Passive Entry of CO₂ and Its Energy-dependent Intracellular Conversion to HCO₃⁻ in Cyanobacteria Are Driven by a Photosystem I-generated ΔμH⁺**

Dan Tchernov‡, Yael Helman‡, Nir Keren‡, Boaz Luz‡, Itzhak Ohad‡, Leonora Reinhold‡, Teruo Ogawa‡, and Aaron Kaplan‡‡

From the ‡Faculty of Science and Mathematics and The Minerva Center for Photosynthesis under Stress, The Hebrew University of Jerusalem, 91904 Jerusalem, Israel and the ‡Bioscience Center, Nagoya University, Chikusa, Nagoya 464–8601, Japan

CO₂ entry into Synechococcus sp. PCC7942 cells was drastically inhibited by the water channel blocker p-chloromercuriphenylsulfonic acid suggesting that CO₂ uptake is, for the most part, passive via aquaporins with subsequent energy-dependent conversion to HCO₃⁻. Dependence of CO₂ uptake on photosynthetic electron transport via photosystem I (PSI) was confirmed by experiments with electron transport inhibitors, electron donors and acceptors, and a mutant lacking PSI activity. CO₂ uptake was drastically inhibited by the uncouplers carbonyl cyanide m-chlorophenylhydrazone (CCCP) and ammonia but substantially less so by the inhibitors of ATP formation arsenate and N,N-dicyclohexylcarbodiimide (DCCD). Thus a ΔμH⁺ generated by photosynthetic PSI electron transport apparently serves as the direct source of energy for CO₂ uptake. Under low light intensity, the rate of CO₂ uptake by a high-CO₂-requiring mutant of Synechococcus sp. PCC7942, at a CO₂ concentration below its threshold for CO₂ fixation, was higher than that of the wild type. At saturating light intensity, net CO₂ uptake was similar in the wild type and in the mutant IL-9 suggesting common limitation by the rate of conversion of CO₂ to HCO₃⁻.

These findings are consistent with a model postulating that electron transport-dependent formation of alkaline domains on the thylakoid membrane energizes intracellular conversion of CO₂ to HCO₃⁻.

On illumination, many photosynthetic microorganisms maintain the concentration of dissolved CO₂ ([CO₂(dis)]) in their surrounding medium below that expected at chemical equilibrium with HCO₃⁻ (1–5). This displacement of [CO₂(dis)] from equilibrium can be observed in the absence of CO₂ fixation and is largely due to CO₂ uptake, intracellular conversion to HCO₃⁻, and release of the latter into the medium (5, 6). The reverse phenomenon has been described in Synechococcus WH 7803 (7) and Nannochloropsis sp. (8, 9) where HCO₃⁻ uptake, internal conversion to CO₂, and efflux of the latter result in elevated [CO₂(dis)] in the medium. HCO₃⁻ transport systems, in Cyanobacteria, are probably located at the cytoplasmic membrane and are believed to be driven by ATP either directly (10) or possibly indirectly (11–13). CO₂ uptake has been observed to result in HCO₃⁻ accumulation in the cytoplasm where [CO₂(dis)] is maintained below that expected at chemical equilibrium, and it has been inferred that a CA¹-like activity is involved in its uptake and intracellular conversion to HCO₃⁻ (6, 14–17). The location of the CA-like activity has not been identified and the mode of energization of the active HCO₃⁻ accumulation is not understood. Active transport of CO₂ across the plasmalemma has also been suggested (5, 18), but it is difficult to distinguish this from diffusion of CO₂ across the plasma membrane with subsequent energy-dependent conversion to HCO₃⁻ (6, 19). Passive entry of CO₂ across the membrane may occur via aquaporins (20), a possibility examined here by the application of a water channel blocker (WCB), p-chloromercuriphenylsulfonic acid. Use of this WCB prevented changes in cell volume and inactivation of PSI and PSII following osmotic stress in Synechococcus sp. strain PCC7942 (21).

Until recently, it was widely accepted that cyclic PSI activity plays the major role in energization of CO₂ uptake in Cyanobacteria (22, 23). Some recent observations appear to conflict with this conclusion. In the ΔndhD1/D2 mutant of Synechocystis sp. strain PCC6803 lacking components of NADP/H dehydrogenase (NDH-1), oxidation of P700 was depressed, but CO₂ uptake was only slightly affected (24). On the other hand, in the ΔndhD3/D4 mutant (25) and the ΔndhD3 mutant of Synechococcus sp. strain PCC7002 (26) CO₂ uptake was drastically depressed with only a small effect on P700 oxidation (i.e. PSI cyclic electron transport, ET). We have therefore reexamined the involvement of PSI in CO₂ uptake and suggest how the former data may be reconciled.

We have recently suggested a working hypothesis according to which CO₂ uptake by Cyanobacteria and its intracellular conversion to HCO₃⁻ may be energized by photosynthetic electron transport via the formation of alkaline domains on the stromal face of the thylakoid membrane (6). Catalyzed conversion of CO₂ to HCO₃⁻ in these domains would maintain an inward diffusion gradient for CO₂. Results presented are con-

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‡ To whom correspondence should be addressed: Dept. of Plant Sciences, The Hebrew Univ. of Jerusalem, 91904 Jerusalem, Israel. Tel.: 972-2-6585234; Fax: 972-2-6584463; E-mail: aaronka@vms.huji.ac.il.

† The abbreviations used are: CA, carbonic anhydrase; WCB, water channel blocker; PSI, photosystem I; PSII, photosystem II; ET, electron transport; chl, chlorophyll; IAC, iodoacetamide; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; DBMQ, 2,5-dimethyl-p-benzoquinone; MV, methyl viologen; CCCP, carbonyl cyanide m-chlorophenylhydrazone.
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**MATERIALS AND METHODS**

*Synechococcus* sp. PCC7942, *Synechocystis* PCC6803, and mutants thereof were grown on BG-11 medium (27) supplemented with 20 mM Hepes-NaOH, pH 8.0, and with 5 mM glucose in the case of the *Synechocystis* PCC6803 mutant ΔpsaA/B (28). The cultures were aerated with either high or low CO₂ concentration (5% CO₂ in air or 1:1 mixture of air and CO₂-free air, respectively), at 30 °C and light intensity of 100 μmol photons m⁻² s⁻¹. The cells were harvested during the log phase of growth and resuspended in growth medium.

Gas exchange measurements were performed with a membrane inlet mass spectrometer (Balzers QMG 421) as described earlier (7). Changes in CO₂ and O₂ concentration in the medium were given above the curves in panels A and B. Note that the slope of the curve relating external [CO₂(dis)] to time (panel B) cannot be taken as a direct indication of the initial rate of CO₂ removal by the cells because CO₂ is being formed continuously in the solution by net dehydration of HCO₃⁻. Moreover, even HCO₃⁻ concentration is virtually constant under the conditions of this experiment. Net dehydration rate is not constant but rises because of the decline in CO₂ hydration rate as [CO₂(dis)] drops. Consequently, the further [CO₂(dis)] deviates from chemical equilibrium with HCO₃⁻, the lower the rate of CO₂ hydration, and therefore the higher the rate of net dehydration. At plateaus in the curve, where the CO₂ concentrations are relatively constant, the net rate of CO₂ uptake by the cells will be equal to the net rate of CO₂ formation by dehydration of HCO₃⁻ in the medium.

**RESULTS**

Displacement of [CO₂(dis)] from Chemical Equilibrium upon Illumination—The extent of [CO₂(dis)] displacement from equilibrium was strongly affected by light intensity (Figs. 1 and 3). Raising the latter from 85 μmol photons m⁻² s⁻¹ (Fig. 1, panel B) to 750 μmol photons m⁻² s⁻¹ (panel C) led to an immediate drop in the ambient [CO₂(dis)] suggesting a higher rate of net CO₂ uptake. The rate of O₂ evolution also increased from 130 to 310 μmol O₂ mg⁻¹ Chl h⁻¹. Return to the light intensity of 85 μmol photons m⁻² s⁻¹ (panel D) caused the [CO₂(dis)] curve to rise again to a level higher than in the preceding light period at this intensity (compare panels B and D). The upward slope of the [CO₂(dis)] curve at high light intensity (Fig. 1, panel C) and the following downward slope at low light (panel D) most likely reflect changes in the light-driven ET rate via the photosystem due to adjustments in the efficiency of energy transfer from the phycobilisomes. Upon darkening (Fig. 1, panel E), the [CO₂(dis)] frequently rose transiently above that expected at equilibrium probably due to formation of CO₂ from HCO₃⁻ in the intracellular C pool and leak of the former to the medium (2). Note that in the second half of the period of high illumination (panel C), the rate of O₂ evolution rose as the ambient concentration of CO₂ increased, i.e. slower net CO₂ uptake. Moreover, while the ambient [CO₂(dis)] declined (during the second half of panel D) indicating a rising rate of CO₂ uptake, the rate of O₂ evolution remained constant. If CO₂ fixation accounted for alterations in [CO₂(dis)], one would have expected that changes in the CO₂ uptake curve to be the mirror image of those in the O₂ evolution curve, but that is not the case. These data provide supporting evidence for the conclusion that CO₂ uptake does not solely reflect CO₂ fixation and may occur even in its absence (5, 6).

**Dependence of CO₂ Uptake on Light Intensity**—To distinguish between the effects of light intensity on CO₂ uptake and on CO₂ fixation, we used the high-CO₂-requiring mutant of *Synechococcus* PCC7942, IL-3 (32) which maintains the [CO₂(dis)] below CO₂/HCO₃⁻ equilibrium even at CO₂ concentrations lower than its threshold for net CO₂ fixation (6). High-CO₂-grown cells of *Synechococcus* PCC 7942 and of mutant IL-3 were exposed to a range of light intensities in the membrane introduction mass spectrometry chamber. The cells were provided with 1 mM Ci, sufficient to saturate photosynthesis in the case of the wild type but too low to enable CO₂-dependent O₂ evolution in the case of the mutant. At light intensities below 200 μmol photons m⁻² s⁻¹, CO₂ uptake by IL-3 was considerably faster than in the wild type (Fig. 2). At higher light intensities, the rates of net CO₂ uptake by the mutant and *Synechococcus* PCC7942 were similar (Fig. 2, inset). The rates of net CO₂ uptake declined when cells of *Synechococcus* or mutant IL-3 were exposed to light intensity higher than 600
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**Inhibition of CO₂ Uptake by a WCB—**Addition of the WCB p-chloromercuriphenylsulfonic acid to a cell suspension of high-CO₂-grown *Synechococcus* PCC 7942 resulted in severe, almost complete, inhibition of net CO₂ uptake by over 90% (as calculated from the CO₂ concentration at the plateau attained after the addition of the WCB, Fig. 3A). Photosynthetic O₂ evolution was also severely depressed. To distinguish between a direct effect of the WCB on CO₂ uptake and a possible indirect effect due to the decline in CO₂ fixation, we applied iodoacetamide (IAC) that completely inhibits CO₂ fixation (5, 6, 29–31). We also made use of the high-CO₂-requiring mutant of *Synechococcus* PCC7942, IL-3 in which the light-saturated rate of CO₂ uptake is similar to that of its wild type (Fig. 2, inset) probably due to photoinhibition.

**Dependence of CO₂ Uptake on Photosynthetic Electron Trans-**

port via PSI—The functional linkage between photosynthetic ET and CO₂ uptake was examined with the aid of electron acceptors, donors, and electron transfer inhibitors. In addition to *Synechococcus* PCC7942 we also examined *Synechocystis* PCC6803, which exhibits a similar displacement of [CO₂(dis)] from equilibrium upon illumination and where mutants impaired in PSI activity are available. As previously reported (33), inhibition of linear electron flow by DCMU abolished CO₂ uptake (Fig. 4A). However, addition of duroquinol that donates electrons to plastoquinol and reduces cytochrome b6f thus priming light-driven PSI electron flow, reestablished CO₂ uptake but not CO₂ fixation (completely inhibited by DCMU). These results provide further evidence that generation of CO₂/HCO₃⁻ disequilibrium is not compulsorily linked to CO₂ removal by the carboxylation reactions but does require PSI activity (Fig. 4A).

**Energy Requirement for the C₄Cycling Activity—**To distinguish between ATP hydrolysis and ΔµH⁺ as the direct source of energy for CO₂ uptake, we have examined the effects of drugs that specifically inhibit ATP synthesis as well as uncouplers that dissipate the ΔµH⁺. Arsenate and DCCD inhibit the formation of ATP at the substrate level and proton gradient driven synthesis respectively while hardly affecting ΔµH⁺ (35, 36). The uncouplers CCCP and ammonia, on the other hand, abolish the generation of ΔµH⁺.

**Fig. 3. The effect of a water channel blocker on net CO₂ uptake by *Synechococcus* PCC 7942 and its mutant IL-3.** Cell density corresponded to 4 µg Chl/ml, and light intensity was 160 µmol photons m⁻² s⁻¹. Other conditions were as in Fig. 1. A, inhibition of net CO₂ uptake following the addition of the water channel blocker (WCB) p-chloromercuriphenylsulfonic acid (21) to *Synechococcus* PCC 7942. B, rates of net CO₂ uptake by *Synechococcus* PCC 7942 and mutant IL-3 following iodoacetamide (IAC, 3.3 mM provided in the dark 8 min prior to illumination) and of WCB treatments. The data are presented as a percentage of those exhibited by the wild type (270 µmol CO₂ mg⁻¹ Chl h⁻¹).

**μmol photons m⁻² s⁻¹ (Fig. 2, inset)** probably due to photoinhibition.
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Fig. 4. The effect of electron acceptors, donors, transfer inhibitors, and mutations in components of the photosynthetic electron transport chain on the extent of the light-induced displacement of [CO₂(dis)] from chemical equilibrium. A, inhibition by DCMU and alleviation of the inhibition by reduced duroquinone (DQH₂). DCMU and DQH₂ were added to final concentrations of 10⁻⁵ M and 2 × 10⁻³ M, respectively. The Synechococcus PCC 7942 cell density corresponded to 10 μg Chl/ml. B, inability of mutant ΔpsaA/B of Synechocystis PCC 6803 to displace [CO₂(dis)] from equilibrium even after addition of 2,5-dimethyl-p-benzoquinone (DMBQ), which accepts electrons from PSI. Cell density corresponded to 10 μg Chl/ml. C, inversion of CO₂/HCO₃⁻ disequilibrium after supply of DMBQ to Synechococcus PCC 7942 and return to equilibrium after supply of carbonic anhydrase (CA). Cell density corresponded to 15 μg Chl/ml. D, inversion of CO₂/HCO₃⁻ disequilibrium after supply of methyl viologen (MV) to Synechococcus PCC 7942, abolition of disequilibrium in the dark, recreation of disequilibrium in a subsequent light period, and return to equilibrium after supply of CA. Cell density corresponded to 8 μg Chl/ml.

54% (from 240 to 112 μmol CO₂ absorbed mg⁻¹ Chl h⁻¹). At the same time, net O₂ evolution (235 μmol O₂ evolved mg⁻¹ Chl h⁻¹) was replaced by respiratory O₂ uptake (67 μmol O₂ absorbed mg⁻¹ Chl h⁻¹). Supply of MV led to a slight rise in [CO₂(dis)] but the rate of increase was much lower than that observed in the absence of DCCD (Fig. 5B) possibly because of lack of sufficient ATP to drive HCO₃⁻ uptake (and consequently CO₂ extrusion). Subsequent CA supply sharply increased the [CO₂(dis)] again indicating that the latter was below the equilibrium value due to net CO₂ uptake. On the addition of the proton conductor CCCP, the rate of O₂ evolution rose transiently (Fig. 5C), most probably because of stimulation of electron transport due to dissipation of the trans-thylakoid ΔμH⁺. Photosynthetic O₂ evolution then ceased, but O₂ uptake at a rate similar to that of dark respiration was detectable. The [CO₂(dis)] rose almost immediately upon addition of CCCP, briefly overshooting equilibrium value. The [CO₂(dis)] level trace resembles that observed when photosynthetic ET is halted by darkening (see Fig. 1). Similar results were obtained using ammonium chloride (not shown).

The [CO₂(dis)] was below CO₂/HCO₃⁻ equilibrium even when the internal ATP was largely exhausted as indicated by the cessation of O₂ evolution following the arsenate or DCCD treatments (Fig. 5, A and B). On the other hand, the uncouplers abolished both CO₂ uptake and fixation (Fig. 5C). These data suggest that CO₂ uptake depends on a ΔμH⁺ and that direct involvement of ATP hydrolysis in the displacement of [CO₂(dis)] below equilibrium is therefore unlikely.

**DISCUSSION**

Severe inhibition of CO₂ uptake by the aquaporin blocker (Fig. 3) suggests that these channels form a major route for CO₂ entry to high-CO₂-grown Synechococcus cells. Because passage of CO₂ through aquaporins is presumably passive either by diffusion or by mass flow together with water molecules, CO₂ transport mediated by specific membrane entities would appear to play a minor role, if any, unless the aquaporin blocker also specifically inhibits these entities.

Results presented in this work confirm that net CO₂ uptake by Cyanobacteria is not directly linked to CO₂ fixation and may proceed in its absence (Figs. 1, 2, and 4). Under low, but not high light intensity, CO₂ uptake by mutant IL-3, which had been maintained at CO₂ concentration lower than its threshold for CO₂ fixation, was faster than in the wild type (Fig. 2). This may reflect consumption of NADPH and/or dissipation of ΔμH⁺ to support CO₂ fixation in the wild type. As discussed below, CO₂ uptake may well be driven by electron transport-dependent ΔμH⁺ (6). At saturating light intensity, net CO₂ uptake was similar in the wild type and in mutant IL-3 suggesting common limitation by the rate of conversion of CO₂ to HCO₃⁻.

In view of the queries recently raised as to the role of PSI as the major energy source for CO₂ uptake (24), we summarize the evidence in favor of this role obtained in the present investigation as follows. 1) CO₂/HCO₃⁻ disequilibrium consequent on net CO₂ uptake was formed even in the presence of DCMU when reduced duroquinone (Fig. 4A) or dithiothreitol (not shown) was added. 2) C₁ cycling was absent in the ΔpsaA/B mutant, lacking PSI activity even in the presence of DMBQ, which enabled a high flow of electrons via PSI (Fig. 4B); 3) Synechocystis PCC6803 mutant M55 or the Synechococcus PCC7942 mutant N5 in which ndhB had been inactivated (37, 38) are defective in cyclic PSI ET. Although they exhibit photosynthetic carbon fixation when supplied with elevated CO₂ levels (38), they are unable to displace the CO₂/HCO₃⁻ equilibrium. 4) Draining electrons from PSI by means of artificial acceptors switched Synechococcus PCC7942 from net CO₂ uptake to net HCO₃⁻ uptake (Fig. 4, C and D, see also Ref. 23).
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These observations together with those reported elsewhere (23, 25, 39) provide a very strong case for the central role of PSI ET in driving CO₂ uptake. The question is, therefore, how these observations can be reconciled with the contrasting effects of inactivation of NDH-1 components on CO₂ uptake and on P700 oxidation in *Synechocystis* PCC6803 (24). Another problem is that the results presented here indicate that a component of NDH-1, NdHB, critical for CO₂ uptake is exclusively located in the thylakoid membrane (25).

The possibility should be considered that only a small fraction of the PSI population is engaged in CO₂ uptake. Depression of CO₂ uptake in the ΔndhD3/ΔndhD4 mutant (24) was in fact associated with some decline in cyclic PSI activity, possibly reflecting this small fraction of PSI. In the ΔndhD1/ΔndhD2 mutant where cyclic PSI activity was largely depressed, CO₂ uptake was little affected presumably because the PSI units engaged in CO₂ uptake were still operating. The marked dependence of the PSI/PSII ratio on the growth conditions, particularly CO₂ concentration (22) and salinity (40), would be in accordance with such a suggestion. The observation that the distribution of PSI in *Synechococcus* PCC7942 is heterogeneous, indicated by the higher abundance of PSI in peripheral as compared with inner thylakoid membranes (41), also lends support to the notion that the PSI population may be functionally heterogeneous. This heterogeneity might relate to different routes for electron flow from NADPH to plastoquinone (42, 43).

Different routes for electron flow might also be the basis of the differences in CO₂ uptake between the various *ndhD* mutants (24). Uptake of CO₂ was observed in each of the single mutants ΔndhD3 and ΔndhD4 but not in the double mutant (24). Ogawa et al. (25) concluded that two discrete systems for CO₂ uptake operate in *Synechococcus* PCC6803. This is based on the differing kinetic parameters for CO₂ uptake between mutants ΔndhD4 and ΔndhD3 and the inducibility of CO₂ uptake by low CO₂ conditions in the wild type and in mutant ΔndhD4 but not in mutant ΔndhD3. The NdhD3- and NdhD4-dependent CO₂ uptake systems may constitute alternative PSI-dependent routes for electron flow. Quantitative consideration shows that the residual rate of P700 oxidation (i.e. the PSI cyclic electron flux) in the ΔndhD1/ΔndhD2 (24) was too low to account for the rate of CO₂ uptake in this mutant. This may indicate the presence of an alternative acceptor of electrons from the NdhD3- and NdhD4-dependent CO₂ uptake systems such as succinate:plastoquinone oxidoreductases (44), a possibility currently being examined.

It has recently been proposed (6) that CO₂ is converted to HCO₃⁻ in alkaline domains on the stromal face of the thylakoid membrane (see the Introduction). Conversion of CO₂ to HCO₃⁻ in such domains maintains the inward diffusion gradient for CO₂ and a cytoplasmic CO₂ concentration below that of equilibrium with HCO₃⁻. Withdrawal of electrons from plastoquinone by either DMBQ or MV (Fig. 4) would prevent the formation of these alkaline domains and thus also of CO₂ uptake. The exclusive reliance of CO₂ uptake on PSI-generated ΔµH⁺ probably involves an electron carrier yet to be identified. In view of the finding that NdhD3 and NdhD4 are essential components of two CO₂ uptake systems (Ogawa et al., submitted) but not of the respiratory electron path they are likely candidates for the formation of the alkaline domains during PSI ET. Another candidate is ferredoxin-NADPH reductase. Recent studies (43) demonstrated that linear ET was functional but that the plastoquinone-cytochrome b6f complex reductase step of cyclic PSI was defective in a mutant where the N-terminal of ferredoxin-NADPH reductase was truncated preventing its association with the thylakoids).

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**Fig. 5.** *A, the effect of arsenate (As), B, N,N,N-dicyclohexylcarbodiimide (DCCD), and C, carbonyl cyanide m-chlorophenylhydrazone (CCCP) on the displacement of external [CO₂] from chemical equilibrium and on O₂ evolution by *Synechococcus* PCC 7942.* On addition of CA external [CO₂] returned to equilibrium. Cell density corresponded to 10⁵ µg Chl/ml. Light intensity (in µmol photons m⁻² s⁻¹) is provided at the top.

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et al., (26) suggested that in *Synechococcus* PCC7002 one type of NDH-1 essential for cyclic ET is located on the thylakoid, whereas another type engaged in CO₂ uptake is located on the cytoplasmic membrane. However, immunolocalization studies have demonstrated that a component of NDH-1, NdHB, critical for CO₂ uptake is exclusively located in the thylakoid membrane (25).

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