig. 5A). As reported previously [24], high fluence of red light (50 $\mu mol\,m^{-2}\,s^{-1}$) alone hardly activated the *psbD* BLRP (Fig. 5A). Interestingly, accumulation of the *psbD* BLRP transcripts appeared at a high level, when dark-adapted plants were illuminated with low fluence of blue light together with high fluence (50 $\mu mol\,m^{-2}\,s^{-1}$) of red light (Fig. 5A).

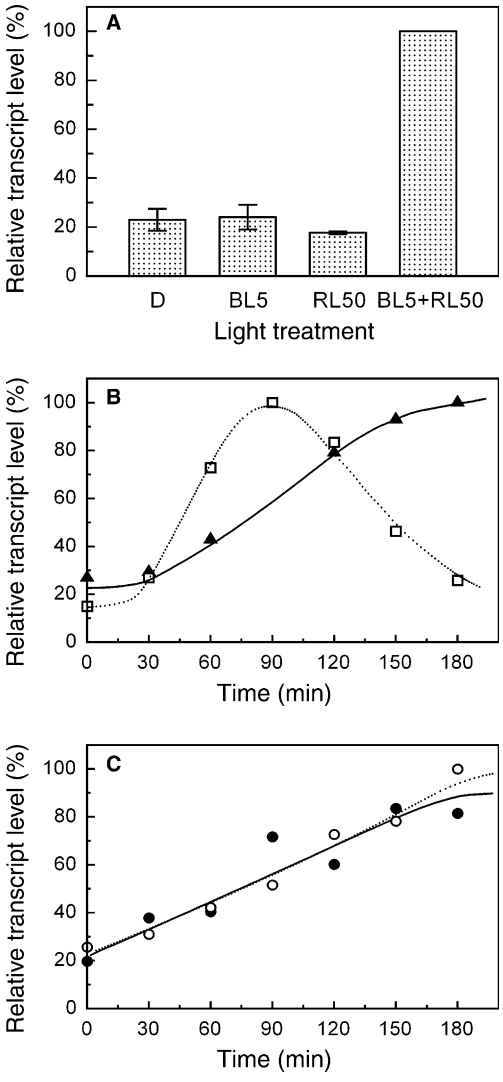

Fig. 5. Effect of blue light and red light on accumulation of transcripts from the *psbD* BLRP. (A) Dark-adapted plants of the wild type (D) were exposed to illumination for 180 min with blue light (5 $\mu mol\,m^{-2}\,s^{-1}$, BL5) or red light (50 $\mu mol\,m^{-2}\,s^{-1}$, RL50) alone, or simultaneously with both these lights (5 $\mu mol\,m^{-2}\,s^{-1}$ blue light and 50 $\mu mol\,m^{-2}\,s^{-1}$ red light, BL5 + RL50). Transcript levels from the *psbD* BLRP were determined by quantitative PCR, as described in Section 2. The data are shown as mean values with standard deviation of three independent measurements. (B) Dark-adapted plants of the wild type were initially illuminated with red light (50 $\mu mol\,m^{-2}\,s^{-1}$) for 180 min, followed by shift to blue light (5 $\mu mol\,m^{-2}\,s^{-1}$) for the times indicated. Transcript levels were analyzed for *SIG5* (□, dot line) and the *psbD* BLRP (▲, solid line) by quantitative PCR with their specific primers. (C) Dark-adapted plants were initially illuminated with blue light (5 $\mu mol\,m^{-2}\,s^{-1}$) for 180 min, followed by shift to red light (●, solid line) or blue light (○, dot line) at the intensity of 50 $\mu mol\,m^{-2}\,s^{-1}$ each for the times indicated. Transcript levels from the *psbD* BLRP were determined by quantitative PCR. Each value was converted to a percentage of the highest transcript level.

To examine the effect of high fluence light on activating the *psbD* BLRP separately from that of blue light, we used red light for high fluence irradiance (50 $\mu mol\,m^{-2}\,s^{-1}$) and low fluence of blue light (5 $\mu mol\,m^{-2}\,s^{-1}$) to induce *SIG5* transcript accumulation without any effect on the *psbD* BLRP activation. When plants were initially exposed to high fluence of red light, the level of *SIG5* transcripts increased with an induction time of 30 min in response to the subsequent shift to low fluence of blue light (Fig. 5B), in a similar profile to blue-light induction of *SIG5* transcript accumulation without red-light preillumination (Fig. 3A). The transcript accumulation from the *psbD* BLRP followed that from *SIG5* but with a timelag (Fig. 5B). On the other hand, in plants pretreated with low fluence of blue light to accumulate SIG5, the level of the *psbD* BLRP transcripts increased immediately in response to the subsequent shift to high fluence of red light (Fig. 5C). In the blue light-preilluminated plants there was no significant difference in the efficiency of the *psbD* BLRP activation between red light and blue light (Fig. 5C). These results indicate that these two light signaling pathways function differentially but cooperatively to activate the *psbD* BLRP.

The *psbD* BLRP has been considered to require high fluence of blue light to be activated, as described in Section 1 [5,12–18]. Those results, however, are obtained by using monochromatic lights. The present results obtained using various combinations of blue and red lights clearly show that requirements of blue light and high fluence light for the *psbD* BLRP activation are attributed to the different origins. One is the induction of the nuclear gene *SIG5* transcript accumulation through cryptochrome-dependent signaling. The other is a high light-dependent event which supposedly occurs in plastids. The mechanism by which high light functions on the activation of the *psbD* BLRP remains to be revealed, but two different scenarios likely to imply its role would be developed from the previous reports.

The *psbD* BLRP contains the well-conserved upstream *cis*-elements, AAG box and PGT box, which are the binding sites of nuclear-encoded AAG box-binding factor (AGF) and PGT box-binding factor (PGTF), respectively [14,17,34,35]. Contrary to AGF constitutively binding to AAG box, PGTF is suggested to bind to PGT box light-dependently so as to activate the *psbD* BLRP, based on the finding that ADP- rather than ATP-dependent PGTF phosphorylation results in loss of the affinity for the PGT box in vitro [36]. The *psbD* BLRP activity might be controlled by the blue light-dependent induction of *SIG5* transcription in the nucleus and by high fluence light-dependent phosphorylation/dephosphorylation of PGTF in plastids independently.

Alternatively, plastid transcription kinase (PTK) has been identified as one of the PEP-associated accessory proteins, to catalyze phosphorylation of PEP core enzyme and/or σ factor ATP-dependently, resulting in inactivation of PEP [10,37,38]. The *psbD* BLRP activity might be controlled by the blue light-dependent induction of *SIG5* transcription in the nucleus via cryptochrome-mediated signaling and by posttranslational modification of SIG5 and/or PEP core through PTK via high

fluence light-dependent signaling in plastids. Such a PTK-mediated mechanism has been found only in *Sinapis alba* so far [37,38].

Recently, a novel FAD-bound protein, At-cry3, has been identified as a chloroplast-targeted cryptochrome-like protein in *Arabidopsis* [39]. It has been shown that the *psbD* BLRP activation is mediated by blue light through cry1 or cry2 [40], which accumulates in the nucleus constitutively or in the cytosol in the light, respectively [41]. This is well explained by the present result that the accumulation of transcripts from the nuclear gene *SIG5* is induced through cry1 and cry2 (Figs. 2 and 3). Such an induction of *SIG5* transcript accumulation is unlikely to be mediated through the chloroplast-targeted cry3. On the other hand, cry3 might be involved in the high light-dependent event for the *psbD* BLRP activation in chloroplasts. However, based on the present results that high fluence of red light is equivalent to that of blue light in the *psbD* BLRP activation (Fig. 5C), the cry3 involvement in this event appears to be unlikely. This should be clear.

Acknowledgements: We thank Dr. Chentao Lin (University of California, USA) for providing seed stocks of *cry1*, *cry2* and *cry1cry2*. We also thank Kazusa DNA Research Institute (Japan) for providing *ACT2* EST clone. This work was supported in part by a special grant-in-aid on Advanced Program of High Profile Research for Academia-Industry Cooperation sponsored by Ministry of Education and Science of Japan (Y.O. and Y.T.) and by Kwansei Gakuen University for Special Individual Research (Y.T.). This work was also founded by the New Energy and Industrial Technology Development Organization (Y.O. and Y.T.).

References

[1] Mattoo, A.K., Hoffman-Falk, H., Marder, J.B. and Edelman, M. (1984) Proc. Natl. Acad. Sci. USA 81, 1380–1384.
[2] Mattoo, A.K., Marder, J.B. and Edelman, M. (1989) Cell 56, 241–246.
[3] Gamble, P.E. and Mullet, J.E. (1989) EMBO J. 8, 2785–2794.
[4] Klein, R.R. and Mullet, J.E. (1990) J. Biol. Chem. 265, 1895–1902.
[5] Christopher, D.A. and Mullet, J.E. (1994) Plant Physiol. 104, 1119–1129.
[6] Hu, J. and Bogorad, L. (1990) Proc. Natl. Acad. Sci. USA 87, 1531–1535.
[7] Allison, L.A., Simon, L.D. and Maliga, P. (1996) EMBO J. 15, 2802–2809.
[8] DeSantis-Maciossek, G., Kofer, W., Bock, A., Schoch, S., Maier, R.M., Wanner, G., Rudiger, W., Koop, H.U. and Herrmann, R.G. (1999) Plant J. 18, 477–489.
[9] Allison, L.A. (2000) Biochimie 82, 537–548.
[10] Link, G. (2001) in: Regulation of Photosynthesis (Aro, E.M. and Andersson, B., Eds.), pp. 85–107, Kluwer Academic, Dordrecht.
[11] Pfannschmidt, T., Nilsson, A. and Allen, J.F. (1999) Nature 397, 625–628.
[12] Christopher, D.A., Kim, M. and Mullet, J.E. (1992) Plant Cell 4, 785–798.
[13] Wada, T., Tsunoyama, Y., Shiina, T. and Toyoshima, Y. (1994) Plant Physiol. 104, 1259–1267.
[14] Allison, L.A. and Maliga, P. (1995) EMBO J. 14, 3721–3730.
[15] Hoffer, P.H. and Christopher, D.A. (1997) Plant Physiol. 115, 213–222.
[16] Nakahira, Y., Baba, K., Yoneda, A., Shiina, T. and Toyoshima, Y. (1998) Plant Physiol. 118, 1079–1088.
[17] Kim, M., Thum, K.E., Morishige, D.T. and Mullet, J.E. (1999) J. Biol. Chem. 274, 4684–4692.
[18] Satoh, J., Baba, K., Nakahira, Y., Tsunoyama, Y., Shiina, T. and Toyoshima, Y. (1999) Plant J. 18, 407–415.
[19] Tanaka, K., Tozawa, Y., Mochizuki, N., Shinozaki, K., Nagatani, A., Wakasa, K. and Takahashi, H. (1997) FEBS Lett. 413, 309–313.
[20] Isono, K., Shimizu, M., Yoshimoto, K., Niwa, Y., Satoh, K., Yokota, A. and Kobayashi, H. (1997) Proc. Natl. Acad. Sci. USA 94, 14948–14953.
[21] Fujiwara, M., Nagashima, A., Kanamaru, K., Tanaka, K. and Takahashi, H. (2000) FEBS Lett. 481, 47–52.
[22] Hanaoka, M., Kanamaru, K., Takahashi, H. and Tanaka, K. (2003) Nucleic Acids Res. 31, 7090–7098.
[23] Tsunoyama, Y., Ishizaki, Y., Morikawa, K., Kobori, M., Nakahira, Y., Takeba, G., Toyoshima, Y. and Shiina, T. (2004) Proc. Natl. Acad. Sci. USA 101, 3304–3309.
[24] Tsunoyama, Y., Morikawa, K., Shiina, T. and Toyoshima, Y. (2002) FEBS Lett. 516, 225–228.
[25] Kinoshita, T., Doi, M., Suetsugu, N., Kagawa, T., Wada, M. and Shimazaki, K. (2001) Nature 414, 656–660.
[26] Ahmad, M., Jarillo, J.A, Smirnova, O. and Cashmore, A.R. (1998) Nature 392, 720–723.
[27] Ahmad, M. and Cashmore, A.R. (1993) Nature 366, 162–166.
[28] Guo, H., Yang, H., Mockler, T.C. and Lin, C. (1998) Science 279, 1360–1363.
[29] An, Y.Q., McDowell, J.M., Huang, S., McKinney, E.C., Chambliss, S. and Meagher, R.B. (1996) Plant J. 10, 107–121.
[30] Lin, C., Yang, H., Guo, H., Mockler, T., Chen, J. and Cashmore, A.R. (1998) Proc. Natl. Acad. Sci. USA 95, 2686–2690.
[31] Moller, S.G., Kim, Y.S., Kunkel, T. and Chua, N.H. (2003) Plant Cell 15, 1111–1119.
[32] Shalitin, D., Yang, H., Mockler, T.C., Maymon, M., Guo, H., Whitelam, G.C. and Lin, C. (2002) Nature 417, 763–767.
[33] Shalitin, D., Yu, X., Maymon, M., Mockler, T. and Lin, C. (2003) Plant Cell 15, 2421–2429.
[34] Kim, M. and Mullet, J.E. (1995) Plant Cell 7, 1445–1457.
[35] Baba, K., Nakano, T., Yamagishi, K. and Yoshida, S. (2001) Plant Physiol. 125, 595–603.
[36] Kim, M., Christopher, D.A. and Mullet, J.E. (1999) Plant Physiol. 119, 663–670.
[37] Tiller, K. and Link, G. (1993) EMBO J. 12, 1745–1753.
[38] Ogrzewalla, K., Piotrowski, M., Reinbothe, S. and Link, G. (2002) Eur. J. Biochem. 269, 3329–3337.
[39] Kleine, T., Lockhart, P. and Batschauer, A. (2003) Plant J. 35, 93–103.
[40] Thum, K.E., Kim, M., Christopher, D.A. and Mullet, J.E. (2001) Plant Cell 13, 2747–2760.
[41] Lin, C. (2002) Plant Cell, S207–S225.