Biochimica et Biophysica Acta, 423 (1976) 303–312

BBA 47037

EFFECT OF $NADP^+$ ON LIGHT-INDUCED CYTOCHROME CHANGES IN MEMBRANE FRAGMENTS FROM A BLUE-GREEN ALGA*

HARRY Y. TSUJIMOTO, BERAH D. McSWAIN, TETSUO HIYAMA and DANIEL I. ARNON
Department of Cell Physiology, University of California, Berkeley, Calif. 94720 (U.S.A.)
(Received July 17th, 1975)

SUMMARY

The effect of $NADP^+$ on light-induced steady-state redox changes of membrane-bound cytochromes was investigated in membrane fragments prepared from the blue-green algae *Nostoc muscorum* (Strain 7119) that had high rates of electron transport from water to $NADP^+$ and from an artificial electron donor, reduced dichlorophenolindophenol ($DCIPH_2$) to $NADP^+$. The membrane fragments contained very little phycocyanin and had excellent optical properties for spectrophotometric assays. With $DCIPH_2$ as the electron donor, $NADP^+$ had no effect on the light-induced redox changes of cytochromes: with or without $NADP^+$, 715- or 664-nm illumination resulted mainly in the oxidation of cytochrome *f* and of other component(s) which may include a *c*-type cytochrome with an α peak at 549 nm. With 664 nm illumination and water as the electron donor, $NADP^+$ had a pronounced effect on the redox state of cytochromes, causing a shift toward oxidation of a component with a peak at 549 nm (possibly a *c*-type cytochrome), cytochrome *f*, and particularly cytochrome b_{559}. Cytochrome b_{559} appeared to be a component of the main noncyclic electron transport chain and was photooxidized at physiological temperatures by Photosystem II. This photooxidation was apparent only in the presence of a terminal acceptor ($NADP^+$) for the electron flow from water.

INTRODUCTION

The role of the cytochrome components of the photosynthetic apparatus in light-induced electron transport has been the subject of much speculation and investigation. The early hypothesis of Hill and Bendall [1] envisaged cytochromes b_6 and *f* as electron carriers in noncyclic electron flow from water to NADP. In more recent formulations (see reviews, refs 2–5) cytochrome b_6 is linked to cyclic electron flow and noncyclic electron flow is thought to involve, in addition to cytochrome *f*, cytochrome

Abbreviations: DCMU, 3-(3′,4′-dichlorophenyl)-1,1-dimethylurea; DCIP, $DCIPH_2$, oxidized and reduced forms of 2,6-dichlorophenolindophenol.

* Paper III in the series "Photochemical Activity and Components of Membrane Preparations from Blue-Green Algae".

b_{559}, which is now recognized as the most abundant *b*-type cytochrome in chloroplasts [6–9].

Cytochrome *f* is generally regarded as an electron carrier in the photoreduction of $NADP^+$, either by the physiological electron donor (i.e., water) or by a non-physiological substitute (e.g., $DCIPH_2$) [3–5] but with respect to cytochrome b_{559} there is a wide range of views occasioned by the diversity and complexity of recent findings. Cytochrome b_{559} exists in chloroplasts in different forms (distinguished by different redox potentials) [10–14] which appear to have different physiological functions. In freshly prepared chloroplasts and at physiological temperatures the high-potential form of cytochrome b_{559} is the predominant one and is strongly correlated with Photosystem II activity [11]; the low-potential form is associated with Photosystem I [15]. Cytochrome b_{559} is the sole cytochrome present in chloroplast fragments that have only Photosystem II "reaction center" activity [16, 17]. This close association with Photosystem II is further supported by the now well-established fact that the high-potential form of cytochrome b_{559} is photooxidized at cryogenic temperatures (−189 °C or lower) only by Photosystem II light; far-red illumination that activates Photosystem I is ineffective (literature cited in ref. 18). It should also be noted that cytochrome b_{559} is now known to be the only chloroplast cytochrome that is photooxidized at cryogenic temperatures.

The wide agreement that at cryogenic temperatures the photooxidation of cytochrome b_{559} involves only Photosystem II is in marked contrast to the disagreement about the photooxidation of this cytochrome at physiological temperatures. Different laboratories were unable to observe the photooxidation of cytochrome b_{559} at physiological temperatures without some special treatment or fractionation of chloroplasts (e.g. refs 19–24). Special treatments of chloroplasts gave conflicting results. For example, Hind [24] observed that a treatment of chloroplasts with carbonyl cyanide *m*-chlorophenylhydrazone induced a photooxidation of cytochrome b_{559} by Photosystem I light but Hiller et al. [22] found that this treatment induced photooxidation of cytochrome b_{559} by either Photosystem II or Photosystem I light. The work of this laboratory at physiological temperatures revealed a photooxidation of cytochrome b_{559} by Photosystem II light only when the photoreduction by water was prevented by Tris treatment of chloroplasts [19–21].

The experimental difficulties of measuring cytochrome b_{559} at physiological temperatures in untreated chloroplasts prompted us to use other material for investigating the role of this and other cytochromes in photosynthetic electron transport. Especially promising were membrane fragments from the blue-green alga *Nostoc muscorum* (Strain 7119) that retain complete Photosystem II and Photosystem I electron transport activity [25, 25a]. Blue-green algae contain basically the same *b*-type cytochromes as those found in chloroplasts, i.e., cytochrome b_6 and high- and low-potential forms of cytochrome b_{559} [18, 26–28] but more than one form of cytochromes of the *c* type: in addition to the cytochrome *f* (c_{554}) present in chloroplasts, blue-green algae contain a major (c_{549}) and a minor (c_{552}) *c*-type cytochrome component [27–30]. The *b*-type cytochomes were found to be tightly bound to membrane fragments [18, 18a, 26–28], whereas the *c*-type cytochromes were more or less readily solubilized [27–30].

A report on cytochrome changes in a preparation of algal membrane fragments that still retained appreciable amounts of phycocyanin (Fraction A, ref. 25) has

been published [31]. The membrane fragments used here (Fraction C, ref. 25) were practically free of phycocyanin and formed a suspension of very low turbidity that was exceptionally favorable for spectrophotometric assays at physiological temperatures of redox changes in membrane-bound cytochromes, including cytochrome b_{559}.

The effect of $NADP^+$ on absorbance changes in the α-region of cytochromes was measured at physiological temperatures under two conditions: first, when water was the electron donor for $NADP^+$ reduction and second, when $DCIPH_2$ replaced water as the electron donor for $NADP^+$ reduction [32]. With $DCIPH_2$ as the electron donor, cytochromes were photooxidized about equally by either 664- or 715-nm monochromatic light, regardless whether $NADP^+$ was present or not. By contrast, with water as the electron donor, 664-nm illumination induced a markedly more oxidized steady state of the membrane-bound cytochromes (particularly cytochrome b_{559}) but only when $NADP^+$ was present as a terminal electron acceptor.

METHODS

The membrane fragments used in these experiments consisted of Fraction C prepared from *Nostoc muscorum* cells (Strain 7119) grown in an N_2-CO_2 atmosphere with N_2 serving as the sole source of nitrogen [25, 25a]. Changes in absorption were measured at room temperature under nitrogen with an Aminco Model DW-2 spectrophotometer operated in the dual-wavelength mode. The reference wavelength was 540 nm. Actinic illumination was provided by monochromatic light beams (from a 150-watt tungsten-halogen lamp, Type FCS) with the aid of Baird-Atomic interference filters having half-band widths of 20 nm. Chlorophyll *a* was determined [33] and ferredoxin and ferredoxin-NADP reductase were isolated and purified from spinach by procedures previously reported from this laboratory [34, 35].

RESULTS

Effect of $NADP^+$ on cytochrome changes in the presence of an artificial electron donor

Before dealing with changes in the steady-state redox levels of cytochromes that accompanied the photoreduction of $NADP^+$ with water we examined changes that occurred in cytochromes when $NADP^+$ was photoreduced by the artificial electron donor $DCIPH_2$ (i.e., by small amounts of DCIP kept in the reduced state by large amounts of ascorbate); the reaction mixture contained the inhibitor DCMU to block electron flow from water. The experiments were divided into two parts. First, we measured the chemical reduction of cytochromes by $DCIPH_2$ before the light was turned on. Secondly, we measured the effect on the chemically reduced cytochromes of 664-nm and 715-nm monochromatic illumination in the presence and absence of the $NADP^+$ electron acceptor system ($NADP^+$, ferredoxin, and ferredoxin-$NADP^+$ reductase).

The spectrum of the chemical reduction of cytochromes by $DCIPH_2$ (or ascorbate) in the dark is shown in Fig. 1. The major peak around 559 nm, which varied in magnitude from preparation to preparation, is accounted for by a combination of reduced cytochrome b_{559} and cytochrome *f*. The minor peak at 548 nm, which also varied in magnitude in different preparations, cannot be identified with

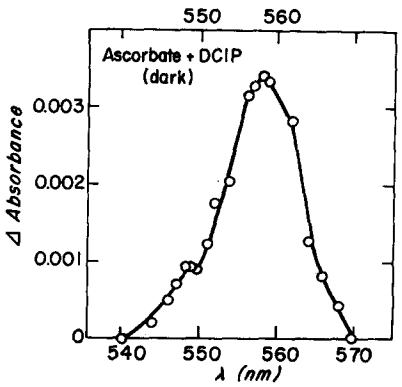

Fig. 1. Spectrum of absorbance changes observed in *Nostoc* membrane fragments following the addition of ascorbate and DCIP. Each point of the spectrum was determined with a fresh sample; reference wavelength, 540 nm. The reaction mixtures were placed in cuvettes (1-cm light path) and contained (per 1.0 ml) membrane fragments (Fraction C, ref. 25) equivalent to 50 μg chlorophyll *a* and the following: Tricine [*N*-Tris(hydroxymethyl)methylglycine] buffer (pH 7.7), 50 μmol; $MgCl_2$, 10 μmol; and DCMU, 1 nmol. After the baseline was determined, a mixture of ascorbate and DCIP was added to give final concentrations of 2 mM and 10 μM, respectively. The extra volume, added with a microburette, was 7 μl per ml. Gas phase, nitrogen; temperature, 20 °C.

certainty. It cannot be attributed to C-550, a component that is not reduced by $DCIPH_2$ (or ascorbate). Moreover, reduction of C-550 appears as a decrease rather than an increase in absorbance at or near 550 nm [36–39]. The peak at 548 nm is suggestive of the reduced state of a *c*-type cytochrome but positive identification must be deferred because the corresponding peak in the Soret region could not be established because of interfering absorption from cytochrome *f*. In our present state of knowledge, it is difficult to attribute this peak to the reduction of cytochrome c_{549} because the midpoint redox potential of this cytochrome, as isolated from *Anacystis nidulans* by Holton and Myers [30] and from the *Nostoc* membrane fragments used here (K. Ando and D. I. Arnon, unpublished data), is much lower than that of $DCIPH_2$ or ascorbate.

The absorbance changes induced by subsequent illumination of the membrane fragments that were reduced in the dark by $DCIPH_2$ are shown in Fig. 2. Illumination by either 664- or 715-nm monochromatic light produced a decrease in absorbance that was not influenced by the presence or absence of the $NADP^+$ electron acceptor system. The spectrum of the decreased absorbance showed a major peak around 555 nm, pointing to cytochrome *f* as the major component that was photooxidized. There was very little change in absorbance near 560 nm, indicating that neither the chemically reduced cytochrome b_{559} (Fig. 1) nor cytochrome b_6 was photooxidized. A distinct shoulder near 550 nm was again suggestive of some photooxidation of the same component that, on chemical reduction, showed an increase in absorbance near 549 nm (Fig. 1).

In sum, when $DCIPH_2$ replaced water as the electron donor, cytochrome b_{559} (which was prereduced in the dark) did not appear to undergo any measurable photooxidation by either 715- or 664-nm light, whether $NADP^+$ was present or not. Neither did $NADP^+$ have any effect on the cytochromes that were photooxidized in the presence of $DCIPH_2$. Cytochrome *f* and, possibly, another *c*-type cytochrome

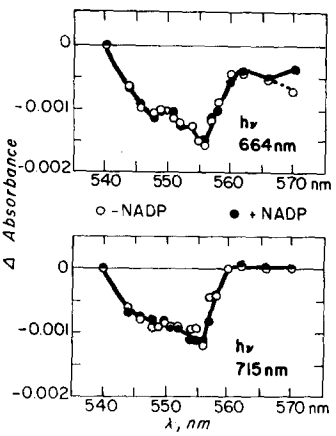

Fig. 2. Effect of $NADP^+$ on the spectrum of light-induced absorbance changes in *Nostoc* membrane fragments prereduced with ascorbate-DCIP. Reaction mixtures (including ascorbate and DCIP) were as in Fig. 1 except that samples with $NADP^+$ (● — ●) contained in addition 2 μmol of $NADP^+$, 25 nmol of ferredoxin, and saturating amounts of ferredoxin-$NADP^+$ reductase (per 1.0 ml). The incident monochromatic light intensities of the 664- and 715-nm light beams were $3.6 \cdot 10^4$ and $1.2 \cdot 10^4$ ergs/cm^2/s, respectively. Other conditions were as in Fig. 1.

were photooxidized by either long- or short-wavelength monochromatic light, whether $NADP^+$ was present or not.

To check on the possibility that the failure of $NADP^+$ to affect the photooxidation of cytochromes might have resulted from their rapid concurrent reduction by $DCIPH_2$, a comparison was made with a similar system which contained DCMU and ascorbate but no DCIP. The results were the same as when DCIP was included (Fig. 2).

Effect of $NADP^+$ on cytochrome changes associated with electron flow from water

The effect of $NADP^+$ on cytochrome changes associated with the electron flow from water were investigated under different experimental conditions. In one set of experiments, cytochromes b_{559} and f were first reduced in the dark by ascorbate or hydroquinone (ascorbate alone, without DCIP, could not serve as an effective electron donor for the photoreduction of $NADP^+$). Next, the effect of $NADP^+$ on the light-induced oxidation of cytochromes was measured either under illumination by 664-nm monochromatic light, capable of activating Photosystem II and sustaining a vigorous electron flow from water to $NADP^+$, or under illumination by 715-nm monochromatic light, which activated predominantly Photosystem I and was incapable of sustaining a vigorous electron flow from water to $NADP^+$.

As shown in Fig. 3, 715-nm illumination produced a decrease in absorbance, most pronounced around 555 nm, that was indicative of photooxidation of cytochromes f and b_{559}; but this photooxidation was unaffected by the presence or absence of $NADP^+$. The absorption changes produced by 664-nm illumination differed from those produced by 715-nm illumination in being strongly influenced by the presence of the $NADP^+$ electron acceptor system. In the absence of the $NADP^+$ system, 664-nm produced a small and rather featureless decrease in absorption – an indication that without a terminal electron acceptor the oxidizing power of 664-nm

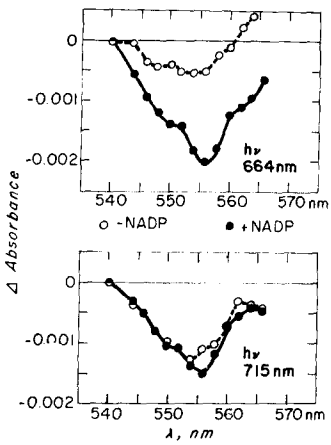

Fig. 3. Effect of $NADP^+$ on the spectra of light-induced absorbance changes in *Nostoc* membrane fragments prereduced with ascorbate. Reaction mixtures and conditions were as in Fig. 2 except that DCMU and DCIP were omitted.

illumination was almost completely balanced by its reducing power generated by the photoactivated electron flow from water. By contrast, in the presence of the $NADP^+$ system, the oxidizing power of 664-nm illumination was greatly enhanced and produced, on balance, a pronounced photooxidation of the cytochromes (Fig. 3). The decrease in absorbance due to photooxidation had a broad peak at 556 nm, indicative of the photooxidation of cytochrome b_{559} and of cytochrome *f*. The shoulder at 549 nm again suggests the photooxidation of a possible cytochrome *c*-type component.

Next, the $NADP^+$-dependent photooxidation of cytochromes by 664-nm light was further investigated by omitting ascorbate from the reaction mixture. Of special interest was the question whether the algal membrane fragments, unlike chloroplasts, could yield clear-cut evidence for the photooxidation of cytochrome b_{559}

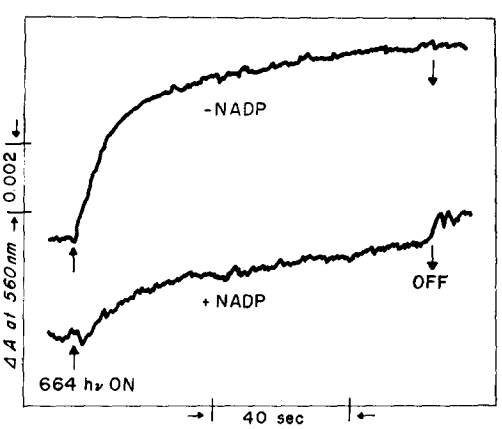

Fig. 4. Traces of absorbance changes at 560 nm induced by 664-nm illumination in *Nostoc* membrane fragments in the presence and absence of NADP. Reaction mixtures and other conditions were as in Fig. 2 except that ascorbate, DCIP, and DCMU were omitted.

by 664-nm light without special treatments to impede electron flow from water. Absorbance changes induced by 664-nm light in the presence or absence of $NADP^+$ were therefore measured first at 560 nm, a wavelength sufficiently removed from the α peak of *c*-type cytochromes to permit unequivocal observations of redox changes in cytochrome b_{559}.

As shown by the traces in Fig. 4, a marked increase in absorbance at 560 nm, indicative of photoreduction of cytochrome b_{559}, was produced by 664-nm illumination in the absence of $NADP^+$. However, in the presence of $NADP^+$, the 664-nm illumination shifted cytochrome b_{559} toward a markedly more oxidized state.

The measurements illustrated in Fig. 4 were extended to cover the range of 540 to 570 nm. The resultant absorption spectra, the magnitude (but not the character) of which varied from preparation to preparation, are shown in Fig. 5a. The difference between the two spectra, one in the presence and one in the absence of $NADP^+$, is presented in Fig. 5b.

The difference spectrum in Fig. 5b, with its major negative peak near 559 nm, indicates that a light-induced electron transport from water to $NADP^+$ is accompanied by a large shift toward oxidation in the steady-state redox level of cytochrome b_{559}. In addition, the shoulder near 554 nm and the secondary negative peak near 549 nm suggests a similar shift in cytochrome *f* and possibly in another *c*-type cytochrome.

In the experiments represented by Figs. 5a and 5b, the effect of $NADP^+$ on shifting the light-induced redox state of the cytochromes toward oxidation was demonstrated by comparing the redox state of the cytochromes in two separate cuvettes, one containing and one lacking $NADP^+$. Similar results were obtained in other experiments (not included here) in which the shift toward oxidation caused by $NADP^+$ was demonstrated directly in one cuvette, i.e., by recording first the photoreduction (increase in absorbance) of cytochromes by 664-nm light in the absence of

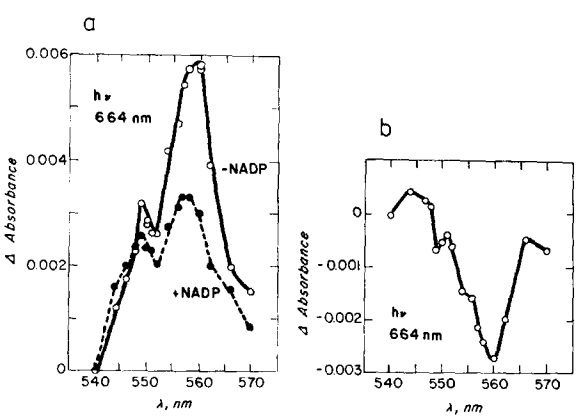

Fig. 5. a. Light-induced absorption spectra in the presence and absence of electron transport from water to $NADP^+$. Reaction mixtures and conditions were as in Fig. 2 except that ascorbate, DCIP, and DCMU were omitted and only 664-nm monochromatic illumination was used throughout. b. Difference spectrum showing the shift toward oxidation of cytochromes as a result of electron transport from water to $NADP^+$. The individual points were obtained by subtracting the $-NADP^+$ spectrum from the $+NADP^+$ spectrum in Fig. 5a.

$NADP^+$ and then adding $NADP^+$ (without turning off the light) and recording the resultant photooxidation of cytochromes (decrease in absorbance).

DISCUSSION

The initial purpose of this study was to measure light-induced steady-state redox changes of membrane-bound cytochromes, particularly cytochrome b_{559}, at physiological temperatures and without special treatments that impair the capacity for complete photosynthetic electron transport from water to $NADP^+$. This aim was realized by using a preparation of *Nostoc* membrane fragments that had complete and stable Photosystem I and Photosystem II electron transport activities as evidenced by high rates of $NADP^+$ reduction by either water or $DCIPH_2$ (but low rates of phosphorylation, see ref. 25) and that had optical properties that were especially favorable for spectrophotometric assays. The absorbance changes due to cytochrome b_{559} were larger than we have ever observed at physiological temperatures with any other photosynthetic system.

The light-induced cytochrome changes were of two kinds. One kind, observed when $DCIPH_2$ was the reductant and the electron flow from water was severely impeded or completely blocked by the use of 715-nm light (with or without DCMU) or by 664-nm light in the presence of DCMU, was not affected by the addition of $NADP^+$ (Fig. 2 and Fig. 3, lower part). Under these conditions, illumination induced absorbance changes that pointed in the main to the photooxidation of cytochrome *f*. There was very little change in absorbance around 560 nm, indicating that neither the high-potential form of cytochrome b_{559} (prereduced in the dark by DCIP-ascorbate) nor cytochrome b_6 were being photooxidized. That the high-potential form of cytochrome b_{559} was not photooxidized we consider significant; the autooxidizable cytochrome b_6, as well as the autooxidizable low-potential form of cytochrome b_{559} [18, 15], were probably already in the oxidized state prior to illumination.

It seems reasonable to ascribe the light-induced cytochrome changes that were not affected by the presence of $NADP^+$ to a cyclic electron flow system, activated by Photosystem I, involving cytochromes *f*, b_6 (see reviews, refs 3, 5), and probably the low-potential form of cytochrome b_{559} [15]. Since neither the *b*-type cytochromes nor cytochrome *f* (as observed here and in earlier investigations [40, 31]) changed their redox state when $NADP^+$ was photoreduced by $DCIPH_2$, it is likely that no cytochrome serves as an electron carrier in this photoreduction. Work is underway to determine whether in *Nostoc* membrane fragments electrons from reduced DCIP enter the photosynthetic apparatus via plastocyanin or *P*-700 (cf. refs 3, 4).

The second kind of light-induced cytochrome change was strongly influenced by the presence of $NADP^+$ as the terminal electron acceptor and was observed only under 664-nm illumination that activated an electron flow from water. This noncyclic electron flow from water to $NADP^+$ was accompanied by a shift of cytochrome b_{559} from a predominantly reduced state to a distinctly more oxidized state (Fig. 5a and b). In addition, there was evidence for the photooxidation of cytochrome *f* and possibly of another *c*-type cytochrome with an α peak around 549 nm, although more evidence is needed to substantiate the latter conclusion. Thus, in *Nostoc* membrane fragments, cytochrome *f* appears to be involved both in noncyclic and cyclic electron transport (cf. ref. 41). (Similar effects of a terminal electron acceptor were reported by Avron

and Chance [40] for cytochrome *f* in chloroplasts and by Knaff [31] for cytochromes b_{559} and *f* in another preparation of *Nostoc* membrane fragments.)

The results of this investigation bear on two questions that are important for the assessment of the physiological role in photosynthesis of the high-potential form of cytochrome b_{559} (E_m = +0.35 V at pH 7), the predominant form in chloroplasts [11] and in our membrane preparations (cf. ref. 18): (a) is cytochrome b_{559} an electron carrier in the main noncyclic electron transport chain between water and $NADP^+$; and (b) is cytochrome b_{559} at physiological temperatures photooxidized by Photosystem II or by Photosystem I? As for the first question, several investigators concluded that cytochrome b_{559} is not a component of the main noncyclic electron transport chain but that it is located on a side path from Photosystem II with an additional link to Photosystem I [22, 23, 42, 43]. Other investigators, however, consider cytochrome b_{559} as an electron carrier that is situated between Photosystem II and Photosystem I on the main noncyclic electron path from water to $NADP^+$ [9, 31, 44–46]. A similarly wide diversity of views exists as to whether Photosystem II or Photosystem I photooxidizes cytochrome b_{559} at physiological temperatures: some assign this function to both Photosystem II and Photosystem I [22, 26], some to Photosystem II only [20, 21, 47, 48], and some to Photosystem I only [9, 31, 44–46].

In this investigation, the essential conditions for observing a pronounced and consistent photooxidation of cytochrome b_{559} at physiological temperatures were 664-nm illumination, the presence of $NADP^+$, and the use of *Nostoc* membrane fragments that had the capacity for a vigorous noncyclic electron flow from water to $NADP^+$. Although not bearing on the question of two light reactions in Photosystem II, these findings are nonetheless consistent with the concept [20, 49, 47] that cytochrome b_{559} is normally photooxidized by Photosystem II and is an electron carrier in the direct noncyclic electron transport chain from water to $NADP^+$. Photosystem I was ineffective in photooxidizing cytochrome b_{559}; little photooxidation of this cytochrome (prereduced by ascorbate) was brought about by 715-nm light or by 664-nm light in the presence of DCMU. The argument [50] that photooxidation of cytochrome b_{559} by Photosystem II occurs only under special conditions (such as low temperature or Tris treatment) that stop the normal path of electron transport from water to the oxidant of Photosystem II does not apply to our experiments with *Nostoc* membrane fragments, in which photooxidation of cytochrome b_{559} was observed at physiological temperatures during active electron transport from water to $NADP^+$.

ACKNOWLEDGEMENTS

This investigation was supported in part by NSF Grant BMS 71-01204 to one of us (D.I.A.). We thank Donald E. Carlson, Jr. for excellent assistance in maintaining the *Nostoc* algal cultures and preparing the Fraction C membrane fragments.

REFERENCES

1 Hill, R. and Bendall, F. (1960) Nature 186, 136–137
2 Bendall, D. S. and Hill, R. (1968) Annu. Rev. Plant Physiol. 19, 167–186
3 Boardman, N. K. (1968) Adv. Enzymol. 31, 1–79
4 Bishop, N. I. (1971) Annu. Rev. Biochem. 40, 197–226

5 Trebst, A. (1974) Annu. Rev. Plant Physiol. 25, 423–458
6 Lundegårdh, H. (1962) Physiol. Plant. 15, 390–398
7 Boardman, N. K. and Anderson, J. (1967) Biochim. Biophys. Acta 143, 187–203
8 Arnon, D. I., Tsujimoto, H. Y., McSwain, B. D. and Chain, R. K. (1968) in Comparative Bio chemistry and Biophysics of Photosynthesis (Shibata, K., Takamiya, A., Jagendorf A. T. an Fuller, R. C., eds), Univ. Park Press, State College Pa, pp. 113–132
9 Cramer, W. A. and Butler, W. L. (1967) Biochim. Biophys. Acta 143, 332–339
10 Bendall, D. S. (1968) Biochem. J. 109, 46p
11 Wada, K. and Arnon, D. I. (1971) Proc. Natl. Acad. Sci. U.S. 68, 3064–3068
12 Erixon, K., Lozier, R. and Butler, W. L. (1972) Biochim. Biophys. Acta 267, 375–382
13 Cramer, W. A., Fan, H. N. and Böhme, H. (1971) Bioenergetics 2, 289–303
14 Cox, R. P. and Bendall, D. S. (1972) Biochim. Biophys. Acta 283, 124–135
15 Anderson, J. M. and Boardman, N. K. (1973) FEBS Lett. 32, 157–160
16 Ke, B., Vernon, L. P. and Chaney, T. H. (1972) Biochim. Biophys. Acta 256, 345–357
17 Wessels, J. S. C., Van Alphen-Van Waveren, O. and Boorn, G. (1973) Biochim. Biophys. Acta 292, 741–752
18 Aparicio, P. J., Ando, K. and Arnon, D. I. (1974) Biochim. Biophys. Acta 357, 246–251
18a Aparicio, P. J., Ando, K. and Arnon, D. I. (1974) Biochim. Biophys. Acta 368, 459
19 Knaff, D. B. and Arnon, D. I. (1969) Proc. Natl. Acad. Sci. U.S. 63, 956–962
20 Knaff, D. B. and Arnon, D. I. (1969) Proc. Natl. Acad. Sci. U.S. 64, 715–722
21 Knaff, D. B. and McSwain, B. D. (1971) Biochim. Biophys. Acta 245, 105–108
22 Hiller, R. G., Anderson, J. M. and Boardman, N. K. (1971) Biochim. Biophys. Acta 245, 439–452
23 Fork, D. C. (1972) Biophys. J. 12, 909–921
24 Hind, G. (1968) Photochem. Photobiol. 7, 369–375
25 Arnon, D. I., McSwain, B. D., Tsujimoto, H. Y. and Wada, K. (1974) Biochim. Biophys. Acta 357, 231–245
25a Arnon, D. I., McSwain, B. D., Tsujimoto, H. Y. and Wada, K. (1974) Biochim. Biophys. Acta 368, 459
26 Fujita, Y. (1974) Plant Cell Physiol. 15, 861–874
27 Biggins, J. (1967) Plant Physiol. 42, 1447–1456
28 Ogawa, T. and Vernon, L. P. (1971) Biochim. Biophys. Acta 226, 88–97
29 Holton, R. W. and Myers, J. (1967) Biochim. Biophys. Acta 121, 362–374
30 Holton, R. W. and Myers, J. (1967) Biochim. Biophys. Acta 131, 375–384
31 Knaff, D. B. (1973) Biochim. Biophys. Acta 325, 284–296
32 Vernon, L. P. and Zaugg, W. S. (1960) J. Biol. Chem. 235, 2728–2733
33 Arnon, D. I. (1949) Plant Physiol. 24, 1–15
34 Losada, M. and Arnon, D. I. (1964) in Modern Methods of Plant Analysis (Linskens, H. W., Sanwal, D. B. and Tracey, M. V., eds.), Vol. 7, pp. 569–615, Springer-Verlag, Berlin
35 Shin, M., Tagawa, K. and Arnon, D. I. (1963) Biochem. Z. 338, 84–96
36 Knaff, D. B. and Arnon, D. I. (1969) Proc. Natl. Acad. Sci. U.S. 63, 963–969
37 Erixon, K. and Butler, W. L. (1971) Biochim. Biophys. Acta 234, 381–389
38 Boardman, N. K., Anderson, J. M. and Hiller, R. G. (1971) Biochim. Biophys. Acta 234, 126–136
39 Bendall, D. S. and Sofrova, D. (1971) Biochim. Biophys. Acta 234, 371–380
40 Avron, M. and Chance, B. (1966) in Currents in Photosynthesis, pp. 455–463, Donker Publ., Rotterdam
41 Biggins, J. (1973) Biochemistry 12, 1165–1170
42 Satoh, K. and Katoh, S. (1972) Plant Cell Physiol. 13, 807–820
43 Satoh, K., Yakushiji, A. and Katoh, S. (1973) Plant Cell Physiol. 14, 763–767
44 Levine, R. P. and Gorman, D. S. (1966) Plant Physiol. 41, 1293–1300
45 Ikegami, I., Katoh, S. and Takamiya, A. (1970) Plant Cell Physiol. 11, 777–791
46 Ben-Hayyim, G. and Avron, M. (1970) Eur. J. Biochem. 14, 205–213
47 Huzisige, H. and Takimoto, N. (1974) Plant Cell Physiol. 15, 1099–1113
48 Amesz, J., Pulles, M. P. J., Visser, J. W. M. and Sibbing, F. A. (1972) Biochim. Biophys. Acta 275, 442–452
49 Arnon, D. I., Knaff, D. B., McSwain, B. D., Chain, R. K. and Tsujimoto, H. Y. (1971) Photochem. Photobiol. 14, 397–425
50 Erixon, K. and Butler, W. L. (1971) Photochem. Photobiol. 14, 427–433