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DNA microarray analysis of redox responsive genes in the genome of the cyanobacterium *Synechocystis* sp. PCC 6803

running title: redox responsive genes in *Synechocystis*

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Abstract

The whole genome DNA microarrays were used to evaluate the effect of redox state of photosynthetic electron transport chain on gene expression of *Synechocystis* sp. PCC 6803. Two specific inhibitors of electron transport, 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU) or 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (DBMIB), were added to the cultures and changes in accumulation of transcripts were examined. About 140 genes were highlighted as reproducibly affected by the change of redox state of photosynthetic electron transport chain. It was shown that some stress-responsive genes but not photosynthetic genes were under the control of the redox state of the plastoquinone pool in *Synechocystis* sp. PCC 6803.

Photosynthetic organisms must cope with changes in their light environment by various acclimation responses. In order to acclimate to light regime, organisms need sensors to monitor changing light regime, signal transduction systems and output mechanisms for rearrangement of their photosynthetic machinery. The sensing mechanism of changing light conditions and subsequent signal transduction are poorly understood (14). However, it has recently become clear that the light-induced electron transport plays a very important role in both transcriptional and post-transcriptional regulations in various photosynthetic organisms. In green algae and higher plants, transcription (9, 17, 25), mRNA stability (2), splicing (8), translation (7, 18) and protein phosphorylation (29) were reported to be regulated by the redox state of photosynthetic electron transport chain. In cyanobacteria, main targets for redox regulation are transcription and stability of mRNA. mRNA levels of several photosynthesis-related genes such as *psbA* encoding the reaction center D1 polypeptide of photosystem II (PSII), *psaE* coding for a subunit of photosystem I (PSI), *cpcBA* encoding β

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and α subunits of phycocyanin and *rbcLS* encoding subunits of RuBP carboxylase were shown to be affected by the redox state of photosynthetic electron transport chain (3, 4, 6, 21). Also thioredoxin gene (23), genes for fatty acid desaturases (19), some nitrogen-regulated genes (5, 10, 28), and heat shock genes (12) were reported to be under the control of photosynthetic electron transport. Then, how many genes in the whole genome are regulated by photosynthetic electron transport? To answer this question, we investigated the entire profile of accumulation of transcripts upon the change in redox state of intersystem electron transfer components in a cyanobacterium *Synechocystis* sp. PCC 6803 with the whole-genome DNA microarray. Using the microarray, we recently examined the temporal program of gene expression during the acclimation from low- to high-light intensity (15).

Effects of DCMU and DBMIB on the redox state of photosynthetic electron transport chain. In the current study, we employed two different electron transport inhibitors, 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU) and 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (DBMIB) to modulate the redox state of photosynthetic electron transport chain. DCMU blocks transfer of electrons from PSII to the plastoquinone (PQ) pool and DBMIB prevents the oxidation of PQ by the cytochrome b_6/f complex (31). The two inhibitors have opposite effects on the net redox state of the PQ pool: more oxidized in the presence of DCMU and more reduced in the presence of DBMIB. By comparing the effects of two inhibitors, we aimed at the categorization of ORFs of *Synechocystis* sp. PCC 6803 according to the patterns of redox regulation. First, the specificity of DBMIB was checked because DBMIB is also known to inhibit the electron transfer from Q_A to Q_B at higher concentrations. In the presence of inhibitors, we determined the fluorescence decay kinetics in millisecond range that represent the electron transfer from Q_A to Q_B. The cyanobacterial cells were dark-adapted for five minutes, and then a single-turnover flash (XST, Heinz Waltz, Germany) was applied. The fluorescence level was

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monitored by PAM fluorometer (PAM 101/103, Heinz Waltz, Germany) using Auto 100 kHz mode with high sensitivity measurement unit (ED-101US, Heinz Waltz, Germany). The triggering of the single-turnover flash and the sampling of the fluorescence signal were conducted using an A/D converter (AnalogPro II, Canopus, Japan) and a laboratory-made software with sampling frequency of 25 kHz and 14 bit resolution. As shown in Fig. 1, addition of 10 μM DCMU totally inhibited the decay of fluorescence, while 10 μM DBMIB only slightly affected the decay kinetics. When the concentration of DBMIB was raised to 100 μM, the rapid phase of the fluorescence decay kinetics in millisecond range was largely eliminated (data not shown). Thus, 10 μM DBMIB showed only a slight effect on the electron transfer from Q_A to Q_B, if any, under our experimental conditions. Since 10 μM DBMIB totally blocked the electron transfer from PQ to b_6/f complex judging from the complete inhibition in the overall electron transfer (not shown), the PQ pool should be highly reduced in the presence of DBMIB at this concentration. Although DBMIB was reported to be relatively unstable (4), we found that 10 μM DBMIB totally blocked oxygen evolution of cultures even after 2 h of incubation in our experimental conditions. At that time, cells were still alive since oxygen evolution was partially restored after the removal of DBMIB.

Growth condition, RNA isolation and DNA microarray analyses. A glucose-tolerant wild-type strain of *Synechocystis* sp. PCC 6803 was grown at 20 μmol photons $m^{-2}s^{-1}$ to a cell density of A_{730} = 0.5 and then incubated with or without inhibitors. Isolation of total RNA from cultures and successive DNA microarray analysis were performed as previously described (15). For image acquisition and analysis, two scanning and quantification systems were used in this study. Image acquisition by 418 Array Scanner (Affymetrix, Santa Clara, Calif.) and data analysis by Imagene version 3.0 software (BioDiscovery, Los Angeles, Calif.) were performed as previously described (15). Image acquisition by ScanArray 4000 (GSI Lumonics, Watertown, Mass.) was performed using

auto-balance/auto-range feature. By this feature, the sensitivity of the instrument can be automatically adjusted by changing the laser power and PMT gain settings so that the signal is within 90% of maximum to prevent saturation. The raw data obtained by ScanArray 4000 were analyzed by QuantArray version 2.0 software (GSI Lumonics). The fluorescence intensity of each spot in both Cy3 and Cy5 images was quantified, and fluorescence levels of the local background were subtracted. Normalization of Cy3 and Cy5 images was performed by adjusting the total signal intensities of the two images ("global normalization"). After normalization, the ratio of transcript level of each gene with electron transport inhibitors to that without inhibitors was calculated to determine the induction ratio by inhibitors. All induction ratios are shown as averages of three independent and each duplicate experiments (n =6). The raw data are available on Internet (http://www.genome.ad.jp/kegg/).

The effect of the addition of inhibitors on the overall profile of accumulation of transcripts. At first, accumulation profiles of transcripts were compared between 15 min and 1 h after the addition of inhibitors. Many genes were affected similarly at both time points. However, extent of induction or repression was much higher at 1 h than at 15 min (not shown). Modulation of gene expression in response to the new redox state seemed to be still in progress at 15 min, whereas prominent induction or repression was achieved in many genes at 1 h. Therefore, we chose 1 h as the incubation time for this study to obtain clear and reproducible results of DNA microarrays. Figure 2 shows the effect of the addition of inhibitors on the transcript levels of the whole set of genes in *Synechocystis* after 1 h of incubation. The majority of the genes were aligned along the diagonal line from the lower left to the upper right, which means they are similarly affected by both inhibitors. However, most of them did not seem to be redox responsive since they located inside of two lines representing induction by two-fold and repression to the half. Only a few genes were located on the diagonal line from the lower right to the upper left, indicating that it is rare case to be

oppositely affected by two inhibitors. Genes, whose expression was significantly affected by the addition of inhibitors, were classified into four groups as shown by the shaded area in Fig. 2: group I, induction by DCMU was more than three times larger than that by DBMIB; group II, induction by DBMIB was more than three times larger than that by DCMU; group III, induced by both inhibitors more than two fold; and group IV, repressed by both inhibitors to less than half. Representative genes belonging to each group are listed in Table 1.

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Genes differently affected by the two inhibitors. Genes categorized to group I and II could be regulated by the redox state of the PQ pool since DCMU and DBMIB showed quite different effects on the expression of these genes. It is of note that many genes were strongly induced by DBMIB (group II) but not by DCMU (group I). Heat shock genes such as *groESL*, *groEL-2*, *dnaK*, *dnaJ*, *htpG*, *clpB* and *hspA*, high-light inducible proteins such as *hliA* and *hliB* and nitrate assimilation-related genes such as *nrtABCD* and *nirA* were induced by the addition of DBMIB. The reduced state of the PQ pool might be important as the signal for accumulation of transcripts. At any rate, no genes related to photosynthesis were shown to be regulated by the redox state of the PQ pool in this study. Even when inhibitors were added to the high-light grown cultures, photosynthesis-related genes were not categorized to group I or II (not shown). This observation is quite unexpected since the transcriptional regulation of photosynthesis-related genes by the redox state of the PQ pool is well-established in green algae and higher plants. For example, transcription of *cab* genes in *Dunaliella tertiolecta* (9), *psaAB* operon in mustard (25), *psaB* gene in pea (32) and *petE* gene in tobacco (26) were shown to be oppositely affected by addition of DCMU or DBMIB, indicating the coupling between the redox state of the PQ pool and the rate of transcription. The regulation system for the expression of photosynthetic genes seems to be totally different between these organisms and cyanobacteria.

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Genes similarly affected by the two inhibitors. It is assumed that the redox state

of the components upstream of Q_A or downstream of the PQ pool responds similarly to DCMU and DBMIB. Thus, the candidates for the redox sensor responsible for group III and IV genes could be electron transfer components such as PSII, cytochrome b_6/f complex and ferredoxin/thioredoxin system. In group III genes that are induced by both inhibitors, we could observe genes encoding proteases, house-keeping enzymes and some PSII-related components such as *psbA* and *psb28*. On the other hand, genes encoding ribosomal proteins, phycocyanin, ATP synthase and enzymes for biosynthesis of photosynthetic pigments were repressed by both inhibitors and categorized to group IV. Although the extent of repression by both inhibitors was not high enough to be listed in Table 1, some genes reported as redox-responsive so far, such as *trxA* encoding thioredoxin (23), *desA*,*B*, and *D* encoding fatty acid desaturases (19) and nitrogen-regulated genes (5, 10, 28), could be also categorized into group IV. It is of interest that genes activated by NtcA, such as *glnA*, *glnB*, *ntcA*, *sigE* and *amt1* (13), were all down-regulated (not shown) and genes repressed by NtcA, such as *gifA* and *gifB* (11), were up-regulated (Table 1) by both inhibitors. Although nitrate assimilation-related genes such as *nrtABCD* and *nirA* also have NtcA-binding motif, their response was quite different from that of other NtcA-regulated genes. In addition to NtcA, NtcB contributes to the regulation of the nitrate assimilation-related genes (1), which may confer special characteristics on the expression profiles of these genes. As for genes encoding subunits of PSI, we observed only slight down-regulation by addition of inhibitors (not shown) although several groups reported that PSI genes were largely repressed by inhibitors (3, 20). Some differences in experimental conditions, such as light intensity and/or quality, might be the cause of this discrepancy. We think that PSI genes are redox-responsive at least under high-light conditions, since the marked down-regulation of PSI genes under high light (22) was abolished by the addition of inhibitors (not shown). In summary, genes encoding subunits of phycobilisome, photosystems, ATP synthase, ribosomes and various enzymes

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were affected similarly by both inhibitors and therefore supposed to be under the control of the redox state of electron transport components other than the PQ pool. The PQ pool might be used for the sensor of rapid induction of stress-responsible genes, whereas other redox components of the photosynthetic electron transport chain might be important for slower acclimation responses. Q_0 site of cytochrome b_6/f complex is one of the possible candidates for the redox sensor for the regulation of photosystem genes as suggested by Bissati and Kirilovsky (6).

Relationship between redox- and stress- responsibility. To date, gene expression in response to high-light (15), low-temperature (30), high-salinity and high-osmolality conditions (16) has been investigated using DNA microarrays. We noticed that many redox-responsive genes in Table 1 were also listed in these studies. As shown in Table 1, many genes in group II and III were induced (indicated by "I") and those in group I and IV were repressed (indicated by "R") under various stress conditions. On the other hand, among the genes listed as stress-responsive under high-light, low-temperature, high-salinity and high-osmolality conditions, 24, 24, 43 and 40% of them were listed as redox-responsive in this study, respectively. From these observations, we could conclude that transcript levels of many genes involved in acclimation responses to environmental stresses could be regulated by the redox state of photosynthetic electron transport chain. The sigma-70 factors listed in Table 1, *sigB* (sll0306) and *sigD* (sll2012), may work on the induction of these genes under stress conditions.

Characterization of multigene families according to the redox responsibility. There are many genes present in multiple copies in the genome of *Synechocystis* sp. PCC 6803 and the characteristics of each copy was obscure in most cases. However, we found that a copy of some genes could be clearly distinguished from the other copies by their redox responsibility. For example, among *ndh* genes encoding subunits of NAD(P)H dehydrogenase,

only one copy, *ndhD2*, was largely induced by DBMIB and categorized in group II, indicating that this copy has a special role. On the other hand, copies involved in CO_2 uptake such as *ndhD3* and *ndhF3* (24) were repressed by both inhibitors (not shown). Most of the other *ndh* genes did not respond to the addition of inhibitors. Another example is *clpP4*. There are four copies for catalytic subunit of Clp protease in *Synechocystis* sp. PCC 6803. Among them, the putative product of only *clpP4* gene lacks the amino acid residues required for the proteolytic activity and is therefore unlikely to function as a true ClpP protease (27). The fact that this gene but not the other *clpP* genes responded to the addition of the inhibitors implies the physiological significance of *clpP4*. It is also noteworthy that *psb28*, slr1739, is one of a few photosystem-related genes listed in Table 1. This gene is homologous to *psbW*, sll1398, encoding the small subunit of PSII. Although we do not know whether this gene product is also the component of PSII, its redox-responsibility suggests that it has its own physiological role. Information on redox regulation of these genes could be important clues for the elucidation of physiological function of each gene product.

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Conclusion. In addition to several genes reported to be redox-responsive so far (3, 5, 10, 12, 19, 23, 28), more than 100 genes involved in various cellular functions in *Synechocystis* sp. PCC 6803 were shown to be affected by the redox state of the photosynthetic electron transport chain. The photosynthetic electron transport chain seems to play an important role in monitoring the environmental changes and in acclimating to them.

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Figure legends

Fig. 1

Decay kinetics of fluorescence in millisecond range after excitation with single turn-over flash. The data are normalized with Fv. Measurement were performed (A) without inhibitors, (B) in the presence of 10 μM DCMU and (C) in the presence of 10 μM DBMIB.

Fig. 2

Induction or repression ratio of each gene by addition of inhibitors. The horizontal axis indicates the induction ratio by addition of DCMU and the vertical axis indicates that of DBMIB. Induction by twofold and repression to a half are shown by solid lines. More than threefold difference in the effect between two inhibitors is shown by dashed line. Four groups categorized by effects of inhibitors are indicated by shaded areas and numbers.

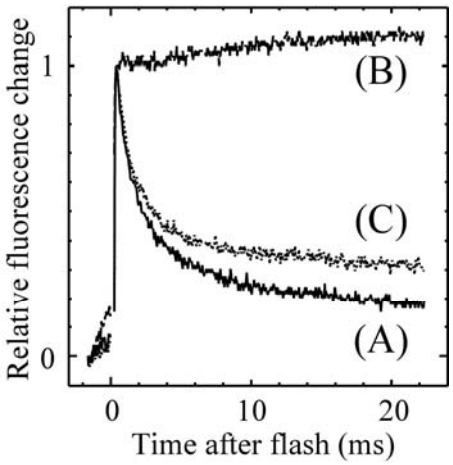
Relative fluorescence change
1
0
(B)
(C)
(A)
0
10
20
Time after flash (ms)

Fig .1

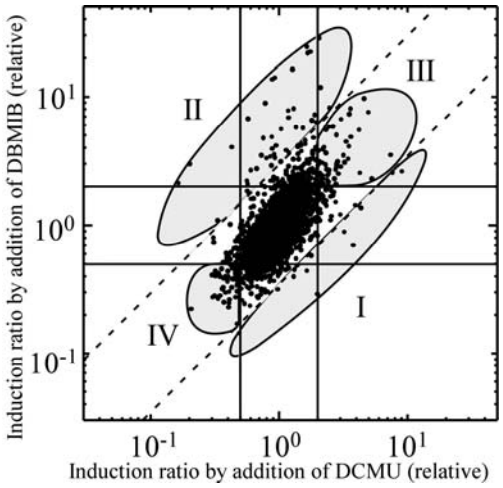
Induction ratio by addition of DBMIB (relative)
Induction ratio by addition of DCMU (relative)
II
III
IV
I
10^1
10^0
10^{-1}
10^{-1}
10^0
10^1

Fig. 2

TABLE 1. The list of genes whose expression was significantly affected by the addition of inhibitors of photosynthetic electron transport.

ORF number and description	Effects of addition of inhibitors		Effects of stress conditions			
	DCMU[a]	DBMIB[b]	HL[c]	LT[d]	HS[e]	HO[f]
Group I: Induction by DCMU was more than three times larger than that by DBMIB.						
sll1647	3.23 ± 2.36	0.89 ± 0.35				
sll1677 spore maturation protein B	0.98 ± 0.27	0.31 ± 0.05				
sll1864 chloride channel protein	3.80 ± 0.46	0.70 ± 0.10			I[g]	
slr0358	1.30 ± 0.20	0.40 ± 0.14				
slr0373	9.13 ± 4.74	2.61 ± 0.57	R[h]			
slr0376	4.36 ± 1.25	1.35 ± 0.25	R			
slr0772 *chlB* protochlorophyllide reductase subunit	0.49 ± 0.04	0.14 ± 0.07	R			
slr0888	1.79 ± 0.30	0.58 ± 0.06	R			
slr1634	1.99 ± 0.28	0.29 ± 0.05	R			R
slr1908 probable porin	1.66 ± 0.22	0.44 ± 0.17				
ssl1911 *gifA* glutamine synthetase inactivating factor IF7	7.70 ± 1.70	2.55 ± 0.71				
Group II: Induction by DBMIB was more than three times larger than that by DCMU.						
sll0170 *dnaK2*	0.89 ± 0.19	4.83 ± 0.99	I		I	I
sll0306 *sigB*, *rpoD*	1.90 ± 0.36	7.20 ± 3.67			I	I
sll0416 *groEL-2*	0.33 ± 0.15	1.09 ± 0.52	I		I	I
sll0430 *htpG*	0.43 ± 0.23	4.02 ± 0.39	I			I
sll0528	1.76 ± 0.30	22.21 ± 7.85	I		I	I
sll0549	1.81 ± 0.32	6.54 ± 2.49				
sll0786	0.58 ± 0.44	1.78 ± 1.85				
sll0788	2.06 ± 0.35	28.42 ± 9.51			I	I
sll0789 *copR*, *rre34*	1.66 ± 0.34	24.34 ± 13.32			I	I
sll0790 *hik31*	0.93 ± 0.22	13.95 ± 5.25				
sll0846	1.05 ± 0.38	5.55 ± 2.37	I		I	I
sll1159	0.73 ± 0.17	3.46 ± 1.60				
sll1167 *pbp* penicillin-binding protein 4	0.89 ± 0.37	3.92 ± 2.19			I	
sll1450 *nrtA* nitrate transport protein NrtA	0.67 ± 0.24	2.74 ± 0.95				
sll1451 *nrtB* nitrate transport protein NrtB	0.93 ± 0.35	3.33 ± 0.87				
sll1452 *nrtC* nitrate transport protein NrtC	1.20 ± 0.42	3.61 ± 1.26				
sll1453 *nrtD* nitrate transport protein NrtD	0.92 ± 0.42	3.55 ± 0.63			R	
sll1514 *hspA*	1.28 ± 0.31	16.14 ± 4.74	I		I	I
sll1620	0.80 ± 0.16	3.30 ± 0.45				
sll1621 membrane protein	0.78 ± 0.21	8.84 ± 1.95		R	I	
slr0074 *ycf24* ABC transporter subunit	0.88 ± 0.18	2.90 ± 0.42				
slr0075 *ycf16* ABC transporter subunit	0.56 ± 0.09	2.21 ± 0.13				
slr0076	0.58 ± 0.04	2.09 ± 0.23				
slr0093 *dnaJ*	1.36 ± 0.33	5.76 ± 2.83			I	I
slr0095 O-methyltransferase	1.14 ± 0.19	3.80 ± 1.97			I	
slr0272	1.55 ± 0.68	5.66 ± 2.86	R			
slr0898 *nirA* ferredoxin--nitrite reductase	0.57 ± 0.25	1.83 ± 0.28				
slr1285 *hik34*	0.58 ± 0.09	6.72 ± 4.02				
slr1291 *ndhD2*	1.68 ± 0.32	21.70 ± 11.16	I	I		
slr1413	0.76 ± 0.23	4.74 ± 2.33				
slr1544	2.48 ± 0.80	8.16 ± 4.76	I	I	I	I
slr1603	2.12 ± 0.44	11.72 ± 6.94			I	I
slr1641 *clpB1*	1.18 ± 0.27	7.75 ± 2.31	I		I	I
slr1674	1.30 ± 0.13	8.05 ± 1.77	I		I	I
slr1675 *hypA1*	0.89 ± 0.39	13.59 ± 6.55	I		I	I
slr1738	0.95 ± 0.12	4.15 ± 1.28			I	
slr1963 water-soluble carotenoid protein	0.61 ± 0.11	2.66 ± 0.38	I		I	I
slr2075 *groES*	0.20 ± 0.06	2.99 ± 0.83	I			
slr2076 *groEL*	0.17 ± 0.06	2.11 ± 0.51	I			
ssl2542 *hliA*, *scpC*	1.19 ± 0.40	6.84 ± 6.70			I	I
ssl2971	0.86 ± 0.07	3.41 ± 1.61			I	I
ssr2595 *hliB*, *scpD*	1.98 ± 0.73	6.28 ± 6.28			I	I
Group III: Induced by both inhibitors more than twofold.						
sll0020 *clpC* ATP-dependent Clp protease regulatory subunit	2.01 ± 0.30	2.50 ± 0.24				
sll0141 putative HlyD family secretion protein	2.89 ± 0.48	3.10 ± 0.25				
sll0297	3.04 ± 0.50	2.84 ± 1.31				
sll0749	2.02 ± 0.27	3.80 ± 1.39				
sll0843	2.47 ± 0.48	5.79 ± 1.16				
sll0891 *citH*, *ldh* malate dehydrogenase	3.20 ± 0.52	3.48 ± 0.49				
sll0939	4.63 ± 1.01	9.64 ± 4.87			I	I
sll0992 esterase	2.02 ± 0.70	3.77 ± 1.47				
sll1086	3.62 ± 1.14	8.01 ± 2.73			I	
sll1201	2.38 ± 0.15	2.23 ± 0.75				
sll1268	2.31 ± 0.42	2.93 ± 0.56				
sll1432 *hypB*	2.92 ± 0.79	2.49 ± 0.44				
sll1483 transforming growth factor induced protein	4.99 ± 2.14	7.55 ± 3.58	I		I	I
sll1515 *gifB* glutamine synthetase inactivating factor IF17	7.44 ± 1.67	3.50 ± 0.68			I	
sll1774	2.80 ± 0.17	2.08 ± 0.85				
sll1867 *psbA3* photosystem II D1 protein	2.17 ± 0.52	2.42 ± 0.27	I			
sll2012 *sigD*, *rpoD*	2.82 ± 0.56	5.00 ± 2.40	I	I		
slr0164 *clpP4*, *clpR* Clp protease proteolytic subunit	2.07 ± 0.14	3.12 ± 1.04				
slr0211	2.84 ± 0.31	2.94 ± 0.15				
slr0374 cell division cycle protein	5.33 ± 1.64	2.29 ± 0.24	R			

slr0397		2.12	± 0.37	2.37	± 0.27				
slr0451	*ski2* antiviral protein	2.02	± 0.59	2.90	± 0.36				
slr0551		2.83	± 0.45	2.97	± 1.08		I		
slr0581		2.73	± 0.30	2.27	± 0.66			I	I
slr0599	eukaryotic protein kinase	2.40	± 0.18	2.42	± 0.89				
slr0600	putative thioredoxin reductase	2.21	± 0.19	3.94	± 0.68				
slr0839	*hemH*, *scpA* ferrochelatase	2.18	± 0.33	3.04	± 0.40				
slr0942	aldehyde reductase	2.20	± 0.28	2.30	± 0.28				
slr0967		3.55	± 0.92	7.30	± 0.85			I	
slr1113	ABC transporter	2.77	± 0.30	2.70	± 0.79				
slr1114		3.26	± 0.77	3.05	± 0.63				
slr1127		3.35	± 0.31	4.43	± 0.56				
slr1128	"erthyrocyte band 7 integral membrane protein"	4.21	± 0.82	5.49	± 1.41		R		
slr1129	*rne* ribonuclease E	2.33	± 0.21	3.61	± 0.79				
slr1253		2.83	± 0.51	2.86	± 0.54				
slr1259		4.29	± 0.80	2.53	± 0.68			I	
slr1260		3.47	± 0.24	3.27	± 0.63				
slr1262		3.64	± 0.20	4.82	± 0.45				
slr1604	*ftsH* cell division protein FtsH	2.07	± 0.17	3.01	± 0.33	I		I	
slr1687		3.07	± 1.45	6.67	± 3.21			I	I
slr1712		2.47	± 0.10	2.67	± 0.51				
slr1739	*psb28* photosystem II 13 kDa protein homolog	2.64	± 0.93	5.82	± 1.68				
slr1751	*prc* or *tsp* carboxyl-terminal protease	2.45	± 0.15	2.50	± 0.58			I	
slr1830	*phbC* poly(3-hydroxyalkanoate) synthase	2.36	± 0.27	2.69	± 0.97				
ssl2501		3.53	± 0.73	4.08	± 1.57				
ssl3769		2.33	± 0.50	5.47	± 1.15				
Group IV: Repressed by both inhibitors to less than half.									
sll0017	*hemL*, *gsa* glutamate-1-semialdehyde 2,1- aminomutase	0.49	± 0.16	0.37	± 0.12				
sll0026	*ndhF* NADH dehydrogenase subunit 5	0.48	± 0.03	0.39	± 0.05				
sll0421	*purB* adenylosuccinate lyase	0.50	± 0.05	0.45	± 0.02				
sll1077	*speB* agmatine ureohydrolase	0.41	± 0.09	0.30	± 0.06				
sll1091	*chlP* geranylgeranyl hydrogenase	0.47	± 0.02	0.34	± 0.23	R	R	R	R
sll1185	*hemF* coproporphyrinogen III oxidase	0.32	± 0.03	0.28	± 0.16	R	R		
sll1212	*rfbD* GDP-D-mannose dehydratase	0.40	± 0.05	0.44	± 0.22				
sll1305		0.44	± 0.03	0.46	± 0.26	R	R	R	R
sll1323	*atpG* ATP synthase subunit b'	0.35	± 0.06	0.28	± 0.03				
sll1324	*atpF* ATP synthase subunit b	0.30	± 0.04	0.41	± 0.18				
sll1325	*atpD* ATP synthase d subunit	0.32	± 0.05	0.40	± 0.15				
sll1326	*atpA* ATP synthase a subunit	0.42	± 0.06	0.50	± 0.17				
sll1471	*cpcG2* phycobilisome rod-core linker polypeptide	0.21	± 0.23	0.22	± 0.25				
sll1526		0.34	± 0.07	0.27	± 0.08				
sll1530		0.36	± 0.08	0.46	± 0.31				
sll1531		0.37	± 0.12	0.32	± 0.08				
sll1550	probable porin	0.46	± 0.12	0.27	± 0.12		R		
sll1577	*cpcB* phycocyanin b subunit	0.32	± 0.09	0.44	± 0.40	R		R	R
sll1579	*cpcC* phycocyanin associated linker protein	0.41	± 0.12	0.26	± 0.10	R	R		
sll1580	*cpcC* phycocyanin associated linker protein	0.37	± 0.05	0.39	± 0.28	R	R	R	R
sll1799	*rpl3* 50S ribosomal protein L3	0.35	± 0.02	0.40	± 0.10	I	I	I	
sll1800	*rpl4* 50S ribosomal protein L4	0.38	± 0.07	0.30	± 0.05	I	I		
sll1801	*rpl23* 50S ribosomal protein L23	0.38	± 0.04	0.40	± 0.04	I	I		
sll1802	*rpl2* 50S ribosomal protein L2	0.34	± 0.05	0.42	± 0.10	I			
sll1804	*rps3* 30S ribosomal protein S3	0.40	± 0.06	0.36	± 0.07	I			
slr0468		0.44	± 0.15	0.38	± 0.11				
slr0676	*cysC* adenylylsulfate 3'-phosphotransferase	0.40	± 0.05	0.40	± 0.13				
slr0750	*chlN* protochlorophillide reductase subunit	0.47	± 0.22	0.17	± 0.07	R			
slr0909		0.37	± 0.07	0.38	± 0.16				
slr1056		0.49	± 0.28	0.38	± 0.07				
slr1064	*rfbU* or *mtfA* mannosyltransferase B	0.45	± 0.10	0.38	± 0.07				
slr1161		0.41	± 0.05	0.23	± 0.16	R			
slr1162		0.31	± 0.09	0.27	± 0.24				
slr1329	*atpB* ATP synthase b subunit	0.48	± 0.08	0.36	± 0.03				
slr1396		0.38	± 0.06	0.25	± 0.06				
slr1618		0.44	± 0.05	0.49	± 0.09				
slr1619		0.46	± 0.04	0.41	± 0.05				
slr1854		0.37	± 0.05	0.32	± 0.14	R	R	R	R
slr1855		0.41	± 0.05	0.25	± 0.02	R	R	R	R
slr1859	anti-sigma f factor antagonist	0.45	± 0.08	0.38	± 0.08			R	R
slr1860	*icfG*	0.46	± 0.13	0.43	± 0.06				

[a] Changes in expression levels by addition of DCMU (fold). Values are averages±SD of three independent and each duplicate experiment (n = 6).

[b] Changes in expression levels by addition of DBMIB (fold). Values are averages±SD of three independent and each duplicate experiment (n = 6).

[c] Effects of high light according to Hihara et al. (2001).

[d] Effects of low temperature according to Suzuki et al. (2001).

[e] Effects of high salinity according to Kanesaki et al. (2002).

[f] Effects of high osmolality according to Kanesaki et al. (2002).

[g] Induced under the stress condition indicated above.

[h] Repressed under the stress condition indicated above.