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Inhibition by plastoquinone analogues of ferricyanide reduction by a Photosystem II chloroplast preparation

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There is wide agreement that inhibition of plastoquinone function by plastoquinone analogues, of which the best known is dibromothymoquinone (DBMIB), inhibits electron transport from Photosystem II (PS II) to Photosystem I (PS I), but does not inhibit electron transport that is dependent on PS II alone. In contrast, we have recently presented evidence in support of a hypothesis for a hitherto unrecognized additional role of plastoquinone in the conductance of protons derived from the photooxidation of water by PS II (Arnon, D.I. and Tang, G.M.-S. (1985) Biochim. Biophys. Acta 809, 167–172). Since the transport of water-derived electrons and protons is coupled, this hypothesis predicts that photosynthetic electron transport by PS II alone would still be sensitive to DBMIB inhibition. We now report that this prediction has been verified by obtaining inhibition by low concentrations of DBMIB of electron transport from water to ferricyanide in a PS II preparation depleted of plastocyanin and PS I. The preparation consisted of inside-out vesicles isolated by the aqueous polymer two-phase partition method. The pattern of DBMIB inhibition in the PS II preparation was the same as that in unfractionated chloroplasts: inhibition of ferricyanide reduction was relieved by the addition of catalytic amounts of one of several lipophilic acceptors that appear to bypass the site of DBMIB inhibition at the Rieske FeS center of the cytochrome f/b_6 complex. The bearing of these findings on the role of plastoquinones in the conductance of protons released by the photooxidation of water is discussed.

Introduction

Current concepts of the photosynthetic role of plastoquinone, the most abundant redox component of chloroplast membranes [1,2], have evolved to a large extent from work with plastoquinone analogues, used to inhibit either the oxidation or reduction of plastoquinone. The most widely used analogue is DBMIB, which at low concentrations inhibits at the site of plastoquinone oxidation [3–5]. Such DBMIB inhibition now constitutes prima facie evidence for involvement of the plastoquinone pool in photosynthetic electron transport [3]. Among other plastoquinone analogues is 5-*n*-undecyl-6-hydroxy-4,7-dioxobenzothiazole (UHDBT) which at low concentrations (< 1 μM) inhibits at the site of plastoquinone reduction [6–8].

The currently prevalent view, embodied in the Z scheme, holds that the role of plastoquinone is to transport to PS I (via the FeS-cytochrome f/b_6 complex and plastocyanin) electrons liberated in

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Abbreviations: PS, Photosystem; DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (dibromothymoquinone); DCIP, 2,6-dichlorophenolindophenol; ϕ-BQ, phenylbenzoquinone; 2,5(Cl_2)-BQ, 2,5-dichlorobenzoquinone; TMPD, *N,N,N'N'*-tetramethyl-*p*-phenylenediamine; UHDBT, 5-*n*-undecyl-6-hydroxy-4,7-dioxobenzothiazole; Tricine, *N*-[2-hydroxy-1,1-bis(hydroxymethyl)ethyl]glycine.

the photooxidation of water by PS II and, concomitantly, to act as a transmembrane shuttle of protons from stroma into the thylakoid inner aqueous space (lumen) [3–5,9]. It is widely held that when plastoquinone function is inhibited by DBMIB, intersystem electron transfer from PS II to PS I is blocked and the remaining oxygenic electron flow (i.e., flow of electrons originating from water) reflects only PS II activity. In fact, oxygenic electron transport limited to PS II is often defined as that segment in the overall noncyclic electron transport in chloroplasts which is not inhibited by DBMIB [3,10–15].

The view that electron flow from water via PS II and PS I is sensitive to DBMIB, whereas electron flow from water that involves PS II alone is not sensitive had its origin in the work of Böhme et al. [10]. They found that in intact thylakoids photoreduction of ferricyanide and 2,6-dichlorophenolindophenol (DCIP) was inhibited by DBMIB but no inhibition occurred in fragmented (sonicated) thylakoids. They interpreted these findings as showing that in intact thylakoids ferricyanide and DCIP were reduced mainly by PS I, but in sonicated thylakoids both oxidants were reduced solely by PS II. They concluded that sonication removed plastocyanin, and thereby disconnected PS II from PS I; sonication also made the acceptor site of PS II accessible to polar oxidants such as ferricyanide [10,3].

A different perspective on the effect of DBMIB on oxygenic electron transport that is limited to PS II emanates from a recent hypothesis for an additional hitherto unrecognized, role of the plastoquinone pool in processing protons derived from the photooxidation of water by PS II [16–18]. This 'plastoquinone in PS II' hypothesis rests on the premise that the photooxidation of water takes place in the hydrophobic core of the thylakoid membrane which is poorly permeable to the released protons whose conductance depends on DBMIB-sensitive plastoquinone-plastohydroquinone transitions [16]. Since proton extrusion must accompany electron transport from water to an oxidant such as ferricyanide that does not accept protons, this hypothesis predicts that photoreduction of ferricyanide, even when carried out solely by PS II, would be sensitive to DBMIB.

The aim of this investigation was to subject these opposing views to a direct experimental test by the use of a PS II preparation, capable of photooxidizing water and reducing ferricyanide but devoid of PS I activity. The prevailing view predicts that an oxygenic photoreduction of ferricyanide by such a PS II preparation would be insensitive to DBMIB inhibition whereas, as stated, the 'plastoquinone in PS II' hypothesis predicts that it should be sensitive to DBMIB inhibition.

We report here the inhibition by DBMIB of electron transport from water to ferricyanide in a PS II preparation derived from the appressed grana lamellae rich in PS II and physically separated, by a relatively mild procedure that is likely to minimize artifacts, from the stroma-exposed lamellae rich in PS I. The PS II preparation was made without sonication or detergents. It consisted of plastocyanin-free chloroplast membrane vesicles isolated by the aqueous polymer two-phase partition method [19,20], and turned inside-out with respect to the original sidedness of the membrane [21–24].

The PS II in the inside-out vesicles (henceforth referred to as the oxygenic photosystem) reduced ferricyanide at rates comparable to those in unfractionated thylakoids. The oxygenic reduction of ferricyanide was inhibited by the same concentrations of DBMIB that inhibit ferricyanide reduction in unfractionated thylakoids in which PS II is connected to PS I.

A process that was not inhibited by DBMIB was the reduction by the oxygenic photosystem of DCIP. Furthermore, DCIP and other lipophilic hydrogen (electron plus proton) acceptors were also effective as mediators in reversing the DBMIB inhibition of ferricyanide reduction by the oxygenic photosystem. This pattern of reversal of DBMIB inhibition of ferricyanide reduction by lipophilic mediators was previously observed only in unfractionated thylakoids (reviewed in Refs. 3 and 15). However, the same lipophilic mediators did not relieve the inhibition of ferricyanide reduction by low concentrations of UHDBT that inhibit plastoquinone reduction [6–8].

The bearing of these findings on the role of plastoquinone in the conductance of protons liberated by the photooxidation of water by PS II is discussed.

Methods

Chloroplasts were isolated from freshly harvested spinach leaves (*Spinacia oleracea*, var. Marathon) grown in a greenhouse in nutrient solution culture [25]. Whole (unfractionated) chloroplasts were prepared as previously described [16,25]. Inside-out vesicles enriched in PS II were prepared as described by Åkerlund and Andersson [26], with minor modifications. The main steps included Yeda-press disruption of thylakoid membranes in the presence of $MgCl_2$, followed by four or five (the respective preparations are designated B4 and B5) successive aqueous polymer two-phase (dextran-poly(ethylene glycol)) partitionng steps. Chlorophyll was estimated [25] and $NADP^+$ photoreduction was measured [27] as described. Spinach ferredoxin and plastocyanin were prepared by conventional methods. The A_{278}/A_{597} ratio of oxidized plastocyanin was 2.0. Photoreduction of ferricyanide was measured spectrophotometrically by absorbance changes at 420 nm. DBMIB and UHDBT were the gifts, respectively, of Professor A. Trebst and Professor R. Malkin. *N*,*N*,*N'*,*N'*-tetramethyl-*p*-phenylenediamine (TMPD) was purchased from British Drug Houses Ltd., and 2,5-dichlorobenzoquinone (2,5(Cl_2)BQ) and phenylbenzoquinone (ϕ-BQ) from Eastman Kodak Co. DBMIB and UHDBT were dissolved in dimethyl sulfoxide and the lipophilic mediators were dissolved in methanol. Equal concentrations of solvents were added to the respective controls.

Results

Electron transport capacity of the oxygenic photosystem

Table I provides evidence that, as isolated, the oxygenic photosystem in the inside-out vesicles had the characteristics of a typical PS II preparation. It was devoid of plastocyanin (lost during the Yeda-press fractionation step) and, without plastocyanin, was incapable of oxygenic reduction of $NADP^+$. However, it exhibited an almost undiminished capacity for oxygenic reduction of high-potential acceptors such as ferricyanide. The oxygenic photosystem reduced ferricyanide at an appreciable rate that was virtually uninfluenced by the addition of plastocyanin. The relatively high rates of photoreduction of ferricyanide indicate that the inside-out orientation of the oxygenic photosystem gave polar acceptors such as ferricyanide ready access to reducing equivalents from PS II on the inside surface of the thylakoids.

In agreement with previous results [28], the addition of catalytic amounts (2 μM) of plastocyanin to the oxygenic photosystem resulted in an appreciable rate of $NADP^+$ reduction (Table I). This effect of plastocyanin on $NADP^+$ reduction by the oxygenic photosystem was not due to an artifactual interaction of reduced plastocyanin with the small PS I contaminant remaining in the inside-out vesicles. The subject was dealt with in detail elsewhere [28] and will not be pursued further here because all subsequent experiments were done without the addition of plastocyanin, i.e., under conditions in which the oxygenic photosystem functioned like a typical PS II preparation: it was capable of photoreducing only high-potential (PS II) acceptors.

Differential sensitivity of ferricyanide and DCIP reduction to DBMIB

In agreement with the results of Böhme et al. [10], we found differences between intact and fragmented thylakoids in DBMIB sensitivity of DCIP reduction. The reduction of DCIP by unfractionated thylakoids (containing plastocyanin,

TABLE I

OXYGENIC PHOTOREDUCTION OF FERRICYANIDE AND $NADP^+$ BY INSIDE-OUT THYLAKOID VESICLES AS INFLUENCED BY THE ADDITION OF PLASTOCYANIN

The basic reaction mixture contained 50 mM Mops (4-morpholinepropanesulfonic acid) buffer (pH 6.7) and 5 mM $MgCl_2$ and, when added, 2 μM plastocyanin. Additions to the basic reaction mixture for the ferricyanide treatments included 4 mM potassium ferricyanide and inside-out thylakoid vesicles (B5) equivalent to 100 μg chlorophyll per ml. Additions to the basic reaction mixture for the $NADP^+$ treatments included 10 μM ferredoxin, 2 mM $NADP^+$, a saturating amount of ferredoxin-$NADP^+$ reductase and inside-out thylakoid vesicles equivalent to 50 μg chlorophyll per ml.

	(μequiv. reduced per mg Chl per h)	
	Ferricyanide	$NADP^+$
Control	414	2
+ Plastocyanin	432	110

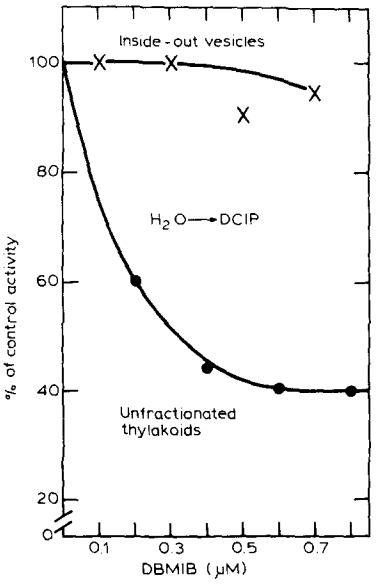

Fig. 1. Inhibition of DBMIB of electron transport from water to DCIP by unfractionated thylakoids and the oxygenic photosystem (PS II) of inside-out vesicles. For unfractionated thylakoids the basic reaction mixture contained 50 mM Tricine buffer (pH 8.2)/5 mM $MgCl_2$/2.5 mM ADP/2.5 mM K_2HPO_4. For inside-out vesicles, the basic reaction mixture contained 50 mM Mops (4-morpholinepropanesulfonic acid) buffer (pH 6.7) 5 mM $MgCl_2$ and inside-out thylakoid vesicles (B4) equivalent to 50 μg chlorophyll. Other additions were 40 μM DCIP and the appropriate concentration of DBMIB. The reaction mixtures were incubated for 3 min in the dark and illuminated at room temperature in cuvettes (2 mm light path) open to air. Monochromatic illumination (670 nm) was 50 $J \cdot m^{-2} \cdot s^{-1}$. The rate of reduction without inhibitor was 162 μequiv. per mg Chl per h for unfractionated thylakoids and 114 for DCIP.

PS II and PS I) was inhibited by low concentrations of DBMIB, whereas reduction of DCIP by inside-out vesicles (lacking plastocyanin and a functional PS I) was not inhibited by DBMIB (Fig. 1).

However, unexpectedly different from the observations with sonicated thylakoids [10] were the effects of DBMIB on the reduction of ferricyanide. Ferricyanide reduction was inhibited to virtually the same extent by low concentrations of DBMIB in the unfractionated thylakoids as in the inside-out vesicles lacking plastocyanin and containing only a functional PS II (Fig. 2).

Interestingly enough, although the DBMIB effect on ferricyanide reduction by the oxygenic photosystem (PS II) in inside-out vesicles differed from the results of Böhme et al. [10] with sonicated thylakoids, it was in agreement with observations on *Euglena* chloroplasts reported by Trebst [29]. In *Euglena* chloroplasts, as in sonicated spinach chloroplasts, PS II and PS I were deemed to be disconnected because the water-soluble cytochrome-552 (equivalent to plastocyanin in spinach chloroplasts) was lost during preparation; the oxygenic photoreduction of ferricyanide and DCIP was thus attributed solely to PS II [29]. However, *Euglena* chloroplasts differed from sonicated spinach chloroplasts [10] in that ferricyanide reduction was strongly inhibited by DBMIB whereas DCIP reduction was only mildly inhibited [29].

Effect of lipophilic mediators

Further evidence that the pattern of DBMIB sensitivity of ferricyanide reduction by the inside-out vesicles was similar to that in unfractionated thylakoids was provided by the reversal of DBMIB inhibition by lipophilic mediators. Other investi-

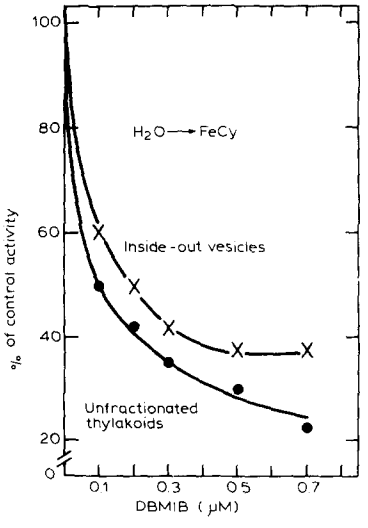

Fig. 2. Inhibition by DBMIB of electron transport from water to ferricyanide by unfractionated thylakoids and the oxygenic photosystem (PS II) of inside-out vesicles. The basic reaction mixtures were as in Fig. 1. Other additions were 4 mM potassium ferricyanide and the appropriate concentration of DBMIB. Monochromatic illumination (650 nm) was 50 $J \cdot m^{-2} \cdot s^{-1}$. Other conditions were as in Fig. 1. The rate of reduction without inhibitor was 432 μequiv. per mg Chl per h for unfractionated thylakoids and 288 for inside-out vesicles.

gations have amply documented that in unfractionated thylakoids, DBMIB inhibition of ferricyanide reduction is relieved by the mediation of catalytic amounts of lipophilic PS II oxidants that include various substituted phenylenediamines and benzoquinones [3,11–15,33–35]. Fig. 3 shows a similar effect of lipophilic mediators in inside-out vesicles. ϕ-BQ, 2,5-(Cl_2)-BQ and *p*-phenylenediamine relieved the DBMIB inhibition of ferricyanide reduction by the oxygenic photosystem [11–13].

Do mediators bypass the plastoquinone pool in ferricyanide reduction?

The efficacy of mediators in reversing the DBMIB inhibition of ferricyanide reduction by the oxygenic photosystem (Fig. 3) raised the question of wheter the mediators interact with Q_B and thereby bypass the plastoquinone pool entirely. Q_B is a protein-bound plastoquinone that serves as a two-electron gate for the reduction of the plastoquinone pool (reviews in Refs. 4, 31 and 32). Indeed, in investigations with unfractionated thylakoids, the suggestion was made that lipophilic mediators reverse DBMIB inhibition of ferricyanide reduction because they are reduced at an intermediate reduction site buried in the lipid membrane "before the electrons from PS II are pooled", i.e., before the reduction of the plastoquinone pool [35]. The mediator thus reduced would in turn be reoxidized by ferricyanide [35].

If the mediators were reduced by Q_B, they could reverse the inhibition of ferricyanide reduction by agents, such as UHDBT, that inhibit the reduction of plastoquinone [6–8]. Fig. 4 shows that UHDBT at concentrations of less than 1 mM strongly inhibited the reduction of ferricyanide by the oxygenic photosystem. However, the inhibition by UHDBT was not reversed by the addition of ϕ-BQ, 2,5-Cl_2)-BQ or DCIP, each of which was effective as a mediator in reversing the inhibition by DBMIB. It appears, therefore, that in inside-out vesicles, as in unfractionated thylakoids [3,11–14], lipophilic mediators were not reduced by Q_B but by plastohydroquinone and were in turn reoxidized by ferricyanide.

We have also investigated the mediator effect of TMPD (an N-substituted phenylenediamine), which is a lipophilic electron carrier but not a proton carrier [13,14]. In previous studies with unfractionated thylakoids, TMPD in the presence of DBMIB was practically inactive in restoring electron flow from water to ferricyanide but was highly active in restoring DBMIB-inhibited electron flow from water to $NADP^+$ [13]. In the oxygenic photosystem, TMPD, although somewhat less effective than the other mediators, relieved to an appreciable degree the DBMIB inhibi-

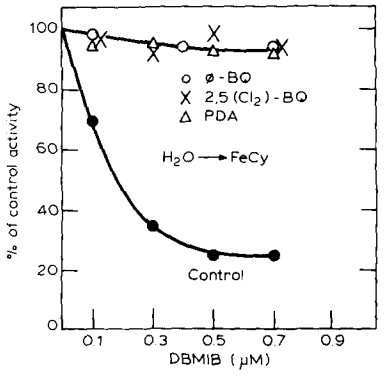

Fig. 3. Reversal by mediators of DBMIB inhibition of electron transport from water to ferricyanide. The concentration of mediators was 100 μM for phenylbenzoquinone (φ-BQ) and phenylenediamine (PDA) and 50 μM for 2,5(Cl_2)-BQ. The uninhibited rats of ferricyanide reduction (μequiv. per mg Chl per h) were: control (no mediator, 590, ϕ-BQ, 594; PDA, 630; and 2,5(Cl_2)-BQ, 783. Other conditions were as in Fig. 2.

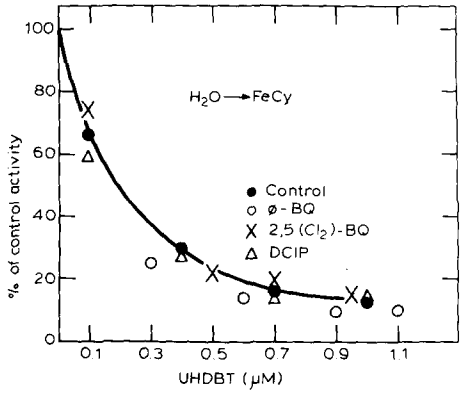

Fig. 4. Ineffectiveness of mediators in relieving UHDBT inhibition of electron transport from water to ferricyanide. The concentration of DCIP was 40 μM; 2,5(Cl_2)-BQ, 50 μM; ϕ-BQ, 100 μM. The uninhibited rates of ferricyanide reduction (μequiv. per mg Chl per h) were: control (no mediator), 482; φ-BQ, 648; 2,5(Cl_2)-BQ, 711; and DCIP, 388. Other conditions were as in Fig. 2.

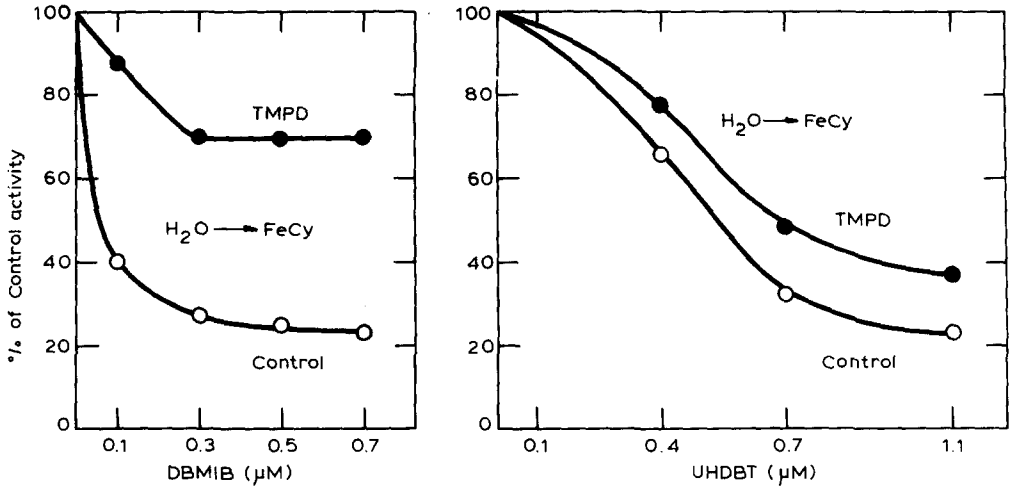

Fig. 5. TMPD as a mediator in relieving inhibition by DBMIB and UHDBT of electron transport from water to ferricyanide. TMPD concentration was 100 μM. The uninhibited rates of ferricyanide reduction (μequiv. per mg Chl per h) were (without TMPD) 468 in the DBMIB series and 378 in the UHDBT series and (with TMPD) 513 and 441, respectively. Other conditions were as in Fig. 2.

tion of electron flow from water to ferricyanide (Fig. 5). Like the other mediators, TMPD had little effect in relieving the UHDBT inhibition of the oxygen ferricyanide reduction (Fig. 5).

To recapitulate, it appears that lipophilic mediators restored oxygenic ferricyanide reduction in the presence of DBMIB by providing an alternative way for the oxidation of plastohydroquinone, but were ineffective in the presence of low concentrations of UHDBT which inhibit plastoquinone reduction [6–8]. These results suggest that the reduction of the plastoquinone pool, which we regard as the carrier of protons released by the photooxidation of water, is a critical step for the functioning of the oxygenic photosystem. When the reduction of plastoquinone is blocked, the photooxidation of water cannot be restored by mediators.

Discussion

Investigations with DBMIB over the last 15 years led to wide agreement that this inhibitor of plastoquinone oxidation blocks only intersystem electron transport from PS II to PS I and does not inhibit oxygenic electron transport that is limited to PS II (reviewed in Refs. 3 and 15). As stated, oxygenic electron transport that is limited to PS II is often defined as that portion of the overall noncyclic electron transport in chloroplasts which is not inhibited by DBMIB [3,15].

The main import of our findings is that they contradict this popular interpretation. They demonstrate the same sensitivity of ferricyanide reduction to DBMIB inhibition by a PS II preparation as by unfractionated thylakoids. The PS II preparation (the oxygenic photosystem) consisted of inside-out vesicles depleted of plastocyanin and, without plastocyanin, capable of reducing high-potential PS II oxidants but not low-potential PS I oxidants.

From the standpoint of the 'plastoquinone in PS II' hypothesis we interpret our results as follows. Regardless of whether ferricyanide or DCIP was the oxidant, plastoquinone was involved in the processing of water-derived protons by undergoing reduction and protonation to plastohydroquinone. The processing, i.e., the removal, of water-derived protons from the hydrophobic domain of the vesicles is essential for the functioning of PS II and can only be completed if plastohydroquinone is reoxidized. Plastohydroquinone remained in the hydrophobic domain of the vesicles, where it was accessible to the lipophilic DCIP, by which it was chemically oxidized. The chemical oxidation of plastohydroquinone by DCIP was not sensitive to DBMIB, rendering the

overall reaction from water to DCIP insensitive to DBMIB.

When the polar ferricyanide was the oxidant in the absence of lipophilic mediators, plastohydroquinone in the vesicles remained inaccessible to chemical oxidation. We propose that it was oxidized, as in unfractionated thylakoids, by the Rieske iron-sulfur center of the FeS-cytochrome f/b_6 complex [4,5,30–32,36] generating reduced cytochrome f which in the inside-out vesicles was readily accessible to direct oxidation by ferricyanide. Since the oxidation of plastohydroquinone by the cytochrome complex is sensitive to DBMIB [4,5], the overall electron transport from water to ferricyanide was also sensitive to DBMIB. Lipophilic mediators reversed DBMIB inhibition because they provided an alternative way for oxidizing plastohydroquinone. This interpretation would explain why ferricyanide reduction by the inside-out vesicles, i.e., by PS II alone, showed the same sensitivity to DBMIB as by unfractionated thylakoids.

We are thus led to the conclusion that the FeS-cytochrome f/b_6 complex is a functional component of the oxygenic photosystem even when it operates without the anoxygenic system (PS I). Consistent with this conclusion are findings that the FeS-cytochrome f/b_6 complex is present in about the same abundance in the oxygenic photosystem of inside-out vesicles as in unfractionated chloroplasts (Refs. 37 and 38, and unpublished data from this laboratory; see also Ref. 39). The sole report to the contrary [40] has not been confirmed. On the other hand, in PS II preparations made with detergents, cytochrome f is depleted in some [41] or totally absent in others [41–43].

Our findings provide further support for the proposed role of the plastoquinone pool in the conductance of protons liberated by photochemical reactions in chloroplasts. As discussed elsewhere [16–18], we envision, aside from electron transport, a dual role for the plastoquinone pool in the transfer of protons into the thylakoid lumen: one, in the conductance of protons that originate from the intramembrane photooxidation of water by PS II; and another, linked to PS I, in the transmembrane shuttle of protons originating in the stroma.

We have previously assigned the FeS-cytochrome f/b_6 complex only to the anoxygenic photosystem (see Ref. 17 and Fig. 3 in Ref. 44) but it now appears more likely that, like plastoquinone and plastocyanin, this complex also has a dual function, one in the oxygenic and another in the anoxygenic photosystem. A more detailed elaboration of this concept will be presented separately.

References

1 Henninger, M.D. and Crane, F.L. (1963) Biochemistry 2, 1168–1171
2 Amesz, J. (1973) Biochim. Biophys. Acta 301, 35–51
3 Trebst, A. (1980) Methods Enzymol. 69, 675–715
4 Hauska, G., Hurt, E., Gabellini, N. and Lockau, W. (1983) Biochim. Biophys. Acta 726, 97–133
5 Malkin, R., Chain, R.K. and Lam, E. (1983) Chem. Scripta 21, 75–80
6 Oettmeier, W., Masson, K. and Goode, D. (1981) Z. Naturforsch. 36B, 272–275
7 Hsu, B.-D. and Arnon, D.I. (1982) Photobiochem. Photobiophys. 4, 187–193
8 Whitmarsh, J., Bowyer, J.R. and Crofts, A.R. (1982) Biochim. Biophys. Acta 682, 404–412
9 Trebst, A. and Avron, M. (1977) in Photosynthesis I. Encyclopedia of Plant Physiology, New Series, Vol. 5, pp. 1–4, Springer-Verlag, Berlin
10 Böhme, H., Reimer, S. and Trebst, A. (1971) Z. Naturforsch. 26B, 341–352
11 Trebst, A. and Reimer, S. (1973) Biochim. Biophys. Acta 305, 129–139
12 Trebst, A. and Reimer, S. (1973) Biochim. Biophys. Acta 325, 546–557
13 Trebst, A. and Reimer, S. (1973) Z. Naturforsch. 28C, 710–176
14 Selman, B.R. (1976) J. Bioenerg. Biomembranes 8, 143–156
15 Izawa, S. (1980) Methods Enzymol. 69, 413–434
16 Arnon, D.I. and Tang, G.M.-S., (1985) Biochim. Biophys. Acta 809, 167–172
17 Arnon, D.I., Tsujimoto, H.Y. and Tang, G.M.-S. (1981) Proc. Natl. Acad. Sci. USA 78, 2942–2946
18 Arnon, d.I., Tsujimoto, H.Y. and Tang, G.M.-S. (1982) Biochem. Biophys. Res. Commun. 106, 450–457
19 Åkerlund, H.-E., Andersson, B. and Albertsson, P.-Å. (1976) Biochim. Biophys. Acta 449, 525–535
20 Albertsson, P.-Å., Andersson, B., Larsson, C. and Åkerlund, H.-E. (1982) Methods Biochem. Anal. 28, 115–150
21 Andersson, B., Åkerlund, H.-E. and Albertsson, P.-Å. (1977) FEBS Lett. 77, 141–145
22 Andersson, B. and Åkerlund, H.-E. (1978) Biochim. Biophys. Acta 503, 462–472
23 Andersson, B., Sundby, C. and Albertsson, P.-Å. (1980) Biochim. Biophys. Acta 599, 391–402
24 Andersson, B. and Anderson, J.M. (1980) Biochim. Biophys. Acta 593, 427–440

25 Arnon, D.I. (1949) Plant Physiol. 24, 1–15
26 Åkerlund, H.-E. and Andersson, B. (1983) Biochim. Biophys. Acta 725, 34–40
27 McSwain, B.D. and Arnon, D.I. (1968) Proc. Natl. Acad. Sci. USA 61, 989–996
28 Albertsson, P.-Å., Hsu, B.-D., Tang, G.M.-S., and Arnon, D.I. (1983) Proc. Natl. Acad. Sci. USA 80, 3971–3975
29 Trebst, A. (1972) Proceedings of the 2nd International Congress on Photosynthesis Research, Stresa, 1971 (Forti, G., Avron, M. and Melandri, A., eds.), Martinus Nijhoff/Dr. W. Junk Publishers, Dordrecht, The Netherlands
30 Malkin, R. (1983) Biochemistry 21, 2945–2950
31 Crofts, A.R. and Wraight, D.A. (1983) Biochim. Biophys. Acta 726, 149–185
32 Rich, P.R. (1984) Biochim. Biophys. Acta 768, 53–79
33 Izawa, S., Gould, J.M., Ort, D.R., Felker, P. and Good, N.E. (1973) Biochim. Biophys. Acta 305, 119–128
34 Gould, J.M. and Izawa, S. (1973) Biochim. Biophys. Acta 314, 211–223
35 Gould, J.M. and Izawa, S. (1973) Eur. J. Biochem. 37, 185–192
36 Lam, E. and Malkin, R. (1982) FEBS Lett. 144, 190–194
37 Anderson, J.M. (1981) FEBS Lett. 124, 1–10
38 Cox, R.P. and Andersson, B. (1982) Biochem. Biophys. Res. Commun. 103, 1336–1342
39 Anderson, J.M. and Malkin, R. (1982) FEBS Lett. 148, 293–296
40 Henry, L.A. and Lindberg-Møller, B. (1981) Carlsberg Res. Commun. 46, 226–242
41 Dunahay, T.G., Staehelin, L.A., Seibert, M., Ogilvie, P.D. and Berg, S.P. (1984) Biochim. Biophys. Acta 764, 179–193
42 Berthold, D.A., Babcock, G.T. and Yocum, C.F. (1982) FEBS Lett. 134, 231–234
43 Whitford, D., Gounaris, K. and Barber, J. (1984) in Advances in Photosynthesis Research (Sybesma, C., ed.), Vol. I, pp. 497–500, Martinus Nijhoff/Dr. W. Junk Publishers, Dordrecht, The Netherlands
44 Arnon, D.I. (1984) Trends Biochem. Sci. 9, 258–262