High CO₂ Concentration Alleviates the Block in Photosynthetic Electron Transport in an ndhB-Inactivated Mutant of Synechococcus sp. PCC 7942

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The high-concentration CO₂-requiring mutant N5 of Synechococcus sp. PCC 7942 was obtained by the insertion of a kanamycin-resistant gene at the EcoRI site, 12.4 kb upstream of rbc. The mutant is unable to accumulate inorganic carbon internally and exhibits very low apparent photosynthetic affinity for inorganic carbon but a photosynthetic Vₚmax similar to that of the wild type. Sequence and northern analyses showed that the insertion inactivated a gene highly homologous to ndhB, encoding subunit II of NADH dehydrogenase in Synechocystis sp. PCC 6803 (T. Ogawa [1991] Proc Natl Acad Sci USA 88: 4275-4279). When the mutant and the wild-type cells were exposed to 5% CO₂ in air, their photosynthetic electron transfer capabilities, as revealed by fluorescence and thermoluminescence measurements, were similar. On the other hand, a significant decrease in variable fluorescence was observed when the mutant (but not the wild-type) cells were exposed to low CO₂ under continuous light. The same treatment also resulted in a shift (from 38-27°C) in the temperature at which the maximal thermoluminescence emission signal was obtained in the mutant but not in the wild type. These results may indicate that subunit II of NADH dehydrogenase is essential for the functional operation of the photosynthetic electron transport in Synechococcus under low but not high levels of CO₂. We suggest that the inability to accumulate inorganic carbon under air conditions stems from disruption of electron transport in this mutant.

The cyanobacterial CCM (see Aizawa and Miyachi, 1986; Kaplan et al., 1990; Miller et al., 1990; Coleman, 1991; Kaplan et al., 1991; Raven, 1991; Badger and Price, 1992, for recent reviews) involves an active transport of Ci, activated and energized by light (Kaplan et al., 1987). It has been proposed that PSII activity is required for the activation and that energization is via PSI activity (Ogawa et al., 1988). Recent studies (Miller et al., 1988, 1991) demonstrated a strong correlation between the rates of Ci uptake and of fluorescence quenching when Synechococcus cells were provided with a range of Ci concentrations. These data indicate a strong effect of Ci uptake on the photosynthetic electron transport, but the underlying mechanisms are not understood.

High-concentration CO₂-requiring mutants are being used as a tool to study the physiological and molecular mechanisms involved in the operation of the CCM (Marcus et al., 1986; Ogawa et al., 1987; Schwarz et al., 1988; Friedberg et al., 1989; Price and Badger, 1989; Bedu et al., 1990; Kaplan et al., 1990; Ogawa, 1990; Coleman, 1991; Kaplan et al., 1991; Lieman-Hurwitz et al., 1991; Ogawa, 1991; Badger and Price, 1992; Ogawa, 1992; Price et al., 1992; Schwarz et al., 1992). These mutants are also being used to test predictions made by a quantitative model that ascribes a central role in the functioning of the CCM to the structural organization of the carboxysomes (Price and Badger, 1989; Reinhold et al., 1989; Reinhold et al., 1991; Schwarz et al., 1992). In the case of Synechococcus sp. PCC 7942, the lesions in all of the mutants (Kaplan et al., 1991) were mapped in the genomic region of rbc, the operon encoding the large and small subunits of Rubisco. Clustering of genes involved in a particular physiological activity such as the transport of sulfate has already been demonstrated in Synechococcus (Green et al., 1989).

Inactivation of ndhB, encoding subunit II of NADH dehydrogenase in Synechocystis PCC 6803, resulted in a reduced ability to accumulate Ci internally. Consequently, the mutant could only grow in the presence of a high concentration of CO₂ (Ogawa, 1991). In the present report we present certain physiological characteristics of a mutant of Synechococcus PCC 7942 obtained following inactivation of ndhB, located 12 kb upstream of rbc. It is suggested that NADH dehydrogenase is required for photosynthetic electron transport in the presence of low, but not of high, CO₂.

MATERIALS AND METHODS

Cultures of Synechococcus sp. strain PCC 7942 and the mutant N5 obtained following a modification of this strain (see below) were grown in BG11 medium (Stanier et al., 1971) supplemented with 20 mM Hepes-NaOH (pH 8.0) and kanamycin (25 µg/mL) in the case of the mutant) in the presence of high (5% CO₂ in air) or low (air) levels of CO₂.

Abbreviations: Ci, inorganic carbon; CCM, inorganic carbon-concentrating mechanism; Fₚmax, fluorescence intensity in the presence of 5 µM DCMU; Fₑ, signal intensity at 2 ms when the shutter is completely open; Fₛ, steady-state signal in the absence of DCMU; Kmr, kanamycin resistance; ORF, open reading frame; Qₐ, primary electron-accepting plastoquinone of PSII; Qb, second electron-accepting plastoquinone of PSII.
as previously described (Marcus et al., 1986). The rate of growth was determined from the increase in A750 using a Shimadzu recording spectrophotometer UV200. The rates of Ci uptake and of Ci-dependent O2 exchange were measured by the filtering centrifugation and the O2 electrode techniques, respectively, as described earlier (Kaplan et al., 1988). Genomic DNA was isolated as described elsewhere (Williams, 1988). Standard rDNA techniques (Sambrook et al., 1989) were used for Southern analysis and cloning. Analysis of the DNA sequence was performed with the Sequenase kit (United States Biochemical Corp.). Both strands were sequenced with no ambiguities. Synthetic oligonucleotides (Biotechnology General, Rehovot, Israel) were used in cases in which the subclones did not overlap.

The high-concentration CO2-requiring Km mutant N5 was obtained as follows: a genomic SalI fragment (3.8 kb) located 10.1 to 13.9 kb upstream of rbc (Fig. 1) was subcloned from a genomic library of Synechococcus sp. PCC 7942 (kindly provided by Dr. A. Grossman) into the SalI site of a modified Bluescript SK plasmid. The latter was obtained following digestion of the plasmid with EcoRV and SmaI and blunt-end ligation. The PstI and EcoRI sites were therefore removed. The 1.3-kb EcoRI fragment bearing npII (encoding Km') from pUC71 (Friedberg et al., 1989) was inserted in the EcoRI site (originally located 12.4 kb upstream of rbc, see schematic map in Fig. 1). Synechococcus cells were then transformed by this Km' cartridge was inserted, the genomic DNA from N5, was chosen for further analysis.

Confirmation that the Km' cartridge was inserted in the desired EcoRI site in mutant N5 was obtained by Southern analysis (see below) and by sequence analysis of the relevant genomic fragment from N5. To sequence the region where the Km' cartridge was inserted, the genomic DNA from N5 was digested with EagI, and the fragments of 3.5 to 4.0 kb were isolated and ligated within the EagI site in Bluescript SK. Competent cells of Escherichia coli (DH5a) were transformed with a small volume of double-distilled water. Formaldehyde gel electrophoresis, blotting of the RNA, and hybridization with the DNA probe were performed as described elsewhere (Sambrook et al., 1989).

Fluorescence kinetics and thermoluminescence glow curves were recorded using high-concentration CO2-grown cells (4 μg of Chl/mL in the former and 140 μg of Chl/mL in the latter). The cell suspensions were divided into two parts; one part was aerated for 1 h with 5% CO2 in air, and the other was bubbled with air. The cells were then dark adapted for 5 min at room temperature, and the fluorescence kinetics or thermoluminescence glow curves were recorded. These measurements were also performed on cells that were illuminated (1500 μE m⁻² s⁻¹ of white light) for 2 min after the dark period.

Fluorescence induction was measured with the aid of a homemade fluorimeter connected to a PC computer, which operated the shutter and recorded the digitized signal as described previously (Ohad et al., 1988). Excitation (260 μE m⁻² s⁻¹) was provided by blue light (filter 4–96; Corning Glass, Corning, NY), and the photodiode was protected by a red cut-off filter (filter 6–65; Schott Glaswerke, Meinz, Germany). The signal was sampled at a rate of 15,000 points/s for the first 10 ms after the opening of the shutter and 200 points/s for the rest of the recording duration (2 s). The results are provided as Fmax, F0, and F. The ratios (Fmax - F0)/F0 and (Fmax - F)/F were calculated because the former provides an indication of the capability of the reaction center of PSII to transfer electrons to Qa, whereas the latter expresses the photosynthetic capability beyond the Qa site (Ohad and Hirschberg, 1992).

Thermoluminescence glow curves (Inoue, 1987) were recorded using a homemade apparatus. The cells (400 μL) were placed on the cell holder of the thermoluminescence apparatus and dark adapted for 5 min at room temperature. One to six saturating single-turnover light flashes were provided, followed by immediate cooling by liquid nitrogen. Charge recombination was elicited by heating the sample (0.7°C/s). The intensity of the light emitted resulting from charge recombination between the Qa⁻ or Qb⁻ and the S-states as a

Figure 1. Schematic maps of the genomic region 10 kb upstream of the rbc operon of Synechococcus sp. PCC 7942 in the wild type (A) and the mutant N5 (B). Km, Kanamycin-resistance cartridge (the arrow indicates the orientation of the Km' gene); S, SalI; P, PstI; E, EagI; C, Clal; A, Apal; R, EcoRI. 

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function of the temperature during the heating was recorded by a photon counter and transferred to a PC computer programed to control the heating rate and to display the results.

RESULTS

Southern, Northern, and Sequence Analyses

Southern analyses (Fig 2) were performed on genomic DNA isolated from the wild type and from mutant N5, digested with PstI, Eagl, and Clai using the 1.3-kb EcoRI fragment (bearing nptII) and the genomic 1.3 kb PstI fragment (Fig 1) as probes. The nptII probe hybridized only with DNA fragments from the mutant, exhibiting bands of approximately 1.3 kb (PstI), 4.2 kb (Eagl), and 4.9 and 1.0 kb (Clai). The genomic PstI probe hybridized with fragments of 1.3 kb (PstI), 2.7 kb (Eagl), and 4.2 kb (Clai) in DNA from the wild type and with fragments of 1.3 kb (PstI), 4.3 kb (Eagl), and 5.0 kb (Clai) in DNA from the mutant. The expected 1.0-kb fragment in the Clai digest of the mutant was not detected, presumably because the homology with the probe extends over only 60 bp (Figs. 2 and 3). Hybridization with a probe prepared from the Bluescript SK plasmid did not detect any homologous fragment in the mutant, confirming that no plasmid sequences were present (not shown). These data clearly indicated that the cloned fragment had been inserted into the desired EcoRI site via a double-crossover recombination event and that complete segregation (i.e. replacement of the original with the modified region) was obtained. The insertion of the Km' cassette at the EcoRI site was also confirmed by sequence analysis of the relevant genomic DNA fragment cloned from N5 (not shown).

Sequence analysis of 2094 nucleotides between the Clai site and the upstream SalI site (Fig. 1) of the genomic DNA from the wild type (Fig. 3) revealed an ORF on the strand complementary to the one encoding rbc, starting from position 336 and extending to a stop codon in position 1898 (from the Clai site). The EcoRI restriction site, where the Km' gene was inserted is underlined. A putative −10 box (position 264−269) is underlined, and a possible ribosome-binding site (position 323−325) is labeled s.d and underlined. The sequence of the protein encoded by

Figure 2. Southern analysis of the genomic DNA from mutant N5 and the wild type. A 1.3-kb EcoRI fragment containing the gene encoding Km' and a 1.3-kb PstI fragment from the genomic DNA containing the EcoRI site (see Fig. 1A) were used as probes in A and B, respectively. Genomic DNA from mutant N5 in lanes a, c, and e; genomic DNA from wild type is in lanes b, d, and f. The restriction enzymes used were PstI (a and b), Eagl (c and d), and Clai (e and f). M, Molecular size markers.

Figure 3. Nucleotide sequence of the wild-type DNA between the Clai site and the upstream SalI site and the deduced amino acid sequence of the protein encoded by

Figure 3. Nucleotide sequence of the wild-type DNA between the Clai site and the upstream SalI site and the deduced amino acid sequence of the protein encoded by ndhB. The EcoRI site (position 668−673) where the Km' gene was inserted is underlined. A putative −10 box (position 264−269) is underlined, and a possible ribosome-binding site (position 323−325) is labeled s.d and underlined. The inverted repeat (positions 1947−1956 and 1964−1973) in the downstream region of ndhB is also underlined.
The initiation and termination points of the RNA were not determined, but analysis of the sequence in Figure 3 indicated a putative −10 sequence in position 264 (Schneider et al., 1991). The inverted repeat downstream of the ORF (underlined in Fig. 3) may be involved in transcription termination.

The ORF encodes a highly hydrophobic protein of 521 amino acids with an estimated molecular mass of 55,067 D. Sequence comparison (Fig. 5) showed 71.2% identity with the gene product of ndhB encoding subunit II of NADH dehydrogenase from Synechocystis sp. PCC 6803 (Ogawa, 1991). The analysis also indicated high homology to the relevant genes from the chloroplast genome of Marchantia polymorpha and human mitochondria, as was earlier reported by Ogawa (1991). Therefore, we concluded that the ORF is the Synechococcus equivalent of ndhB.

**Physiological Characterization of Mutant N5**

The mutant N5 was unable to grow in the presence of air levels of CO₂ in either solid or liquid BG11 medium. In the presence of 5% CO₂ on the other hand, the rate of growth exhibited by the mutant was identical with that of the wild type. A short lag was observed in the growth rate of N5 in the presence of kanamycin (25 μg/mL), but the lag could be diminished when kanamycin was provided 24 h after the beginning of the growth experiment. The curves relating the rate of photosynthetic O₂ evolution by N5 as a function of the extracellular Ci concentration showed a photosynthetic Vₘₐₓ similar to that of the wild type (320 μmol of O₂ mg⁻¹ of Chl h⁻¹) but approximately 100-fold lower apparent photosynthetic affinity for extracellular Ci. [Kᵢ(Ci) was 0.1 and 10 mM Ci in the wild type and the mutant, respectively.] The detailed analyses of the growth curves and of the response of photosynthetic rate to the extracellular Ci concentration are not presented, because the data were similar to those reported in the case of other high CO₂-requiring mutants of Synechococcus (Marcus et al., 1986; Schwarz et al., 1988; Price and Badger, 1989; Lieman-Hurwitz et al., 1991). The low apparent photosynthetic affinity for Ci in mutant N5 is most probably attributable to the lack of accumulation of Ci internally (Table I). The Ci uptake activity of high CO₂-grown wild-type cells was rather low, but it increased following exposure to air levels of CO₂. On the other hand, N5 cells grown in the presence of high CO₂ and exposed to low CO₂ conditions showed very little activity of Ci uptake and consequently a relatively low internal Ci pool.

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**Figure 4.** Northern analysis of total RNA from the wild type. A 1.3-kb PstI fragment containing most of ndhB was used as a probe. Lane a, Cells grown under high CO₂; lane b, cells transferred to low CO₂ 90 min before the isolation of the RNA. M, RNA markers in kb.

**Figure 5.** Alignment of the deduced amino acid sequence of ndhB from Synechococcus PCC 7942 (A) with the putative ndhB product of Synechocystis PCC 6803 (B) from Ogawa (1991). Identical amino acids are marked by asterisks (*); the termination codon is marked by X.
Table 1. Ci accumulation by wild-type (WT) and N5 cells of Synechococcus PCC 7942

<table>
<thead>
<tr>
<th>[Ci]</th>
<th>WT High CO₂</th>
<th>N5 High CO₂</th>
<th>WT Low CO₂</th>
<th>N5 Low CO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>nmol of Ci/μg of Chl</td>
<td>0.38</td>
<td>0.028</td>
<td>0.011</td>
<td>0.173</td>
</tr>
<tr>
<td></td>
<td>4.00</td>
<td>0.966</td>
<td>0.344</td>
<td>1.616</td>
</tr>
</tbody>
</table>

Uptake of Ci in cyanobacteria is activated and energized by photosynthetic light (see introduction). The lower Ci transport activity in mutant N5, a consequence of the inactivation of ndhB, might, therefore, be due to a defect in the activation or energization of the process. Fluorescence and thermoluminescence measurements were used to assess the effect of an ambient concentration of CO₂ on the photosynthetic electron transport capabilities (Figs. 6 and 7). The fluorescence parameters \( F_{\text{max}} \), \( F_{\text{m}} \), and \( F_{\text{s}} \) and the ratios \( \left( F_{\text{max}} - F_{\text{s}} \right) / F_{\text{s}} \) and \( \left( F_{\text{m}} - F_{\text{s}} \right) / F_{\text{s}} \) calculated therefrom were rather similar in wild-type and mutant cells grown at and exposed to high CO₂ (Fig. 6). These results were expected, because in the presence of high CO₂ the growth rates of the wild type and the mutant were similar. On the other hand, when N5 cells were exposed to low CO₂ the steady-state fluorescence intensity in the presence \( F_{\text{max}} \) and absence \( F_{\text{s}} \) of DCMU were almost identical. Therefore, although the ratio \( \left( F_{\text{max}} - F_{\text{s}} \right) / F_{\text{s}} \) (which indicates the ability to transfer electrons to the Qₐ site) was hardly affected by exposure to low CO₂, the \( \left( F_{\text{max}} - F_{\text{s}} \right) / F_{\text{s}} \) ratio decreased from 0.47 and 0.45 in wild-type and mutant cells exposed to high CO₂, respectively, to 0.30 and 0.05 in wild-type and mutant cells exposed to low CO₂, respectively. These data indicate that the mutant is essentially unable to transfer electrons in PSII beyond the Qₐ site under low CO₂ conditions and continuous light.

The conclusion that the inactivation of ndhB in mutant N5 resulted in inhibition of photosynthetic electron transport under low, but not under high CO₂ was further supported by the analysis of the thermoluminescence signal (Fig. 7). When wild-type and mutant cells were exposed to high CO₂, the maximum emissions were obtained at 37 to 39°C (in different experiments). Treatment with DCMU (5 μM), conditions under which the Qₐ signal is observed instead of that of Qₐ, resulted in a shift of the maximal signals to 24 and 27°C in the wild type and N5, respectively. Exposure of wild-

![Figure 6](https://example.com/image6.png)

**Figure 6.** The fluorescence intensity of wild-type (WT) and N5 cells exposed to high or low levels of CO₂ in the presence (+) or absence (−) of DCMU (5 μM). The cells were dark adapted for 5 min at room temperature, and the first measurement was taken following a single flash (dark adapted). The cells were then kept in the light for 2 min, and the data provided are those obtained following the light treatment. (See “Materials and Methods” for experimental details.)
type cells to low CO₂ did not affect the temperature of maximum emission. On the other hand, the same treatment resulted in a shift of the maximal signal to 27°C in the cases of the mutant. This shift was completely reversible following transfer of the cells back to high CO₂. When the wild-type cells were exposed to a series of flashes, they exhibited the well-established pattern (Inoue, 1987) of maximum emission after the second and sixth flash (not shown). This was not the case in N5 cells exposed to low CO₂, in which a clear, repeatable pattern was not observable. These data indicate that, in the mutant N5, the photosynthetic electron transport in PSII is severely inhibited following reduction in the ambient CO₂ concentration.

Inactivation of  ndhB in mutant N5 resulted in 50 to 70% inhibition of the rate of dark respiration. Unlike the case of photosynthetic electron transport, the inhibition of dark respiration was not affected by the concentration of CO₂. However, because of the release of CO₂ in respiration, it was difficult to control its concentration in these experiments. Inhibition of dark respiration was also reported in the case of Synechocystis PCC 6803 and Synechococcus PCC 7002 mutants in which  ndhB (Ogawa, 1991) or  ndhF (Yu et al., 1993), respectively, were inactivated.

**DISCUSSION**

Insertion of the Km' cartridge within an ORF that is highly homologous to ndhB (encoding subunit II of NADH dehydrogenase from Synechocystis PCC 6803 [Ogawa, 1991]) inactivated the gene and resulted in a high CO₂-requiring mutant, N5. It has been proposed (Ogawa, 1991, 1992) that this phenotype stems from a defect in the mutant's ability to accumulate Ci internally, as also suggested by the data presented in Table I. Consequently, there was a 100-fold reduction of the apparent photosynthetic affinity for extracellular Ci, which was too low to enable growth of the mutant under air levels of CO₂.

This is the first report of a high CO₂-requiring mutant of Synechococcus in which inability to concentrate Ci has been observed. The lesion in all of the other mutants that exhibit the same phenotype is due to either defective carboxysomes (Friedberg et al., 1989; Price and Badger, 1989; Price et al., 1992) or inability to produce purines following exposure to a low concentration of CO₂ (Schwarz et al., 1992). The RKn and M55 mutants of Synechocystis sp. PCC 6803, in which the  ndhB was inactivated (Ogawa, 1991), were also unable to accumulate Ci internally.

Earlier studies demonstrated that uptake of Ci by cyanobacteria is light dependent and that photosynthetic light is involved in the activation and energization of the Ci-transporting system (Kaplan et al., 1987). Fluorescence and thermoluminescence measurements (Figs. 6 and 7) indicated severe inhibition of the photosynthetic electron transport capability beyond the Qb site in the mutant cells exposed to low CO₂. Transfer of the cells from low to high CO₂ alleviated the inhibition. Therefore, we conclude that the high CO₂-requiring phenotype of the mutant most probably results from the block in photosynthetic and respiratory electron transport observable when the cells are exposed to low CO₂. The reduced ability to accumulate Ci internally is likely to stem from the low-energy state of these cells consequent on this inhibition.

Studies by Myers (1993) have indicated that exposure of Synechococcus 6301 to a light of 680 nm (which is absorbed primarily by PSI) diverted the respiratory electron flow from oxygen reduction to P700⁺. The role of subunit II of NADH dehydrogenase in cyanobacterial photosynthetic and respiratory electron transport and its possible involvement in the interaction between these processes (Peschek, 1987), however, are not yet understood. Furthermore, there are contradictory reports regarding the location of the subunits of NADH dehydrogenase. Ogawa (1992) suggested that they are located in the thylakoid membranes, whereas Berger et al. (1991) indicated both the thylakoid and cytoplasmic membranes as their site. Therefore, in a scheme proposed by Nicholls et al. (1992), the presence of two distinct electron transfer carrier systems, including NAD(P)H dehydrogenase, on both the cytoplasmic and thylakoid membranes was suggested.

Measurements of the rate of reoxidation of P700⁺ in the presence of different electron transport inhibitors and acceptors in whole cells of Synechococcus PCC 7002 and the psaE,  ndhF, and psaE/ndhfF mutants thereof (Yu et al., 1993) suggested that NADH dehydrogenase donates electrons to the plastoquinone pool, providing the link between the photosynthetic and respiratory electron transport in cyanobacteria. The scheme presented by these authors is supported by our observation that the photosynthetic electron transport beyond the Qb site is severely inhibited in N5 exposed to low CO₂. Further studies are, however, required to establish the means by which the presence of high CO₂ overcomes this inhibition. This may have significance for the elucidation of the well-established effect of bicarbonate on the activity of PSI (Eaton-Ray et al., 1986) and the possible involvement of NADH dehydrogenase in this effect.

Cloning of several genes encoding different subunits of the NADH dehydrogenase complex (Ogawa, 1992; Steinmüller, 1992) might help to elucidate the location and role of their gene products and the interaction between them. Inactivation of ndhB, ndhK, and ndhL (Ogawa, 1992), encoding different subunits of NADH dehydrogenase of Synechocystis, resulted in high CO₂-requiring mutants unable to accumulate Ci within the cell. On the other hand, mutants in which  ndhC (Ogawa, 1992) and ndhfF (Yu et al., 1993) were inactivated were able to grow in the presence of low CO₂. We have isolated a revertant of N5 that probably resulted from a suppression mutation as suggested by the fact that Southern analysis (not shown) gave similar results to those obtained in N5. Analysis of such mutants might help to elucidate the role of, and the interaction between, the different subunits of NADH dehydrogenase.

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Cyanobacterial Mutant Defective in NADH Dehydrogenase

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