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Red and far-red light alter the transcript profile in the cyanobacterium *Synechocystis* sp. PCC 6803: Impact of cyanobacterial phytochromes

Thomas Hübschmann[a], Hiroshi Yamamoto[b], Thomas Gieler[a], Norio Murata[b], Thomas Börner[a,*]

[a] *Institute of Biology, Humboldt University Berlin, Chausseestrasse 117, 10115 Berlin, Germany*
[b] *Department of Regulation Biology, National Institute for Basic Biology, Myodaiji, Okazaki 444-8585, Japan*

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Abstract Cyanobacteria possess genes encoding phytochrome-related proteins. We used a DNA microarray approach to evaluate the impact of the phytochromes Cph1 and Cph2 on red light (R)- and far-red light (FR)-dependent gene expression in the unicellular cyanobacterium *Synechocystis* sp. PCC 6803. In cells of wild-type and phytochrome mutants, one-fourth of all 3165 annotated putative protein encoding genes was light-responsive. R predominantly enhanced the expression of genes involved in transcription, translation, and photosynthesis, whereas FR upregulated the transcript level of genes known to be inducible by stress. The absence of Cph1 and/or Cph2 altered the light-dependent expression of about 20 genes. Hence, receptor(s) different from the two phytochromes are supposed to trigger the global R/FR alterations of the expression profile.

Keywords: Cyanobacteria; Red light; Far-red light; *gifA*; Gene expression; Phytochrome

1. Introduction

Photosynthetic organisms are exposed to diurnal and seasonal fluctuations of light. To optimally respond to changing light conditions, they possess wavelength-specific photoreceptors. In higher plants, phytochromes form a family of chromoproteins which are able to reversibly photoisomerize between two stable conformations, a red light (R)-absorbing Pr form, and a far-red light (FR)-absorbing Pfr form. In this way, they act as R/FR controlled switches which affect molecular and physiological processes including gene transcription, seed germination, organelle movement, entrainment of the circadian rhythm, flowering, and phototropism [1,2].

The first prokaryotic genes encoding phytochrome-like proteins have been detected in cyanobacteria [3,4]. Many photosynthetic bacteria and even several non-photosynthetic bacteria possess genes for phytochrome-like proteins [5]. The chromosome of the freshwater cyanobacterium *Synechocystis* sp. PCC 6803 (hereafter *Synechocystis* 6803) contains eight open reading frames (*slr0473*, *sll0821*, *sll1124*, *sll1473*/*sll1475*, *slr1393*, *slr1212*, *slr1969*, *sll0041*) exhibiting different degrees of similarity to plant genes coding for phytochromes [6]. Not all of them will be bona fide phytochromes in terms of an intrinsic bilin lyase activity and R/FR-dependent photochemistry. Indeed, PlpA (SLL1124) seems to be involved in blue-light-dependent growth, SLR1212 binds ethylene and PixJ1 (SLL0041) displays blue–green photoconversion [4,7,8]. Cph1 (cyanobacterial phytochrome 1; SLR0473) and Cph2 (cyanobacterial phytochrome 2; SLL0821), however, show phytochrome activity in vitro [9–13]. Insertional inactivation of *cph1* impaired the growth of mutant cells under continuous FR, whilst inactivation of *cph2* attenuated the growth under continuous R [14]. The molecular targets of Cph1 and Cph2 activity have not been identified yet. Like plant phytochromes, both cyanobacterial photoreceptors may exert their function by controlling gene expression.

We have studied the impact of R and FR on global gene expression patterns in wild-type and phytochrome mutant cells of *Synechocystis* 6803. Genome-wide expression profiling allowed for the identification of 473 genes responsive to R and 605 genes responsive to FR. The lack of functional Cph1 and Cph2 in mutant cells led mostly to quantitative changes of the transcript profiles as compared to the wild type.

2. Materials and methods

2.1. Growth conditions

Synechocystis sp. strain PCC 6803 (kindly provided by Sergey Shestakov, Moscow State University) was cultivated photoautotrophically in BG-11 medium [15] buffered with 20 mM HEPES–NaOH (pH 7.5) at 28 °C, under continuous white light illumination (Philips TLD 58W/865 'day light' white fluorescent tubes) at 70 μmol photons m^{-2} s^{-1} and bubbled with air. Cultivation was done in glass tubes (diameter 4 cm). Growth and cell densities were followed by measuring absorption at 730 nm (A_{730}) with a spectrophotometer (Lambda 35, Perkin–Elmer, USA). Pre-cultures of phytochrome mutants *cph1*$^-$, *cph2*$^-$, and *cph1*$^-$/*cph2*$^-$ [14] were grown under the same conditions with the exception that the medium contained 40 μg/ml kanamycin, and additional 7 μg/ml chloramphenicol in the case of mutant *cph1*$^-$/*cph2*$^-$. Mutant cells were transferred into medium without antibiotics for the final cell culture used for the R and FR experiments.

2.2. Irradiation

A 200 ml culture of wild-type or mutant cells was grown under white light from day light fluorescence tubes (Philips TLD 58W/865) as described up to A_{730} = 0.4–0.5. Each 100 ml of cell culture was transferred into new glass tubes and irradiated with either low R or FR (10 μmol photon m^{-2} s^{-1} each) for 90 min to allow acclimation of the cells to the respective light condition. The fluence rate of

*Corresponding author. Fax: +49 0 30 2093 8141.
E-mail address: thomas.boerner@rz.hu-berlin.de (T. Börner).

Abbreviations: R, red light; FR, far-red light; PSI, photosystem I; PSII, photosystem II

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10 μmol photon m^{-2} s^{-1} matches exactly the fluence rate which was used to photoconvert recombinant Cph1 from Pr into Pfr and vice versa [16]. Upon irradiation, 50-ml aliquots were withdrawn from each of the two cultures and mixed with equal volume of ice-cold ethanol/phenol (95/5, v/v). Cells were harvested by centrifugation at $5000 \times g$ for 5 min. Pellets were immediately frozen in liquid nitrogen and stored at −80 °C. The sample obtained from R-irradiation was referred to as 'R-acclimated'; the sample obtained from FR-irradiation was referred to as 'FR-acclimated'. Cells of the remaining 50 ml of 'R-acclimated' culture were subsequently exposed to FR (10 μmol photons m^{-2} s^{-1}), those of the 'FR-acclimated' culture to R (10 μmol photons m^{-2} s^{-1}). Upon 30 min irradiation, cells were harvested as described above. The first sample was referred to as 'FR-shifted', the second as 'R-shifted'. All irradiations were done under continuous air bubbling. R was provided by BL0307-50-44 LEDs (652 nm ± 20 nm, Kingbright, Taiwan). FR was obtained by filtering white halogen light (Walz, Germany) through a longpass RG9 filter (734 nm ± 30 nm, Schott, Germany). Wavelengths and fluence rates of R and FR were determined using a SR 9901 spectroradiometer (Macam Photometrics, UK).

2.3. DNA microarray analysis

Total RNA was extracted from *Synechocystis* 6803 cells as described [17]. DNA was removed by treatment with RNase-free DNase I (NipponGene, Japan). Synthesis and purification of Cy3-labeled and Cy5-labeled cDNAs (Amersham Biosciences, UK) were done with 10 μg of RNA using the RNA-Fluorescence Labeling Kit (TaKaRa, Japan) as described previously [18].

The *Synechocystis* 6803 DNA CyanoCHIP (ver. 2.1, TaKaRa, Japan) was used for hybridization of Cy3- and Cy5-labeled cDNAs. The microarrays covered 3074 of the 3165 open reading frames of *Synechocystis* 6803 (http://www.kazusa.or.jp/cyano/Synechocystis/index.html). Conditions of hybridization were the same as described [18]. After hybridization, the microarrays were washed in 2× SSC at 60 °C for 10 min, in 0.2× SSC, 0.1% SDS at 60 °C for 10 min, and twice in 0.2× SSC for 5 min at room temperature. Any moisture was completely removed by an air stream.

Hybridization of cDNAs was evaluated with an array scanner (GMS418, Affimetrix, CA). Signals were quantified using the ImaGene software version 5.5 (BioDiscovery, CA). Upon subtraction of the local background of each spot the signals were normalized by reference to the sum of all signals except those for rRNAs. Alterations in the expression level of a gene were calculated from the ratio of the relative amount of a transcript after light shift to the relative amount of a transcript before the light shift (R-shift vs. FR-acclimation and FR-shift vs. R-acclimation). Genes were considered affected by R or FR when the factor of induction/repression was >2.0 [19]. Each gene was spotted twice on the DNA chip which allowed chip-internal evaluation of signals.

2.4. Dot-blot hybridization

1.5 μg RNA samples were dot-blotted with a 'Bio-Dot Microfiltration Apparatus' according to the manufacturer's instructions (Bio-Rad, CA). Riboprobes were generated by PCR amplification of gene specific DNA fragments of *cpcBA* (*sll1577 sll1578*), *psbA2* (*slr1311*), *rpl2* (*sll1802*), *rbp2* (*ssr1480*), *hemH* (*slr0839*), *hspA* (*sll1514*), *gifA* (*ssl1911*), and 16 S rDNA, ligation to a T7-promoter DNA fragment using the 'Lig'nScribe' kit (Ambion, AU), and in vitro transcription in the presence of ^{32}P-γ UTP (Amersham, UK) using the 'MAXIscript' kit (Ambion, AU). Hybridizations were done as described [20]. Quantification of signals was performed with the help of a GS-525 Phosphor Imager (Bio-Rad, CA). Primers for amplification of gene-specific DNA fragments were: *cpcBA*, 5′-TCGTATGGCTGCTTGTTTGC-3′ and 5′-TGACGTAGACGACCGAAAGC-3′; *psbA2*, 5′-ACCGGATTTATGTCGGTTGG-3′ and 5′-GGATGTTGTGCTCAGCTTGG-3′; *rpl2*, 5′-TGCCCTGCTGTTCTACACC-3′ and 5′-AACCATACGCACTTCCTTGG-3′; *rbp2*, 5′-TGTCCATTTATGTCGGGAACC-3′ and 5′-GAGGGGTTCTCGGTCTTGC-3′; *hemH*, 5′-ATAGCCTGATCCCCTCTTGG-3′ and 5′-TCAATTTCTGCCTGGTAGGG-3′; *hspA*, 5′-TGAAACTGAAGAAGCCTATGTGC-3′ and 5′-TTTGGGCAAAGTCAAAGTTAGG-3′; *gifA*, 5′-CGCCACCACCAATTCATC-3′ and 5′-AAGCATTACTGCGGTCATAGG-3′; 16 S rDNA, 5′-AGCGTTATCCGGAATGATTG-3′ and 5′-CTAGCGATTCCTCCTTCACG-3′.

3. Results and discussion

3.1. General response of gene activity to R and FR

Exponentially grown cells of wild-type and mutants *cph1*$^-$, *cph2*$^-$, and *cph1*$^-$/*cph2*$^-$ were acclimated to respectively FR and R for 90 min. Aliquots were taken and the remaining cell cultures were exposed to R (if acclimated to FR) or to FR (if acclimated to R) for 30 min. The fluence rates of R and FR were kept constant throughout the experiment at an intensity of 10 μmol m^{-2} s^{-1}. Wavelengths of R (652 nm ± 20 nm) and FR (734 nm ± 30 nm) matched those at which the Cph1 holo-phytochrome isolated from *Synechocystis* 6803 absorbed maximally [16].

Whereas 15% of all genes of *Synechocystis* 6803 responded to a light shift from FR to R, it was 20% upon a shift from R to FR (Table 1). The highest rate of R/FR-response (55%) was found within the group of genes related to transcription or translation. Genes implicated in DNA replication or regulatory functions displayed the lowest degree of light impact (16%). A comparative analysis of the induction/repression ratios in response to R- and FR-shifts allowed us to classify all genes into four groups (Fig. 1). The partial overlap of groups I and IV in Fig. 1 comprises those genes which were induced by R and repressed by FR, whereas the overlap of groups II and III includes genes which were induced by FR and repressed by R (Table 1). Hence, both subgroups represent genes antagonistically affected by the two light treatments, a response that could be expected for genes controlled by phytochromes. A table of a subset of genes belonging to groups I and II can be found in Supplementary data Table 1. The entire list of affected genes is available in Supplementary data Table 2.

To exemplarily verify the DNA microarray data, we performed RNA dot-blot analysis of four genes. According to their light response, *rpl2* (ribosomal protein L2) and *rbp2* (RNA binding protein) belonged to groups I/IV, whereas *hemH* (ferrochelatase) and *hspA* (heat shock chaperone) belonged to groups II/III (Fig. 1). The results of the RNA dot-blot analysis confirmed the microarray-based classification. The ratios of induction or repression were in good agreement with those obtained by DNA microarray analyses (Supplementary data Fig. 1).

One of the most obvious effects of R was the enhanced expression of genes related to translation (see Supplementary data Table 1). Forty-five genes for ribosomal proteins were strikingly upregulated (up to 12-fold). R also stimulated the expression of subunits of the cyanobacterial RNA polymerase. The transcript levels rose three- to four-fold for *rpoA*, the operon *rpoC2B*, as well as for the essential, principal σ70 factor gene *sigA*. The concomitant upregulation of genes related to tRNA processing (*rnpA*, *rnd*), RNA binding (*rbp1*, *rbp2*) and protein biosynthesis (*tufA*, *efp*) indicates a general activation of housekeeping functions by R. Also genes related to photosynthesis, CO_2 fixation, and processes like biosynthesis of amino acids, fatty acids, purins, and pyrimidins were positively affected upon a shift from FR to R, whereas the reverse light treatment led to an overall decrease in the transcription of those genes. The data suggest a general stimulatory effect of R on the cellular metabolism, an observation that fits with R being the optimal light quality for energy production and growth of *Synechocystis*.

FR mostly diminished gene expression and concomitantly retarded growth under continuous illumination [14]. Although

Table 1
Differentially affected genes in response to a light shift from FR to R and from R to FR according to functional categories as defined by Cyanobase (http://www.kazusa.or.jp/cyano/cyano.html)

General pathway	Total no. of genes	No. of differentially expressed genes upon a				Enhanced by R and reduced by FR	Enhanced by FR and reduced by R
		Shift to R		Shift to FR			
		Enhanced	Reduced	Enhanced	Reduced		
Amino acid biosynthesis	83	9	2	1	18	4	0
Biosynthesis of cofactors, carriers, and prosthetic groups	116	16	7	4	33	14	2
Cell envelope	62	6	2	0	22	5	0
Cellular processes	60	5	9	7	9	1	4
Central intermediary metabolism	31	2	2	1	6	1	1
DNA replication, restriction, recombination, and repair	51	3	0	0	7	2	0
Energy metabolism	86	6	7	3	18	4	3
Fatty acid, phospholipids, and sterol metabolism	35	4	0	0	10	3	0
Other categories	169	8	15	10	29	4	6
Photosynthesis and respiration	128	30	10	3	37	18	2
Purines, pyrimidines, nucleosides, and nucleotides	38	6	0	0	11	3	0
Regulatory functions	156	1	12	3	14	0	2
Transcription	27	9	4	3	3	2	2
Translation	145	53	6	3	37	20	2
Transport and binding proteins	170	4	17	2	15	2	2
Hypothetical/unknown	1717	125	93	56	240	56	34
Total	3074	287	186	96	509	139	60

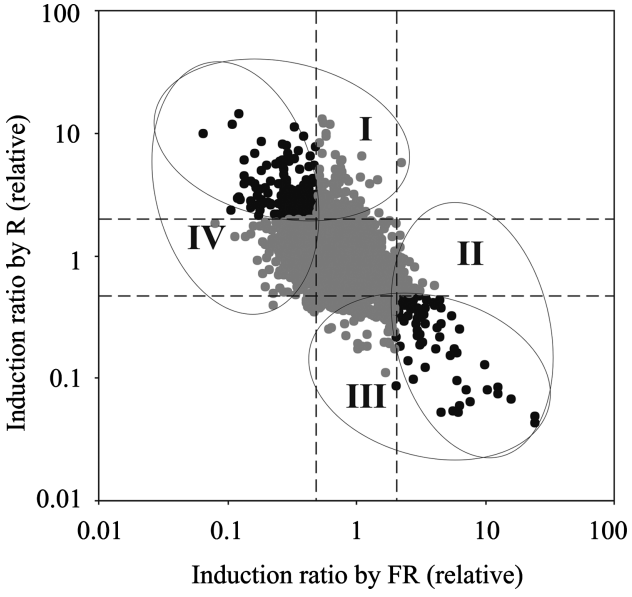

Fig. 1. Induction and repression of *Synechocystis* 6803 gene activity in response to FR and R. The position of a spot indicates the ratio of induction upon a shift from R to FR (abscissa) and upon a shift from FR to R (ordinate). Group I comprises genes induced by R, group II those induced by FR, group III those repressed by R, and group IV those repressed by FR. Black spots indicate genes antagonistically regulated by the two light treatments. The threshold of twofold induction and repression of gene activity is shown by dashed line.

FR had a more suppressive effect on the expression of genes attributed to transcription and translation, there were a few notable exceptions (Supplementary data Table 1). Whereas the mRNA level of *sigA* dropped significantly under FR that of the non-essential group II σ-factors *sigB* and *sigD* and of the group III σ-factor *sigG* rose up. The enhanced expression of the alternative sigma factors may indicate that *sigB*, *sigD*, and *sigG* are involved in reprogramming of the RNA polymerase under FR, thereby stimulating the activity of a number of genes (Supplementary data Table 1). Interestingly, FR led to a rise in the transcript level of chaperone and HLIP (high-light-inducible protein) genes.

The most dramatic light response displayed *hspA* encoding the small heat shock protein Hsp16.6 [21]. The HLIP genes *hliA* (*ssl2542*), *hliB* (*ssr2595*), and *hliC* (*ssl1633*) were upregulated up to 12-fold in FR and downregulated up to 10-fold in R. In cyanobacteria, HLIPs are critical for the acclimation of cells to variations in light intensity [22,23]. Those and other genes stimulated by FR are known to be activated upon exposure of cells to stresses such as high light, UV, high temperature, high salinity and/or high osmolarity [see compilation in Supplementary data Table 1 and Refs. [17,24,25]]. Hence, the response to FR may be mediated at least partly by signal transduction chains which are induced by several environmental cues.

3.2. Phytochrome effects

Nearly 200 genes of *Synechocystis* 6803 responded inversely to R and FR suggesting a possible involvement of phytochrome-like photoreceptors. Profiling the R/FR-dependent transcript accumulation in the single mutants *cph1*$^-$ and *cph2*$^-$ and the double mutant *cph1*$^-$/*cph2*$^-$ revealed differences in the light response of only 21 genes (Table 2) suggesting that R and FR affect the gene activity in *Synechocystis* not only via the phytochromes Cph1 and Cph2. R and FR alter the redox poise of components of the photosynthetic electron transport with known effects on gene transcription [26,27]. Indeed, several (but clearly not all) of the genes affected by R and FR in our experiments have been reported to respond to redox changes induced by inhibitors of photosynthetic electron transport

Table 2
Genes of *Synechocystis* 6803 differentially affected in the absence of the phytochromes Cph1 and Cph2

Gene ID	Name	Changes (fold) in expression level upon[a] light shift			
		wt	cph1$^-$	cph2$^-$	cph1$^-$/cph2$^-$
Shift from FR to R					
sll1814	secY	4.07 ± 0.01	1.71 ± 0.21	1.57 ± 0.09	1.40 ± 0.09
ssl1911	gifA	13.2 ± 1.22	4.92 ± 0.15	6.09 ± 0.16	1.28 ± 0.05
slr0373		6.58 ± 0.03	1.33 ± 0.09	2.43 ± 0.32	2.46 ± 0.04
slr0376		4.14 ± 0.24	1.10 ± 0.08	1.75 ± 0.15	1.49 ± 0.08
slr0749	chlL	4.10 ± 0.35	0.91 ± 0.01	6.10 ± 0.08	9.80 ± 0.22
slr0772	chlB	8.49 ± 0.02	2.07 ± 0.17	12.5 ± 2.14	12.1 ± 0.21
sll1261	tsf	1.82 ± 0.01	3.56 ± 0.18	2.00 ± 0.01	4.43 ± 0.24
sll1688	thrC	3.63 ± 0.16	12.5 ± 0.23	3.56 ± 0.16	9.42 ± 0.24
slr0076		1.29 ± 0.02	5.00 ± 0.10	2.16 ± 0.81	3.74 ± 0.01
Shift from R to FR					
sll1450	nrtA	5.65 ± 1.98	4.55 ± 0.07	0.85 ± 0.03	1.88 ± 0.21
sll1451	nrtB	7.17 ± 0.54	3.49 ± 0.10	0.97 ± 0.10	1.36 ± 0.05
sll0528		24.0 ± 5.35	39.1 ± 1.70	3.09 ± 0.32	25.4 ± 1.74
sll0549		6.05 ± 0.57	3.94 ± 0.00	1.10 ± 0.12	4.84 ± 0.51
sll1514	hspA	24.2 ± 5.74	36.5 ± 3.68	4.99 ± 0.04	12.5 ± 2.69
slr1687		15.8 ± 4.58	12.5 ± 0.31	1.39 ± 0.07	11.9 ± 0.86
ssl1633	hliC	5.95 ± 0.80	3.38 ± 1.62	0.84 ± 0.04	4.06 ± 0.95
ssl2542	hliA	12.5 ± 2.81	15.1 ± 0.89	0.95 ± 0.01	13.7 ± 0.80
ssl3044		9.90 ± 0.75	10.5 ± 0.43	1.40 ± 0.01	6.62 ± 0.05
slr0270		6.29 ± 0.55	4.31 ± 0.40	1.25 ± 0.04	5.18 ± 0.36
ssr2595	hliB	12.6 ± 2.80	17.2 ± 1.72	1.19 ± 0.00	9.13 ± 0.13
slr1544		6.31 ± 3.33	17.0 ± 0.62	0.96 ± 0.05	8.34 ± 0.10

[a] Values are averages ± standard deviation of two experiments; significant deviations from wild type are highlighted in grey; genes are grouped according to similarity in response.

([28]; Supplementary data Table 1). The relatively low number of genes exhibiting altered transcript levels in *cph1* and *cph2* mutants may also be due to the existence of additional phytochromes since the genome of *Synechocystis* 6803 contains several open reading frames encoding phytochromerelated proteins. In contrast to Cph1 and Cph2, these proteins are supposed to bind or attach a bilin without gaining photochromicity [9]. Nevertheless, they might be able to sense light [4,8].

The *Synechocystis* genes affected by the lack of one or both phytochromes included genes that are involved in chlorophyll biosynthesis (*chlL*, *chlB*), nitrogen metabolism (*gifA*, *nrtA*, *nrtB*), stress adaptation (*hspA*, *hliA*, *hliB*, *hliC*), secretion of proteins (*secY*), translation (*tsf*), threonine biosynthesis (*thrC*) and open reading frames of unknown function. The lack of Cph1 and of both phytochromes altered in first line the R response, whereas the absence of Cph2 changed preferably the FR response of genes. The impact of Cph1 and Cph2 on gene expression at the transcript level was rather low, as we observed for many genes just an quantitative effect of mutation, i.e., mostly a lower decrease or rarely a higher increase in transcript levels compared to the wild-type (Table 2).

To verify the effect of phytochromes on gene activity, we selected *gifA* for a more detailed investigation. *gifA* encodes a regulatory protein, the glutamine synthetase (GS)-inactivation factor IF7 [29], and was one of the genes that responded in a qualitative manner to the lack of functional phytochromes. Whereas the shift from FR to R stimulated *gifA* expression 13-fold in the wild-type, the mRNA level remained constant in the *cph1*$^-$/*cph2*$^-$ double mutant (Table 2). Remarkably prior to the light shift into R at the end of the 90 min FR-acclimation period, the relative amount of *gifA* mRNA was seven times higher in the double mutant compared to the wild-type (the average level of *gifA* transcript was 0.03 in wild-type, 0.08 in *cph1*$^-$, 0.07 in *cph2*$^-$, and 0.21 in *cph1*$^-$/*cph2*$^-$ as calculated from the arrays based on the relative level of the *gifA* transcript to the sum of all transcripts excluding rRNAs and tRNAs). This relatively high level of *gifA* mRNA in *cph1*$^-$/*cph2*$^-$ might have been due to a general high level of *gifA* expression in the double mutant or to its FR-dependent upregulation during the period of acclimation. The results of RNA dot-blot hybridizations on *gifA* expression supported the latter explanation (Supplementary data Fig. 2). Under white light, the level of *gifA* transcripts was similar in wild-type and double mutant. After FR-irradiation for 90 min, however, the level of *gifA* mRNA rose significantly in *cph1*$^-$/*cph2*$^-$. At the same time, the transcripts of *psbA2*, encoding the D1 protein of pho-

tosystem II (PSII) and of *cpcBA*, encoding the β- and α-chain of phycocyanin, accumulated at wild-type levels (Supplementary data Fig. 2). Hence, the FR-induced stimulation of *gifA* expression did not result from a global increase of gene activity in the double mutant, but was specific for *gifA*. Whilst we could verify the dramatic effect of the mutation of both phytochromes on the *gifA* expression in five independent hybridization experiments, the relatively weak quantitative differences observed in microarrays between wild-type and the single mutants with regard to *gifA* transcript levels (Table 2) could not be reproduced (Supplementary data Fig. 2). Consequently, more detailed investigations are needed to reach final conclusions about the impact of phytochromes on gene expression and not all the genes listed in Table 2 may prove to be really under phytochrome control. As the stability of *gifA* mRNA was similar in rifampicin-treated cells of wild-type and double mutant under all light conditions (data not shown), we conclude that upregulation of *gifA* expression during FR acclimation in *cph1*$^-$/*cph2*$^-$ was due to enhanced transcription rather than to altered stability of the transcript. The observed alterations (Table 2) cannot explain the effects of defective Cph1 and/or Cph2 on growth of the mutants under R and FR [14] suggesting that the phytochromes act not only at the level of transcription.

Our data suggest a negative control of *gifA* transcription by Cph1 and Cph2 in the wild-type under FR. In the double mutant, both phytochromes are absent. Consequently the negative control is abolished and FR stimulates transcription. Moreover, the conjoined impact of Cph1 and Cph2 on *gifA* expression implies a certain degree of redundancy of action of both cyanobacterial phytochromes, a mechanism which is known from the activity of phytochromes in plants [30]. FR converts phytochromes into their Pr forms. A biological activity of the Pr forms of Cph1 and Cph2 reported here contrasts with the well investigated Pfr activity of most plant phytochromes. It is, however, in accord with the FR high-irradiance response mediated by phytochrome A in plants [31], the Pr-stimulated autophosphorylation of Cph1 followed by a phosphotransfer to the response regulator Rcp1 [12], and a Pr-dependent regulation of gene expression in other bacteria [32,33].

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.febslet.2005.01.075.

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