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Photoinhibition and Recovery of *Anacystis nidulans* Adapted in Blue-Green Light

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Photoinhibition and its recovery of spectrally adapted *Anacystis nidulans* were studied. Phycocyanin and Chl content and phycocyanin/Chl ratio were increased in cells grown under blue-green light compared with those grown in white light. Photosynthetic activities of white light and blue-green light grown cells were reduced by 50% after 15 min and 10 min of photoinhibitory light treatment (1.2 mmol·m^{-2}·s^{-1}), respectively, largely due to the decline of PSII activities. However, their activities were recovered fully after 30 min incubation under weak light. Treatment of rifampicin and chloramphenicol magnified the photoinhibitory effects and suppressed the recovery with disappearance of susceptibility to photoinhibition between two cells. NaF and phenylmethylsuflonyl fluoride also accelerated the photoinhibition and delayed the recovery process, indicating no significant differences in phosphorylation, dephosphorylation and protease activity between two cells. Therefore, it is suggested that the increased sensitivity of blue-green adapted cells might be attributed to the decline of protein synthesis, and phosphorylation-dephosphorylation of protein and protease activity might be involved in the recovery process.

Keywords: *Anacystis nidulans*, spectral adaptation, photoinhibition, recovery, protein synthesis

Light is the driving force of photosynthesis and therefore an absolute prerequisite for the autotrophic growth of photosynthetic organisms, but it can also be harmful to the photosynthetic apparatus (Aro *et al.*, 1993). The term photoinhibition is used to describe the phenomenon of reduction of capacity for photosynthesis induced by exposure of photosynthetic organisms, structures, or organelles to light (Powles, 1984). The photosynthetic organisms have to manage the task of avoiding photodamages at high light as well as maintaining sufficient excitation power under limiting light conditions (Aro *et al.*, 1993).

When photoinhibition is induced by an excessive photon fluence rate (PFR), the main target for the light stress is PSII (Powles, 1984). Electron transport through PSII is inhibited, and reaction center subunits are damaged, in particular D1 protein (Kyle, 1987). Illumination results in the activation of thylakoid protein kinase, and therefore phosphorylation of the D1 proteins in appressed thylakoid membranes occurs (Ikeuchi *et al.*, 1987). Conformational changes in the damaged D1 protein exposes the cleavage site for the protease (Aro *et al.*, 1993). However, phosphorylated D1 protein is a poor substrate for the protease (Salter *et al.*, 1992), and dephosphorylation is probably required prior to the primary proteolytic cleavage. D1 protein depletion should be considered as the first phase in the repair cycle, and after which new copies of the protein should be synthesized and inserted in PSII.

Lönneborg *et al.* (1988) suggested that the degree of net photoinhibition was determined by a balance between the photoinhibitory process and the operation of a repair mechanism. They showed that the capacity of the repair mechanism significantly determined the difference in the susceptibility of photosynthesis to photoinhibition of high and low light

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grown *Anacystis nidulans*. The light dependent patterns of inhibition of photosynthesis were very similar in the presence of translation inhibitor streptomycin for high and low light grown *A. nidulans* cells (Samuelsson *et al.*, 1987).

Higher plants also differ in their susceptibility to photoinhibition, and this is dependent on their capacity to repair photodamaged reaction centers during illumination (Tyystjärvi *et al.*, 1991). The high susceptibility of low-light grown pumpkin (*Cucurbita pepo* L.) plants to photoinhibition is mainly due to a poor capacity of the plants to repair photodamaged PSII centers. The repair cycle of PSII functions more slowly in low-light grown plants as compared with high-light grown plants, and it was suggested that this is because of a poor capacity of the low-light grown leaves to degrade photodamaged D1 protein (Tyystjärvi *et al.*, 1992). These reports suggest that, in different light conditions, the photosynthetic apparatus differs in their susceptibility to photoinhibition since they have different capacities to repair photodamaged PSII.

In cyanobacteria, when the spectral adaptation occurs in response to the changes in spectral distribution, quantitative changes in photosynthetic apparatuses are induced, especially in phycobilin pigment systems. Jones and Myers (1965) reported spectrally induced pigment variation in *A. nidulans*. Unicellular cyanobacteria are especially useful organisms for studying photoinhibition and other stress-induced effects on photosynthesis in whole cells because a homogeneous population can be treated under well-defined conditions, and because the relatively simple structure and small size of the cells facilitate quantitative observations of photosynthesis *in vivo* (Wünschmann and Brand, 1992).

In the present paper, we investigated the effect of the chromatic adaptation of *A. nidulans* to white and blue-green light on the sensitivity to photoinhibition, and attempted to elucidate the mechanism of photoinhibition and recovery.

MATERIALS AND METHODS

Culture conditions

The cyanobacterium *A. nidulans* (Carolina Biological Supply, USA) was grown in batch cultures using modified Allen medium (1968). The temperature was kept at 39± 1℃ and the cultures were exposed to continuous PFR of 20 $\mu mol \cdot m^{-2} \cdot s^{-1}$. White light was provided by fluorescent lamps and blue-green light was given by a couple of fluorescent lamps covered with cellulose acetate sheets (Chapman, 1988). Cells in the logarithmic phase of growth were used for experiments. Cell densities and growth were determined by the absorbance at 750 nm (Lönneborg *et al.*, 1985). Cells were harvested by centrifuging twice at 6000 *g* for 10 min, then the pellet was resuspended in the medium and placed in a water bath at 39± 1℃.

Phycocyanin and Chl a determination

The harvested cells were frozen in liquid nitrogen and ground by glass rod. After adding 0.05 M potassium phosphate buffer (pH 7.2), the extract was centrifuged at 12,000 *g* for 30 min at 4℃. The supernatant was recovered, and the absorbances at 620 nm and 650 nm were read. The concentration of the phycocyanin (PC) and allophycocyanin (APC) was calculated from Chapman (1988). The remaining pellet was ground in dimethyl sulfoxide (DMSO), and was incubated at 65℃ for 15 min. Chl a was determined according to Evans (1988).

Photoinhibition and recovery treatment

The photoinhibitory treatment was done in a glass tube immersed in a water bath kept at 39± 1℃. Photoinhibitory light was supplied by a tungsten halogen lamp (Star, 1000 W, Itami Protex, Japan). The Chl concentration was approximately 2.0 μg/mL. Chlorophyll a and phycocyanin concentrations were spectrophotometerically measured and calculated using the extinction coefficients of Myers *et al.* (1978). The recovery treatment was done in dim light with a PFR of 2 $\mu mol \cdot m^{-2} \cdot s^{-1}$ or in the dark by transferring cells to a test tube covered with aluminum foil after partial photoinhibition (about 50-60 %).

Photosynthetic electron transport measurements

Photosynthetic electron transport was measured

using a Clark type O_2 electrode (Hansatech, Norford, UK). Light source was a projector tungsten lamp (300 W). After various photoinhibition and recovery treatment, 1 mL of algal sample was taken. For the measurement of whole cell photosynthetic O_2 exchange, 50 mM $NaHCO_3$ was added to the solution to avoid CO_2 deficiency. For the measurement of electron transport, cells were first mixed with sucrose stock solution to make a final concentration of 5% sucrose, and frozen in liquid nitrogen, then freeze-dried for 28 h at −86℃ (Gerhardt and Trebst, 1965). Freeze-dried cells were resuspended in reaction mixture containing 40 mM phosphate buffer (pH 7.6), 0.1 mM $MgCl_2$, 5 mM NH_4Cl, 1 mM NaCl, 0.6 mM NaN_3, and 5% sucrose to have a Chl content of 10 μg/mL. The PSI electron transport measurement was done by adding 0.01 mM 3-(3,4-dichlorophenyl)-1, 1-dimethylurea (DCMU), 0.3 mM 2,6-dichlorophenolindophenol (DCPIP), 32 mM sodium ascorbate, and 0.12 mM methyl viologen (MV) and the PSII electron transport measurement was done by adding 0.25 mM *p*-phenylenediamine (*p*-PD) and 0.5 mM ferrycyanide (FeCy). For the whole chain electron transport measurement, 0.12 mM MV was added (Tyystjärvi *et al.*, 1989).

Chemical inhibitors

When used, rifampicin and chloramphenicol (CAP) were added to a final concentration of 250 μg/mL and 60 μM, respectively. The protein kinase inhibitor 5′-p-fluorosulfonyl-benzoyladenosine (FSBA) and protease inhibitor phenylmethyl-sulfonyl fluoride (PMSF) were each added to make a final concentration of 0.5 mM, and protein phosphatase inhibitor NaF, 10 mM. For the photoinhibitory treatment, the inhibitors were added 20 min before cells were illuminated, and for the recovery treatment, the inhibitors were added at the same time when the lamp was turned off.

RESULTS AND DISCUSSION

Effect of blue-green light on pigment contents

To check if white light (WH) and blue-green light (BG) grown cells were spectrally adapted, the absorbance spectra of the cells were examined. The ratio of A_{625} to A_{678} was 1.03 for WH grown cells and 1.36 for BG grown cells, showing a 32% increase in the ratio for BG grown cells (Fig. 1). Because the higher A_{625}/A_{678} in cells grown under BG reflects higher phycobilin (phycocyanin) to Chl content (Sanders *et al.*, 1989), we determined the contents of PC and APC relative to Chl content. The results were shown in Table 1 where the PC, APC, and PC+APC to Chl ratio was 5.45, 4.02, and 4.63 times higher, respectively, in BG grown cells, confirming an increase in the phycocyanin pigment content in BG cells.

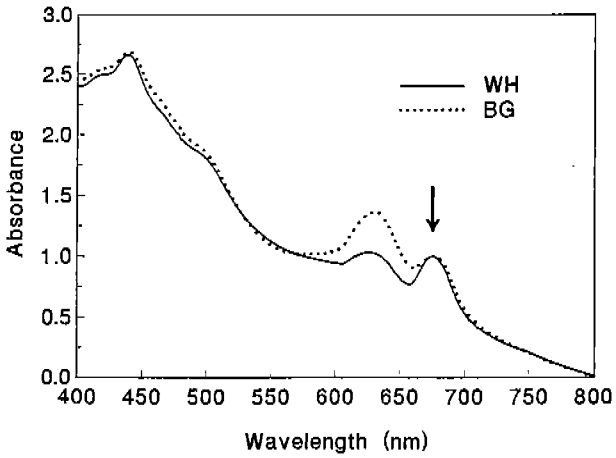

Fig. 1. Absorption spectra of *A. nidulans* cells grown under white light (WH) and blue-green light (BG). The spectra were arbitrarily normalized at the Chl absorbance maximum at 678 nm (↓).

Table 1. Changes of photosynthetic pigment contents in *A. nidulans* cells grown under white light (WH) and blue-green light (BG). All values represent the average of at least 6 measurements± SD

	WH	BG
PC/Chl	1.72± 0.24	9.37± 0.36
APC/Chl	2.30± 0.57	9.24± 0.66
PC/APC	0.78± 0.18	1.02± 0.04
PC+APC/Chl	4.02± 0.68	18.61± 0.99

PC, phycocyanin; APC, allophycocyanin.

It was reported that variation in pigment composition can be induced by light quality in algae (Falkowski and LaRoche, 1991). Thus the relative increase in the phycocyanin pigment contents in BG grown cells is thought to be the result of pigment adjustment of the cells to BG conditions. The chromatic effect can be related to the fact that the two pigments involved are preferentially linked with two different

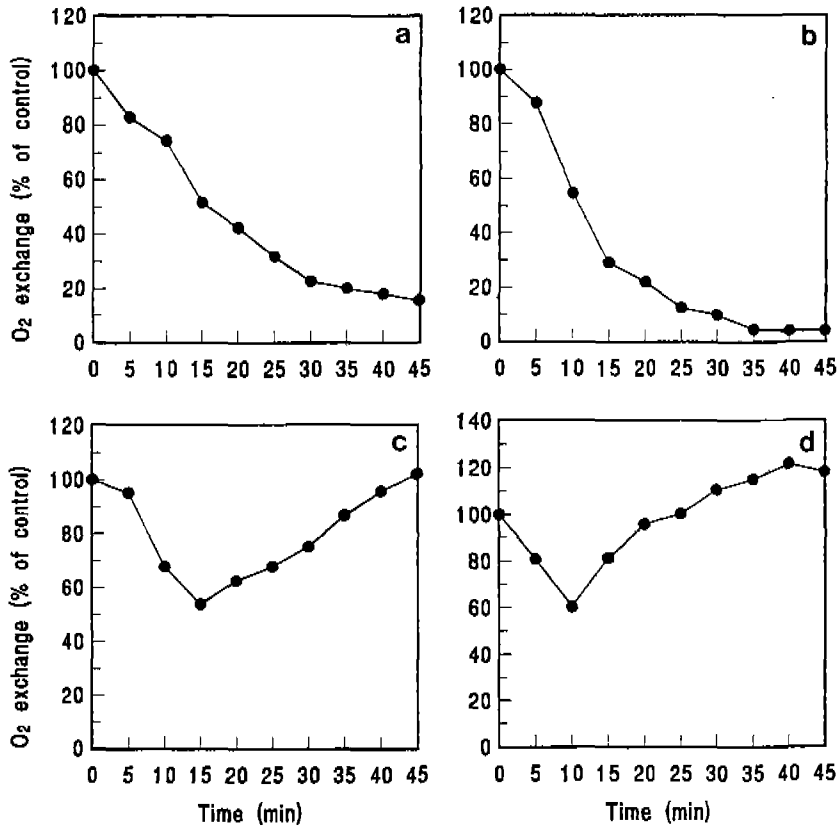

Fig. 2. Photosynthetic O_2 exchange rates (% of control) as a function of photoinhibitory (a, b) and recovery treatment time (c, d). Cells were grown under white light (a, c) and blue-green light (b, d).

photoreactions. The bulk of Chl a collects quanta mainly for PSI, and phycocyanin is the principal absorber for PSII (Jones and Myers, 1965; Wang *et al.*, 1977). Since Chl a mainly absorbs BG, BG grown cells needed more absorber for PSII, and consequently the BG grown cells had a higher phycobilin to Chl content. The chromatic changes in photosystem composition may bring a favorable energy distribution between two photosystems to the light condition. The chromatic adaptation was interpreted as a regulation of photosystem composition for a balanced electron flow under a given light condition (Fujita *et al.*, 1985).

Effect of chromatic adaptation on photoinhibition and recovery

The PFR of 1.2 $mmol \cdot m^{-2} \cdot s^{-1}$ was given for 45 min to *A. nidulans* cells to study difference in the photoinhibitory effects between WH and BG grown cells. About 50% reduction of photosynthetic activity was obtained in WH and BG grown cells after 15 min and 10 min treatment, respectively (Fig. 2). When cells were transferred to a PFR of 2 $\mu mol \cdot m^{-2} \cdot s^{-1}$, after a photoinhibitory treatment for 15 min in WH grown cells and 10 min in BG grown cells, their photosynthetic activity was recovered fully within 30 min in WH cells and 20 min in BG cells (Fig. 2). Though the photoinhibition in photosynthetic activity occurred more rapidly in BG grown cells, it seems that the recovery process is not significantly different between the two cells. In order to check the effect of light on the recovery process, cells were incubated for 30 min in the dark. In the dark, cells gained much less recovery, showing an increase from 49.9% to 53.0% in WH grown cells and 53.3% to 63.1% in BG grown cells after 30 min incubation (Fig. 3). It has been reported by Aro *et al.* (1993) that even though light is the underlying cause for the inactivation of electron transport and irreversible protein damage, light is also required to regain the photosynthetic activity. Our results also suggest that

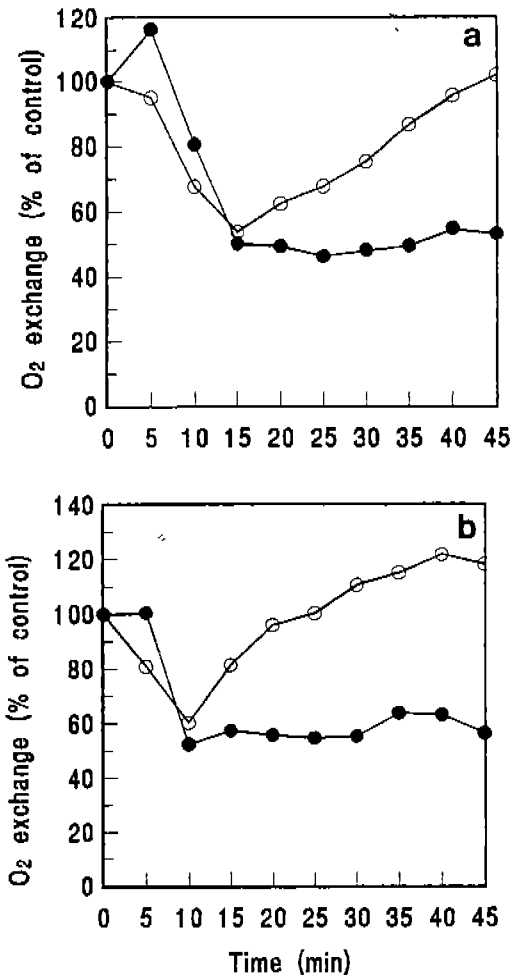

Fig. 3. Comparison of recovery in O_2 exchange rates in the dim light *vs* in the dark. Cells were grown under white light (a) and blue-green light (b). ○, Light condition; ●, dark condition.

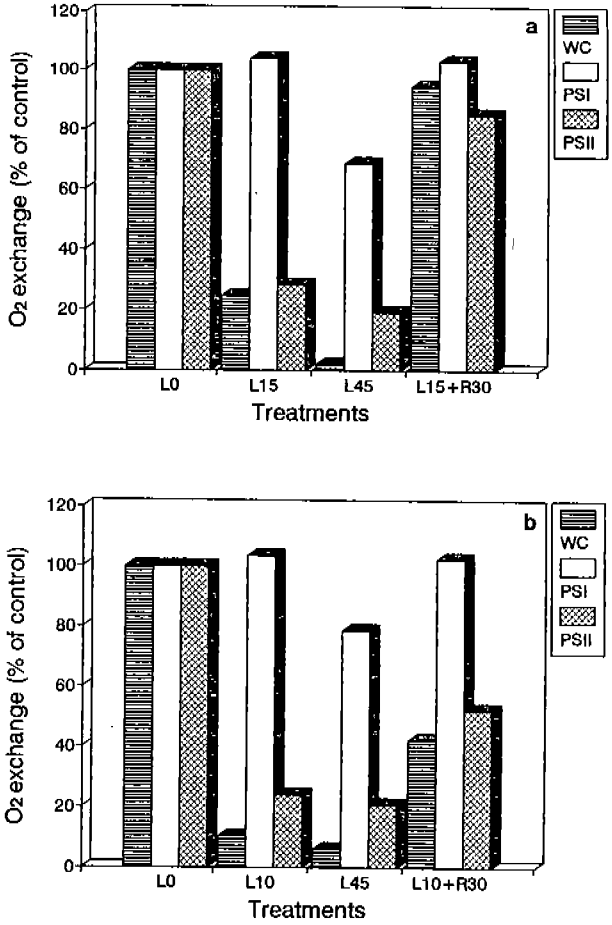

Fig. 4. PSII, PSI and whole chain electron transport (WC) activities of *A. nidulans* after photoinhibitory light treatment at a PFR of 1.2 mmol·m^{-2}·s^{-1}. All values are expressed as percentage of control. Cells were grown under white light (a) and blue-green light (b). (a) L0, control; L15, 15 min-light treatment; L45, 45 min-light treatment; L15+R 30, 15 min-light treatment followed by 30 min-recovery treatment. (b) L10, 10 min-light treatment; L10+R30, 10 min-light treatment followed by 30 min-recovery treatment.

a weak PFR (2 μmol·m^{-2}·s^{-1}) is required for the photoinhibited cells to recover.

To look for the main site of inhibition in the electron transfer chain, time course of change in the electron transport capacity was studied during the light treatments (Fig. 4). Electron transport activity of the whole chain (WC) and PSII was decreased to 24.5% and 28.4% of the control rate, respectively, but little change occurred in PSI activity after 15 min light exposure in WH grown cells. Continuous light illumination of up to 45 min induced more severe inhibition of O_2 exchange rate in WC and PSII, but the effect was less with PSII. When cells were incubated for 30 min under the recovering condition, the electron transport activity was increased to 94.7%, 103.3%, 85.1% of control in WC, PSI, and PSII, respectively (Fig. 4a). Similar results were obtained with BG grown cells except that they did not recover as much after recovery treatment (Fig. 4b). At the time of half inhibition for O_2 evolution (15 min for WH cells and 10 min for BG cells), the electron transport capacity was similarly inhibited both in the WH and BG cells, but PSII was affected most. Although there is a recent report that the photoinhibition site is PSI in leaves of *Cucumis sativus* L. at low temperature (Terashima *et al.*, 1994), it is commonly believed that PSII is much more suscep-

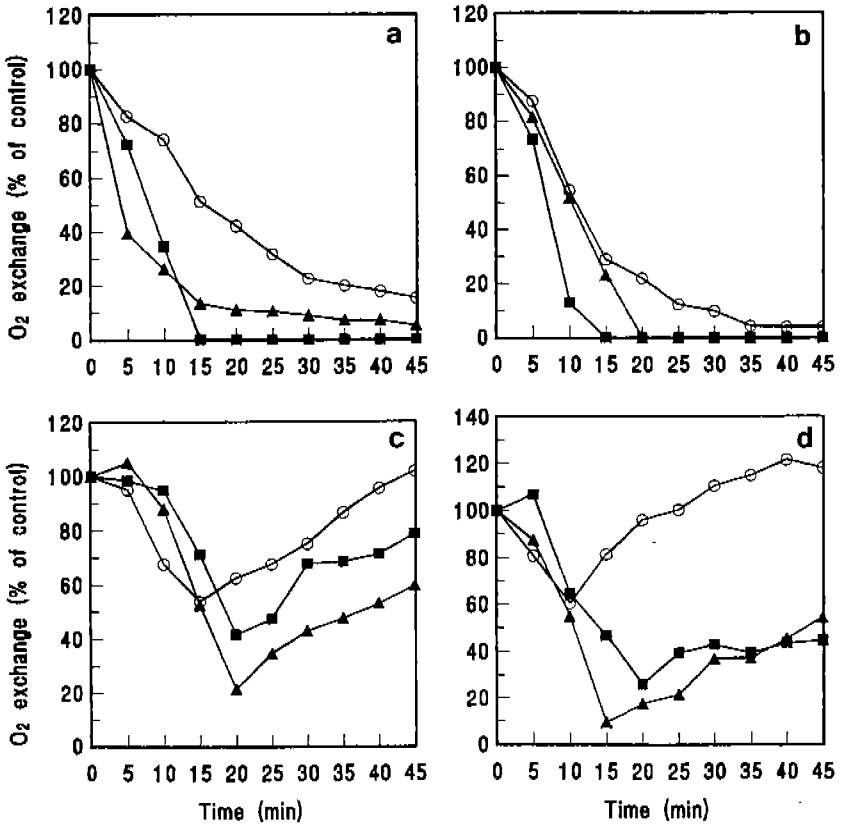

Fig. 5. Effects of rifampicin and chloramphenicol on relative O_2 exchange rates during photoinhibition (a, b) and recovery (c, d). Cells were grown under white light (a, c) and blue-green light (b, d). ○, Control; ▲, rifampicin treatment; ■, chloramphenicol treatment.

tible to photoinhibitory damage. Kyle *et al.* (1984) suggested that quinone anions, which may interact with molecular oxygen to produce an oxygen radical, selectively damage the apoprotein of the secondary acceptor of PSII, thus rendering it inactive and thereby blocking photosynthetic electron flow under conditions of high photon flux densities. In addition, Styring *et al.* (1990) suggested that photoinhibition inhibits the electron transfer between pheophytin and Q_A by impairing the function of Q_A. Godde *et al.* (1992) indicated that photoinactivation of PSII was induced by an inhibition of electron transfer from Q_A to Q_B.

Since O_2 exchange rate of the PSI was also decreased after light illumination up to 40-45 min, it is postulated that continuous high light treatment induces a wider range of damage. If cells were transferred to the recovering condition before severe damage was induced, however, electron transport activity was regained. These results indicate that photoinhibition is a reversible process.

Transcription and translation inhibitors

The photosynthetic activity decreased more rapidly when cells were pretreated with transcription inhibitor rifampicin for 20 min prior to photoinhibitory light treatment (Fig. 5). In WH grown cells, rifampicin treated cells showed faster decrease in the photosynthetic activity, reaching 90% inhibition in 15 min and 100% inhibition after 45 min treatment (Fig. 5a). In BG grown cells, the photosynthetic activity was similarly decreased as in control, reaching 50% inhibition after 10 min exposure to light, but reached 100% inhibition in 20 min (Fig. 5b). When rifampicin was added to an algal suspension at the onset of recovering treatment, in both WH and BG grown cells, O_2 exchange rate was only recovered at most to 60% of the control (Fig. 5c, d).

When the translation inhibitor CAP was added

to an algal sample prior to photoinhibitory treatment, both types of cells exhibited rapid suppression of photosynthetic activity, reaching 100% inhibition in 15 min (Fig. 5a, b). And cells showed little recovery of O_2 exchange rate after recovery treatment (Fig. 5c, d).

The observations that the photoinhibition was accelerated in both cells and that the susceptibilities to photoinhibition became approximately equal in both types of cells by the addition of rifampicin and CAP suggest that different capacities of the recovery process determine the different susceptibilities to photoinhibition of photosynthesis of the two types of cells. It has been suggested that the different susceptibilities to the photoinhibition in high light and low light grown *A. nidulans* cells depend on different turnover rates of target protein in PSII (Samuelsson *et al.*, 1987). Damaged D1 protein, generated by high light treatment, has to be removed to enable a new copy of the protein to be assembled into the PSII complex to regain photosynthetic activity (Aro *et al.*, 1993). Therefore, the difference in susceptibilities to photoinhibition between WH and BG grown cells could be explained by the different turnover rates of D1 protein, having a higher rates of protein synthesis in WH grown cells. Tyystjärvi *et al.* (1992) have shown that the loss of the D1 protein is severely retarded in low-light grown pumpkin leaves compared with high-light leaves, and this suggests that the susceptibility of low-light leaves to strong illumination and their poor ability to recover from photoinhibition largely derived from the low capacity of these leaves for degrading photodamaged D1. The same extent of inhibition cells and suppression of recovery in both types of cells by the inhibitor treatment suggest that recovery of the photosynthetic activity requires some process of transcription and translation.

The degradative process of D1 protein depletion should be considered as the first phase in the repair of photodamaged PSII reaction centers, after which a new copy of D1 protein could be inserted (Aro *et al.*, 1993). The D1 polypeptide is encoded by the *psbA* gene. While Mohamed and Jansson (1989) reported that expression of the *psbA* gene seems to be light-regulated mainly at the level of translation, Aro *et al.* (1993) suggested that regulation of cyanobacterial *psbA* gene expression operates at the level of transcription. In this study, it could be postulated that the regulation occurs both transcription and translation processes, since the reactivation process was suppressed by both the transcription and translation inhibitors. As shown in Fig. 3, both WH and BG grown cells did not show any recovery after recovery treatment in the dark, indicating that the recovery process involves the light-induced *psbA* gene or the D1 polypeptide.

Protein kinase, phosphatase and protease inhibitors

It was suggested that protein phosphorylation is involved in the repair process of photodamaged PSII reaction centers (Aro *et al.*, 1993). To see the effect of protein phosphorylation on photoinhibition, protein kinase inhibitor FSBA was treated and the results were shown in Fig. 6. In WH grown cells, O_2 exchange of FSBA treated cells was little different from that of control up to 15 min, and then the cells showed delayed photoinhibitory process retaining 1.7 times higher rate after 45 min (Fig. 6a). In addition, BG grown cells showed more prominent suppression of photoinhibition which was 2-3 times higher O_2 exchange rate after 25 min exposure to light (Fig. 6b). It is supposed that FSBA partially protected cells from photoinhibition or delayed photoinhibitory process. However, FSBA slightly affected the recovery process (Fig. 6c, d).

In order to check the possibility that dephosphorylation process is involved in the repair process, protein phosphatase inhibitor NaF was treated. While WH grown cells showed notable accerelation of photoinhibitory process, BG grown cells made a little difference (Fig. 6a, b). In the recovery process, both WH and BG grown NaF treated cells were recovered more than CAP treated cells, but did not make full recovery in 30 min as in control cells (Fig. 6c, d).

When cells were treated with protease inhibitor PMSF, both WH and BG cells showed a slight promotion in photoinhibitory process (Fig. 6a, b). The recovery process was delayed slightly in both cells, showing 30% suppression in BG grown cells (Fig. 6c. d). This result indicates that the proteolytic process is somewhat involved in the repair mechanism. Once damaged, the D1 protein has to be removed

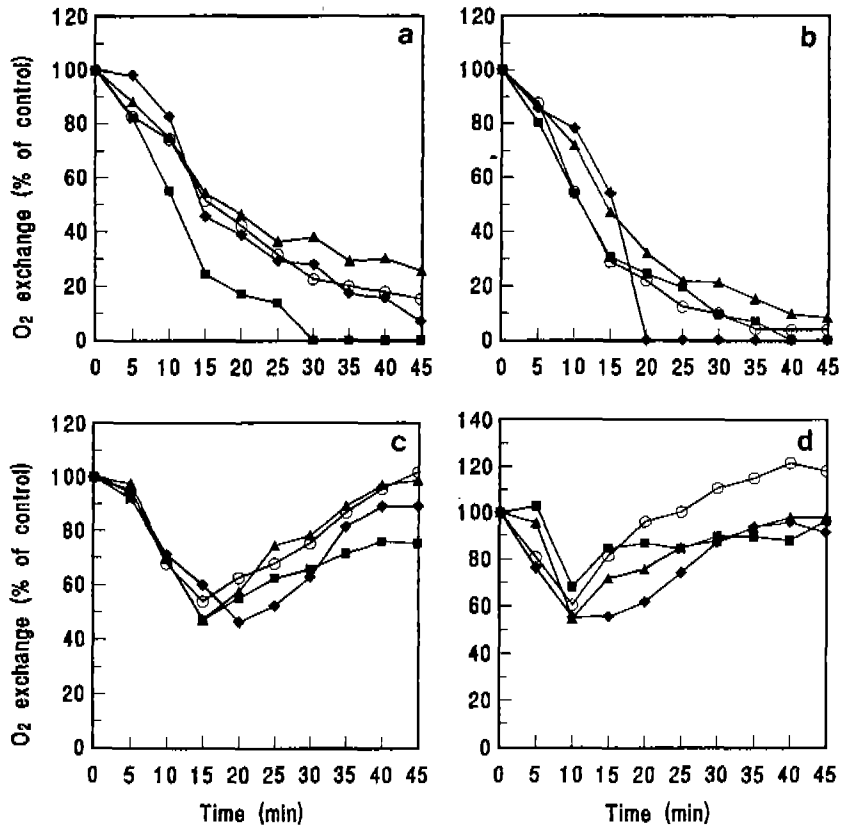

Fig. 6. Effects of 5′-*p*-fluorosulfonyl-benzoyladenosine, NaF and phenylmethylsulfonyl fluoride on relative O_2 exchange rates during photoinhibition (a, b) and recovery (c, d). Cells were grown under white light (a, c) and blue-green light (b, d). ○, Control; ▲, 5′-*p*-fluorosulfonyl-benzoyladenosine treatment; ■, NaF treatment; ◆, phenylmethylsulfonyl fluoride treatment.

to enable a new copy of the protein to be assembled into the PSII complex in order to reestablish photosynthetic function (Aro *et al.*, 1993). Salter *et al.* (1992) suggested that the damaged D1 protein is degraded by a proteolytic process rather than a photosensitized cleavage reactions. Thus the protease must be able to discriminate between a functional D1 protein and a damaged one (Virgin *et al.*, 1992). It is well established that D1 protein is indeed one of the phosphoproteins of PSII (Ikeuch *et al.*, 1987). Phosphorylation takes place at the threonyl residue at the N-terminus of the D1 protein which is exposed to the stromal surface of the thylakoids (Aro *et al.*, 1993).

It could be postulated that phosphorylation allows for coordinated degradation and biosynthesis of the D1 protein during the repair of photodamaged PSII centers. Phosphorylation induces conformational change in the damaged D1 protein, and consequently, the cleavage sites are exposed. Phosphorylated D1 protein, however, is a poor substrate for the protease and dephosphorylation is probably required prior to the primary proteolytic cleavage. A crucial observation was that phosphorylated D1 protein is less susceptible to degradation than the unphosphorylated D1 protein during illumination of isolated thylakoid membranes and PSII core particles (Aro *et al.*, 1992; Salter *et al.*, 1992). This observation, combined with the fact that in higher plants D1 protein degradation seems to proceed *via* a phosphorylated form (Kettunen *et al.*, 1991), suggests that protein phosphorylation is involved in D1 protein degradation.

Though it is probable that phosphorylation step is involved in the process of the D1 protein degradation, the precise mechanism is not known. Our results suggest that protein dephosphorylation is more important than protein phosphorylation in recover-

ing photosynthetic activity. In such cases, protein degradation probably occurs via protein dephosphorylation.

While it could be expected that the protease inhibitor PMSF promotes the photoinhibitory process, the experimental data did not show any marked promotion. The photoinhibitory process of the PMSF treated cells was not significantly changed as that of the control. Consequently, protease activity has some effect on photoinhibitory process, but it is not an important step.

In summary, it is postulated that the D1 protein is phosphorylated via an activation of protein kinase when photoinhibitory light is treated, after which the protein is degraded and removed by proteolytic activity. Because the photoinhibitory process is promoted by the addition of protein phosphatase, the dephosphorylation process may also be involved. Between the phosphorylation-dephosphorylation and proteolytic process, dephosphorylation process was most important. After the protein degradation, a new copy of the D1 protein is synthesized and inserted into the thylakoid membrane. Cells grown in WH and BG conditions show difference in the susceptibility of photoinhibition, and the increased sensitivity of BG adapted cells might be attributed to the decline of protein synthesis rates, in both transcriptional and translational level.

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青綠色光에 適應된 *Anacystis nidulans*의 光抑制와 回復

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적 요

광질 조건을 다르게 하여 생장시킨 *Anacystis nidulans* 세포에서 광억제 유도 및 회복 메카니즘의 차이를 조사하였다. 청록색광에서 자란 세포가 백색광에서 자란 세포에 비해서 피코시아닌과 엽록소 함량 및 피코시아닌/엽록소 비율이 증가하였다. 1.2 mmol·m^{-2}·s^{-1}의 photon fluerce rate에서 광억제를 유도시킨 결과, 백색광에서 자란 세포에서는 빛 조사 15분 후에 광합성능이 50%로 억제되었으나 청록색광에서 자란 세포는 10분만에 같은 정도의 억제 효과를 나타내었는데, 이는 광계 II의 활성도 감소에서 기인하였으며, 낮은 광도에서 30분간 배양하면 광합성능은 회복되었다. 전사억제제인 rifampicin, 번역억제제인 chloramphenicol에 의해서 광억제는 촉진되었고 회복은 억제되었으며, 광억제 과정에서 나타나는 두 세포간의 민감도 차이는 억제제를 처리하면 없어졌다. Protein phosphatase 억제제인 NaF와 protease 억제제인 phenylmethylsulfonyl fluoride에 의해서도 광억제 현상이 촉진되었고 회복은 억제되었으나 광억제의 회복 과정에서 인산화-탈인산화 및 protease의 작용은 각 색성적응된 세포에서 별 차이가 없었다. 이상의 결과로부터 청록색광에서 적응된 *A. nidulans* 세포에서 광억제의 민감도 증가는 단백질 합성 수준의 감소에서 기인하는 것으로 생각되며, 광억제의 회복에는 단백질의 인산화 후 탈인산화 및 protease 작용이 관여하는 것으로 나타났다.

주요어 : *Anacystis nidulans*, 색성적응, 광억제, 회복, 단백질 합성

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